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Life's Big Bangs

Did complex life emerge more than once?

The Great Meteorite Heist

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Mysterious Rocks Could Rewrite Evolution of Complex Life

Controversial evidence hints that complex life might have emerged hundreds of millions of years earlier than previously thought—and possibly more than once

By [Asher Elbein](#) edited by [Kate Wong](#)



Deena So‘Oteh

In his laboratory at the University of Poitiers in France, Abderrazak El Albani contemplates the rock glittering in his hands. To the untrained eye, the specimen resembles a piece of golden tortellini embedded in a small slab of black shale. To El Albani, a geochemist, the pasta-shaped component looks like the remains of a complex life-form that became fossilized when the sparkling mineral pyrite replaced the organism’s tissues after death. But the rock is hundreds of millions of years older than the oldest accepted fossils of advanced multicellular life. The question of whether it is a paradigm-shifting fossil or merely an ordinary lump of fool’s gold has consumed El Albani for the past 17 years.

In January 2008 El Albani, a talkative French Moroccan, was picking over an exposed scrape of black shale outside the town of Franceville in Gabon. Lying under rolling hills of tropical savanna, cut in places by muddy rivers lined by jungle, the rock layers of the Francevillian Basin are up to 2.14 billion years old. The strata are laced with enough manganese to support a massive mining industry. But El Albani was there pursuing riches of a different kind.

Most sedimentary rocks of that age are thoroughly “cooked,” transformed beyond recognition by the brutal heat and pressure of deep burial and deeper time. Limestone is converted to marble, sandstone to quartzite. But through an accident of geology, the Francevillian rocks were protected, and their sediments have maintained something of their original shape, crystal structure and mineral composition. As a result, they offer a rare window into a stretch of time when, according to paleontologists, oxygen was in much shorter supply and Earth’s environments would have been hostile to multicellular organisms like the ones that surround us today.

El Albani had been invited out by the Gabonese government to conduct a geological survey of the ancient sediments. He spent half a day wandering the five-meter-deep layer of the quarry, peeling apart slabs of shale as if opening pages of a book. The rocks were filled with gleaming bits of pyrite that occurred in a variety of bizarre shapes. El Albani couldn’t immediately explain their appearance by any common sedimentary process. Baffled, he took a few samples with him when he returned to Poitiers. Two months later he scraped together funding to head back to the Francevillian quarry. This time he went home with more than 200 kilograms of specimens in his luggage.

In 2010 El Albani and a team of his colleagues [made a bombshell claim based on those finds](#): the strangely shaped specimens they’d recovered in Franceville were fossils of complex life-forms—

organisms made up of multiple, specialized cells—that lived in colonies long before any such thing is supposed to have existed. If the scientists were right, the traditional account of life’s beginning, which holds that complex life originated once around 1.6 billion years ago, is wrong. And not only did complex multicellular life appear earlier than previously thought, but **it might have done so multiple times**, sprouting seedlings that were wiped away by a volatile Earth eons before our lineage took root. El Albani and his colleagues have pursued this argument ever since.



Rocks from the Francevillian Basin in Gabon are filled with gleaming shapes that have been interpreted as fossils of complex life-forms from more than two billion years ago.

The potential implications of their claims are immense—they stand to rewrite nearly the entire history of life on Earth. They’re also incredibly controversial. Almost immediately, prominent researchers [argued](#) that El Albani’s specimens are actually [concretions of natural pyrite](#) that only *look* like fossils. Mentions of the Francevillian rocks in the scientific literature tend to be accompanied by words such as “[uncertain](#)” and “[questionable](#).”

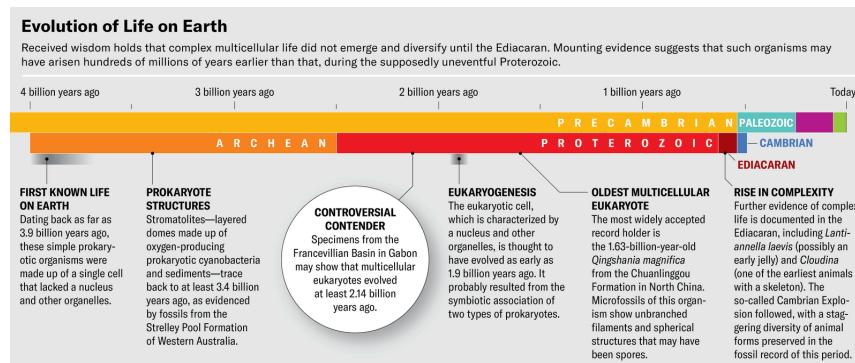
Yet even as most experts regard the Francevillian specimens with a skeptical eye, a slew of recent discoveries from other teams have challenged older, simpler stories about the origin of life. Together with these new finds, the sparkling rock El Albani held in his hands has raised some very tricky questions. What conditions did complex life need to emerge? How can we recognize remains of life from deep time when organisms then would have been entirely different from those that we know? And where do the burdens of proof lie for establishing that complex life arose far earlier than previously thought—and more than just once?

By most accounts, life on Earth first emerged around four billion years ago. In the beginning, the oxygen that sustains most species today had yet to suffuse the world’s atmosphere and oceans. Single-celled microbes reigned supreme. In the anoxic waters, bacteria spread and fed on minerals around hydrothermal vents. Then, maybe 2.5 billion years ago, so-called cyanobacteria that gathered in mats and gave rise to great stone domes called stromatolites began feeding themselves using the power of the sun. In doing so, they kick-started a slow transformation of the planet, pumping Earth’s seas and atmosphere full of oxygen as a by-product of their feeding.

That transformation would eventually devastate the first, oxygen-averse microbial residents of Earth. But amid [a gathering oxygen apocalypse](#), something new appeared. Roughly two billion years

ago a symbiotic union between two groups of single-celled organisms—one of which was able to process oxygen—gave rise to the earliest eukaryotes: larger cells with a membrane-bound nucleus, distinctive biochemistry and an aptitude for sticking together. Somewhere in the vast sweep of time between then and now, in something of a glorious accident, those eukaryotes began banding together in specialized ways, forming intricate and increasingly complex multicellular organisms: algae, seaweeds, plants, fungi and animals.

Scholars have long endeavored to understand when that transition from the single-celled to the multicellular happened. By the mid-19th century researchers noticed that the fossil record got considerably livelier at a certain point, which we now know was around 540 million years ago. During this period, called the Cambrian, multicellular eukaryotes seemed to explode in diversity out of nowhere. Suddenly the seas were filled with trilobites, meter-long predatory arthropods, and even the earliest forerunners of vertebrates, the backboned lineage of animals to which we humans belong.



Jen Christiansen

But it wasn't long before scientists began finding older hints of multicellular organisms, suggesting that complex life proliferated before the Cambrian. In 1868 a geologist proposed that tiny, disk-shaped objects from sediments more than 500 million years old in Newfoundland were fossils—only for other researchers to dismiss them as inorganic concretions. Similarly ancient fossils from

elsewhere in the world turned up over the first half of the 20th century. The most famous of them—discovered in Australia’s Ediacara Hills by geologist Reginald Claude Sprigg, who took them to be jellyfish—helped to push the dawn of complex life back to least 600 million years ago, into what came to be called the Ediacaran period.

Still, a gap of more than a billion years separates the earliest known eukaryotes and their great flowering in the Ediacaran. The contrast between the apparent evolutionary stasis of the bulk of this period and the eventful periods before and after it is so stark that researchers variously refer to it as “the dullest time in Earth’s history” and the “boring billion.” Why didn’t many-celled eukaryotes start diversifying earlier, wonders Susannah Porter, a paleontologist at the University of California, Santa Barbara? Why didn’t they explode until the Ediacaran?

Researchers have historically blamed environmental conditions on ancient Earth for the delay. The dawn of the Ediacaran, they note, coincided with a noticeable shift in global conditions 635 million years ago. In the wake of a world-spanning glacial event—the so-called Snowball Earth period, when great sheets of ice scraped the continents and covered the seas—the available nutrients in the oceans shifted amid a surge in levels of available oxygen. The friendlier water chemistry and more abundant oxygen provided new opportunities for eukaryotic organisms that could exploit them. They diversified quickly and dramatically, first into the stationary animals of the Ediacaran and eventually into the more active grazers and hunters of the Cambrian. It’s a commonly cited explanation for the timing of life’s big bang, one that the field tends to accept, Porter says. And it may well be correct. But if you asked El Albani, he’d say it’s not the whole story—far from it.

As a kid growing up in Marrakech, El Albani wasn’t interested in geology; football and medicine held more appeal. He drifted into the field when he was 20 largely because it let him spend time

outside. He then fell in love with it in part because like his father, a police officer, he enjoys a good investigation, working out what happened in some distant event by laying out multiple lines of evidence.

In the case of the ancient Gabon “fossils,” the first line of evidence involves the unusual geology of the Francevillian formation.

Unlike most sedimentary rocks laid down two billion years ago—fated for deep burial and transformative heat and pressure—the Francevillian strata sit within a bowl of much tougher rock, which prevented them from being cooked. The result: shales able to preserve both biological forms and something close to the primary chemicals and minerals present in the marine sediments. “It gives us the possibility of actually reconstructing this environment that existed in the past, at a scale that we don’t see anywhere around this time,” says Ernest Chi Fru, a biogeochemist at Cardiff University in Wales, who has worked with El Albani on the Francevillian material. If you were searching for fossils of relatively large, soft-bodied multicellular organisms from this period, the Francevillian is exactly the kind of place you’d look in.

“I don’t know what we need to show to prove, to convince.”

—Abderrazak El Albani *University of Poitiers*

El Albani’s team has recovered quite a few such specimens. Three narrow rooms in the geology building at the University of Poitiers house the Francevillian collection. More than 6,000 pieces—all of them collected from the same five-meter scrape of Gabonese shale—sprawl over wood shelves and tables and glass display cabinets, the black slabs arranged in puzzle-piece configurations under white walls. El Albani is eager to show them off. He plucks out rock after rock, no sooner highlighting one when he’s distracted by another. Here are the ripplelike remnants of bacterial mats. There are the specimens encrusted with pyrite: the common, tortellinilike “lobate” forms that made the cover of the journal *Nature* in 2010, “tubate” shapes that resemble stethoscopes and spoons, and other

forms similar to strings of pearls several centimeters long. There are strange, wormlike tracks that the team has suggested could be [traces of movement](#). There are nonpyritized remains, too: sand-dollar-like circles ranging from one to several centimeters across imprinted on the shales.

“*Et voilà,*” El Albani says, tapping one specimen and then another. “You see? This is totally different.” The sheer variety of forms is why he’s always surprised that people could look at them and assume they aren’t in fact fossils. Nevertheless, his lab has been exploring ways to attempt to prove their identity.

One approach El Albani’s lab has taken recently is looking into the chemistry of the specimens. Eukaryotic organisms tend to take up lighter forms, or isotopes, of elements such as zinc rather than heavy ones. When [examining the sand-dollar-shaped impressions in 2023](#), the team found that the zinc isotopes in them were mostly lighter forms, suggesting the impressions could have been made by eukaryotes. (An independent team ran a similar study of one of the pyritized specimens and reached a similar conclusion.)

Earlier this year El Albani’s Ph.D. student Anna El Khoury [reported](#) another potential chemical signal for life in the contested rocks. Organisms in areas thick with arsenic sometimes absorb the poisonous chemical instead of necessary nutrients such as phosphate. Whereas confirmed mineral concretions from the Francevillian show a random distribution of arsenic in the rock, the possibly organic specimens El Khoury looked at showed dramatic concentrations of the toxin only in certain parts of the specimens, as would be expected if an organism’s cells were working to isolate the absorbed substance from more vulnerable tissues.

What El Albani and his colleagues find most telling, however, are the environmental conditions that are now known to have prevailed when the putative fossils formed. The sediments that make up the Francevillian strata appear to have been deposited in something

like an inland sea. [The rocks show signals](#) of dramatic underwater volcanism and hydrothermal vent activity from long before the first fossil specimens appear, which left the basin awash in nutrients such as phosphorus and zinc that are crucial for the chemical processes that power living cells.



Chemical analyses of the Francevillian specimens suggest that they are the remains of eukaryotic organisms.

Abderrazak El Albani/University of Poitiers

What is more, the Francevillian samples, like the Ediacaran fossils, are from a time after a major period of ice ages: [the Huronian glaciation](#) event, wherein a surge in oxygen levels and a reduction in the greenhouse effect 2.4 billion to 2.1 billion years ago unleashed massive walls of ice from the poles. According to some analyses, that spike in oxygen levels might have hit a peak close to that in the Ediacaran before eventually falling again. In other words, the same environmental conditions that are thought to have allowed complex life to flower during the Ediacaran also occurred far earlier and could have set the stage for the emergence of Francevillian life-forms.

Talk with the people in El Albani's lab about the Francevillian, and they'll paint you a picture of an alien world. Ancient shorelines run under the brooding gaze of distant mountains, silent but for the wind and the waves. Thick mats of bacteria stretch across the

underwater sediments. Swim down 20 meters offshore, through waters thick with nutrients and heavy metals such as arsenic, and you might see colonies of spherical and tube-shaped organisms clustered amid the mats. In the oxygen-rich water column, soft-bodied organisms drift like jellyfish, sinking now and then into the mire. Below the silt, unseen movers leave spiraling mucus trails in the ooze.

What were these strange forms of life? Not plants or animals as we understand them. Based on the sizes, shapes and geochemical signatures of the putative fossils, El Albani thinks they might belong to a lineage of colonial eukaryotes—perhaps something resembling a slime mold—that independently developed the complex multicellular processes needed to survive at large sizes. These colonial organisms would have been comparatively early offshoots of the eukaryotic tree, making them an entirely independent flowering of complex multicellular life from the Ediacaran bloom that took place more than a billion years later.

The Francevillian organisms flourished for a time, but they did not last. After a few millennia, underwater volcanism started up again, and oxygen levels crashed. A billion years would pass before another global icebox phase and another oxygen spike gave multicellular eukaryotes another shot at emergence.

This story flies in the face of decades of thinking about how complex life arose. El Albani’s team argues that rather than long epochs of stillness and stasis, rather than the rise of complex life being an extraordinary and long-brewing accident in Earth’s long history, multicellular organisms might not have been a singular innovation. “It seems to me that [the Francevillian material] is showing that complex life might have evolved twice in history,” Chi Fru says. And if ancient complex life can emerge so quickly when conditions are right, who knows where else in Earth’s rocks—or another planet’s—signs of another blossoming might turn up next? “If,” of course, being the operative word.

Skeptics of El Albani's Francevillian "fossils"—and there are many—have tended to gather around similar sticking points, says Leigh Anne Riedman, a paleontologist at the University of California, Santa Barbara. For one thing, the bizarre shapes of the rocks show a lot more variety than tends to be seen in accepted early complex multicellular forms, and with their amorphous, asymmetrical features, they do not scan easily as organisms.

The pyritized nature of the rocks may also be cause for concern. Colonies of bacteria living in oxygen-poor environments often deposit pyrite as a by-product. Although such colonies can grow a sparkling rind around biological material, the mineral concretions can also develop on their own, developing lifelike appearances without any biological process. Critics of the Francevillian hypothesis point to a well-known phenomenon of pyrite "[suns](#)" or "[flowers](#)," superficially fossil-like accumulations of minerals that occasionally turn up in sediments rich in actual fossils. Shuhai Xiao, a paleontologist at Virginia Tech specializing in the Precambrian era, notes that the Francevillian material resembles [similar-looking](#) inorganic structures from Michigan that date to 1.1 billion years ago.

If ancient complex life can emerge so quickly when conditions are right, who knows where else signs of another blossoming might turn up next?

Even scientists who are more amenable to the idea that El Albani's specimens are fossils tend to conclude that the pyritized specimens are probably just the remains of bacterial mats, not complex life-forms. An independent radiation of colonial eukaryotes at such an age? That's a hard sell. "I have no problem with there being oxygen oases and there being certain groups that proliferated during those periods," Riedman says. But the idea that they would have proliferated to that size—a jump in scale that another researcher equated to that between a human and an aircraft carrier—without

any similar fossils turning up elsewhere gives her pause. “It just seems a little bit of a stretch.”

Absence of evidence is not evidence of absence, however. In the case of the Proterozoic fossil record, the lack of other candidate fossils of complex life as old as those from the Francevillian may reflect a lack of effort in searching for them. That is, the apparent quiet of the deep past may be an illusion—less the “boring billion” than, as Porter puts it, the “barely sampled billion.”

The dullness of vast chunks of the Proterozoic has been a self-fulfilling prophecy, Riedman says. After all, who wants to devote time and scarce funding to a period when nothing much is supposed to have happened? “That name, man,” Riedman says of the boring billion. “We’ve got to kill it. Kill it with fire.”

Recent findings may help reform the Proterozoic’s cursed reputation—and cast the Francevillian rocks in a more plausible light. Just last year Lanyun Miao of the Nanjing Institute of Geology and Paleontology at the Chinese Academy of Sciences and her colleagues announced that they had discovered the [oldest unequivocal multicellular eukaryotes](#) in 1.6-billion-year-old rocks from northern China. The fossils preserve small, threadlike organisms. They’re a far cry from the much larger, more elaborate forms associated with complex multicellularity. But they show that these simpler kinds of multicellular life existed some 500 million years earlier than previously hypothesized.

There’s good reason to think the roots of the eukaryote family tree could run considerably deeper than that. Analyses of genome sequences and fossils have hinted that the earliest common ancestor of all living eukaryotes may have appeared as long as 1.9 billion years ago.



Critics argue that the forms evident in the Francevillian rocks are merely mineral concretions, not fossils of complex eukaryotic organisms.

Abderrazak El Albani/University of Poitiers

And complex multicellularity itself may develop surprisingly fast. In a [fascinating experiment](#) published a few years ago, a team at the Georgia Institute of Technology was able to get single-celled eukaryotes—in this case, yeasts—to chain together in multicellular forms visible to the naked eye in just two years. These findings, along with the growing fossil record, suggest to some researchers that multicellular eukaryotes have a deeper history than is generally recognized.

But recognizing early life in the rock is notoriously tricky. Brooke Johnson, a paleontologist at the University of Liège in Belgium, has visited Ediacaran outcrops in the U.K. with his colleagues and sometimes struggled to spot the specific fossils he knows are there.

Assessing unfamiliar structures is even more fraught. Researchers constantly second-guess themselves for fear of overinterpreting any given shape or shadow in the stone. The specter of crankhood—of being the kind of researcher who drives their work off a cliff by refusing to be proved wrong—hangs over everybody. “It’s very easy to get yourself tricked into thinking that you can see something that isn’t there, because you’re used to seeing a particular pattern,” Johnson says.

One spring morning in 2023, while working through hundreds of samples of rock more than one billion years old from drill cores from Australia, Johnson knocked over one of the pieces. The rock rolled into a strip of sunlight cutting through the blinds. Johnson abruptly noticed structures picked out by the low-angle light like tiny, quilted chains across the surface of the stone. A careful reexamination of many of the drill cores—rocks many previous geologists had handled without comment—showed the structures were common across the samples.

Johnson speaks cautiously about the structures and has yet to publish his findings on them formally. But he thinks they might be some type of colony-living eukaryote of a size significantly larger than the microscopic examples known from elsewhere in the early fossil record.

The fact that Johnson noticed the structures in the drill core samples only by chance has shaken his initial skepticism of El Albani’s work. “Something like the Francevillian stuff, people might have found it already in other rocks and just not seen it,” he says. “It just might be because they haven’t looked at it in the right way.”

The sheer variety of forms is why El Albani is surprised that people could look at them and assume they aren't fossils.

Dealing with material like the Francevillian requires trying to understand a time when Earth looked virtually nothing like the world we know now, Porter says. Much of the history of multicellular life occurred across an abyss of time on what was effectively an alien planet, with environmental conditions that were remarkably different from those of the past 600 million years. These conditions affected life in ways that are still only dimly understood. And the further back in time one goes, the more likely it is that any fossils will be difficult to recognize, to say nothing of categorize.

The temptation for the field to dismiss “fossil-ish” forms as mineral concretions or the product of some other nonbiological process rather than a biogenic one therefore exerts a nearly gravitational pull. “I would imagine they’re probably frustrated [and thinking], ‘Why isn’t everybody already excited about this and coming along with us?’” Riedman says of El Albani and his colleagues. “And we’re just like, ‘We’re stuck on step one, man. We haven’t gotten past the biogenic part.’”

“I don’t know what we need to show to prove, to convince,” El Albani says, his expression hangdog. He’s sitting in his office below a poster of [the cover of a June 2024 issue of *Science* in which he and his team published their discovery of a remarkable trilobite fossil](#). “There’s no trouble with trilobites,” he remarks wistfully. El Albani is not a bomb thrower by nature and is not in a rush to name names. But a visible exasperation creeps in when he discusses the Gabonese specimens, along with a tendency to simultaneously pick at and try to dismiss the wound.

At the end of the day, it is a question not really of belief but of arguments, El Albani says. If his critics believe the Gabonese specimens are concretions, they need to try to prove that rather than

simply asserting it. If they disagree that the rocks contain fossils of eukaryotes, nothing is stopping them from subjecting the specimens to their own analyses. So far he feels that nobody has published any research that takes their conclusions apart point by point and reckons with all the strands of evidence they've marshaled. "If I give my opinion that your iPhone is Samsung," he says, pulling a phone across the desk, "I should explain why!"

Porter, the U.C.S.B. paleontologist, agrees. She's not convinced by the team's arguments for what the Francevillian samples represent —an independent lineage of colonial multicellular organisms, swiftly flowering, swiftly snuffed out. But the idea that they're all just mineral concretions has never satisfied her. If they're concretions, that's something researchers need to affirmatively show, she says. Doing so, after all, would add to the field's knowledge about how pseudofossils form in a way that simply writing them off does not. "We don't want to discourage people from publishing these weird structures that are difficult to understand," Porter says.

"It's fine if they're wrong," Porter says of El Albani and his colleagues. Everyone is offering competing hypotheses, which are always subject to new evidence from the fossil record. In the end, "we'll probably all be somewhat wrong about our interpretation, actually."

Seventeen years after El Albani first stopped to examine a glinting blob in the Gabonese shale, his lab shows no signs of slowing down. There are always more specimens to publish, avenues of research to pursue, dissertations to finish. Members of the group are working on closer comparisons between the different environments preserved in the Francevillian quarry and the Cambrian deposits, between the chemistry of the Gabonese specimens and fossils from the Ediacaran and the Burgess Shale.

They're also digging further into the question of how, precisely, chemistry can definitively distinguish between biological and nonbiological origins for a given specimen. Findings from research like theirs could eventually be used to evaluate rock samples from other planets. In 2020 a team of researchers reported that the NASA Mars Science Laboratory rover Curiosity had photographed millimeter-size, sticklike structures in an ancient lake bed that resembled fossils left by miniature tunnelers on Earth. To date, it's been impossible to disprove nonbiological explanations for their presence. But if a lab could develop a reliable conceptual model for chemically distinguishing between signs of life and nonlife, "you could apply this on Mars or another planet based on the sediment," El Albani says.

Every year El Albani and his team make the trip to Gabon to work the scrape of black stone that reoriented his life. There they comb the flaking shales, prying apart slabs, alert to the glimmer of pyrite or the soft, subtle impression of a circular form stamped in the petrified silt. Sometimes El Albani live-streams the expeditions to French schoolchildren, explaining to them how the cellular revolution that gave rise to them lies far back in the mists of prehistory. Sometimes he bends down to examine a glittering form in the rock. It's probably something. The question, as always, is what.

Asher Elbein is a science and culture journalist based in Austin, Tex. His work has appeared in the *New York Times*, *Scientific American* and *Texas Monthly*.

<https://www.scientificamerican.com/article/complex-life-may-have-evolved-multiple-times>

The Slippery Slope of Ethical Collapse—And How Courage Can Reverse It

Your brain gets used to wrongdoing. It can also get used to doing good

By [Elizabeth Svoboda](#) edited by [Madhusree Mukerjee](#)



Tim Bower

It started with an innocent mistake. Texas entrepreneur Chris Bentley had founded a company to buy drilling rights for oil and gas. He realized that a batch of letters he'd sent to landowners, offering to lease their rights, had incorrect information, including monetary amounts and other details.

But instead of correcting the errors, Bentley doubled down, not wanting to admit his mistake. When the letters failed to secure enough land leases to generate big profits, Bentley tried to make up the difference by sinking his investors' money into new, risky deals, some of which faltered and drained the coffers of his company, Bellatorum Resources. Then, as the company's cash flow dried up, Bentley started putting bogus transactions on the books to

keep his employees paid. He didn't stop until he'd committed \$40 million worth of fraud. "I basically did the age-old 'rob Peter to pay Paul,'" says Bentley, who was recently released from prison into home confinement. "Everything started going downhill."

Moral death spirals such as Bentley's happen in every sphere of public life, from business to local government to the highest levels of political leadership. The deterioration often begins with a small dishonest act—such as Bentley's decision to bluff his way through what had been an honest error—and mounts until it reaches a point of no return. Some escalating crimes are financial; others progress toward human rights violations or worse.

Brain and psychology researchers are delving into how slides down the moral slope begin and what keeps them going. Initially we may be horrified at the thought of lying, cheating or hurting someone. But as we engage in wrongdoing over and over, our brains tend to grow numb to it. It's harder to embezzle or kill for the first time than it is for the tenth.

Yet moral snowballing can also happen in the opposite direction. Surprisingly, just as neural habituation can drive ethical collapse, it can also drive escalating spirals of virtue, in which one honest or brave action makes the next one easier to carry out. And because our brains adapt to repeated behaviors, movement in a given moral direction can persist—making it all the more critical to pinpoint where and how that movement begins.

When we first become aware of an ethical violation, we're hardwired to react much as we might to a steaming cow pie.

Carrying out acts of moral courage, such as dodging roadside bombs to get supplies to Iraqi civilians while in the U.S. Army, helped former Capitol police officer Aquilino Gonell to stand strong during the January 6 insurgency. That harrowing experience, which left him with severe injuries, also gave him the resolve to

speak out about what rioters had done to him and others, although he knew telling the truth could put him in extremists' crosshairs. "I couldn't live with myself remaining silent," he told me.

It's easier for people to act morally when they embrace bottom-line values that they'll uphold no matter what. Gonell's grandfather would remind him, "Never tell lies"—a principle that stayed with him. And once people choose to follow their conscience, they often find that the emotional rewards outweigh the hazards. Those who listen to their better angels not only escape the self-reproach that comes from avoiding what they feel is right action, they may even find **deep purpose and joy** in aligning their actions with their value system—as Gonell did in speaking out. "The more I did it, I used that as mental health therapy," he says. "I can live with myself knowing that I have met the moment in time and now history."

When we first become aware of an ethical violation—say, a co-worker's embezzlement scheme or a secret inner desire to hurt someone—we're hardwired to react much as we might to a steaming cow pie. In a 2020 study by researchers in Switzerland, people who'd just thought about an ethically thorny situation **reacted more intensely** to rank smells than control participants did. Areas of their brains that processed physical disgust, such as the anterior insula, were also more active, hinting that the moral violation hit them like a whiff of manure. "Disgust and moral disgust are uniquely connected," says neuroscientist Gil Sharvit, the study's lead author.

Still, if neuroscientists untangling the complex processes that govern moral decisions have reached any overriding conclusion, it is that **no single brain circuit** dominates such choices. In scans using functional magnetic resonance imaging (fMRI), a wide network of brain areas activate when people reach major decision points, reflecting the broad range of social, emotional and instinctual factors that weigh into each moral choice.

Along with the automatic recoil, the brain's fear-processing amygdala activates as people consider the risks they run by doing something wrong. Contemplating an ethical stand may also evoke fear—of retribution. As reflection continues, however, moral decision-making evolves into an inner debate in which logic tempers the quick initial responses. [Multiple areas of the brain, including the prefrontal cortex](#), a general decision center, help to regulate instinctive reactions such as fear and disgust, putting them into a larger context. The anterior cingulate cortex, along with the anterior insula and nucleus accumbens, assesses the net reward or penalty a morally fraught decision will incur and manages emotions tied to the decision, making it feel more palatable—or not. This synthesis can make the way forward seem clearer.

Although these basic neural networks are similar from person to person, factors such as someone's personal history, what feels rewarding to them and what's happening around them can profoundly alter someone's mental processing and subsequent moral response. When Bentley reflects on what kicked off his bogus transaction scheme, he keeps coming back to his ravenous appetite for risk—one he honed during his service with the U.S. Marines in Afghanistan, where members followed the creed of “improvise, adapt and overcome” at any cost.

In the field, Bentley was responsible for getting necessary battle supplies to teams in far-flung locations. He once went off script by enlisting a team of Afghan interpreters to drive out in a pickup truck to drop off gear for U.S. soldiers. “If they would've stolen it and never come back, which I trusted them not to do, it would've been my ass,” Bentley says. “But I saw it as the only option to get the teams what they needed.” The mission's success cemented Bentley's belief that audacious risks confer outsize rewards.

When people develop a slot puller's zest for risk and personal gain, that acquired swagger affects not just what they're willing to put on the line but what kinds of moral choices they make. In [a study](#)

[published in 2024 by researchers in India](#), people who'd grown used to risky gambling games proved more willing to make moral choices others might find loathsome, such as (theoretically!) pushing one person in front of a speeding trolley to save others. This result showed that instead of relying on absolute moral rules to guide their behavior, such as "Never actively kill someone," risk-tolerant gamblers tended to make moral decisions based on more utilitarian cost-benefit calculations. What Bentley hoped to gain through his scheme at Bellatorum—recognition, profits, a chance to give other veterans opportunities—loomed larger in his mind than any absolute moral value.

Just as we adapt to lingering stenches, we seem to adapt to initial wrongdoing in ways that prompt us to go further.

As he weighed whether to go ethically rogue, Bentley says, he also felt under the gun. However people might describe their highest values in moments of calm, those values are prone to precipitous collapse under pressure. As a scrappy small-business owner, Bentley felt immense pressure to deliver on his clients' expectations, and he didn't see any room for error. "My fund didn't allow for losses," he says. "We literally had a zero-mistake structure." He and his team had worked late nights for a week to prepare and deliver 5,000 offer letters to landowners, and when he discovered those letters were defective, he was so horrified at the thought of backpedaling that he scrambled to cover up his mistake. "I was definitely in panic mode," he says.

"When we feel afraid, our bodies are thinking we're in a life-death situation," says ethical consultant Brooke Deterline, founder of the Courageous Leadership consulting firm. In this frenzied state, the body floods with stress hormones such as cortisol, which are known to interfere with higher cognitive functioning. Cognitive shutdown may help explain why people who are told to hurry because they're running late, for instance, assist those in need less often than those who aren't feeling pressured. The Socratic axiom

“To know the good is to do the good” can break down in the heat of the moment.

At least initially, when people lie, steal or hurt someone, they often seethe with self-disgust. The cow-pie stench is coming from inside the house, and its presence is intolerable. The first time that former WorldCom employee Betty Vinson made a multimillion-dollar accounting adjustment to inflate the company’s profits, she felt such dread that she approached her bosses and told them she was resigning.

But just as we adapt to lingering stenches, we seem to adjust to initial wrongdoing in ways that prompt us to go further. In [an Arizona State University experiment](#) in which 73 college students solved math problems, participants could earn a small amount of cash for each correct answer, but they also had chances to take more than they’d earned from an envelope. When people’s opportunities to steal started off small (just a few cents) and grew ever larger, twice as many people stole from the envelope as did people who stood to gain the same amount every turn by cheating.

Organizational psychologist David Welsh, the paper’s lead author, wasn’t surprised by the results. He’d done the study in part because he couldn’t get Stanley Milgram’s work out of his mind. In that classic experiment, participants dubbed “teachers” were told to give electric shocks to “students” who answered questions wrong.

Milgram’s most talked-about finding was how often people obeyed corrupt orders. But what struck Welsh was the moral habituation that appeared to be taking place. “They started out instructing the participants to deliver these very small shocks,” he says, “and then the shocks got larger and larger.” If “teachers” expressed doubt about what they were doing, experiment leaders [urged them to continue](#) with phrases such as “You have no other choice; you must go on.” With such moral coercion easing their complicity, people who’d never have dreamed of zapping anyone with 450 volts

became all too willing to comply when they worked up to that number gradually.

Gradual moral adaptation occurred even in the lower-stakes scenario Welsh set up, where only cash, not people's health, was at stake. An initial, small transgression seemed to embolden participants to commit a bigger one the next time. As soon as people start telling themselves it's not a big deal to massage the numbers on their balance sheet or to take credit for someone else's work, conditions are ripe for a slippery-slope moral descent, Welsh says. "Once they're in that mindset of rationalizing their bad behavior, it becomes that much easier to do it again and again and again."

Researchers at University College London [have described](#) one biological basis for this habituation. While in an fMRI scanner, study participants played a game in which they could enrich themselves by deceiving others. The more people lied to other players, the more exaggerated their lies were likely to be the next time around. These habitual liars also showed reduced activation in the brain's amygdala, which is involved in emotional arousal—and the lower their amygdala activation, the more flagrant their lies were in the next round of the game. The researchers believe gradual neural adaptation is at play: the more times people lie, the less emotionally distressing lying feels, which allows for increasing comfort in dangerous moral waters.

Vinson fell prey to this effect as she got drawn into WorldCom's multibillion-dollar corruption scheme. Although she wanted to resign after her first fraudulent transaction, her boss talked her out of it, telling her she wouldn't be asked to do anything else untoward. So she stayed on, and when executives asked her to perform another bogus transaction, she debated leaving again but decided not to. Soon, Vinson's transactions became regular quarterly tasks, as routine as starting the coffee maker, even though they were staggering [in size](#)—up to \$941 million.

What might have eased Vinson's adjustment to grand-scale fraud was the number of people around her who seemed to be fine with it. Peer pressure warps reasoning skills in predictable ways. In psychologist Solomon Asch's [classic experiments](#), some participants consistently reported that two lines on a card were the same length when others in the room insisted this was the case. It didn't seem to matter that one line was clearly longer than the other.

In some groups, threats from the top amplify members' willingness to abandon their values. The energy company Enron dismissed employees who were exposing or questioning its suspect financial practices. Once this corrupt conformity takes hold, those who state the truth become outliers, as superfluous as runts of the litter—and as vulnerable to being left behind.

As Bellatorum's CEO, Bentley never felt anyone was forcing him into an ethical corner. And although his fraudulent transactions became routine, he says he never really grew numb to what he was doing. "I was personally deteriorating," he says. "I was drinking so much to self-medicate for living a lie." What stopped Bentley from admitting his crimes—which, on one level, he desperately wanted to do—was that he'd convinced himself his wholesale fraud was the lesser of two evils. The way he saw it, his choices were these: confess and shut Bellatorum down, devastating employees and investors who'd trusted him, or continue his money-funneling scheme so he could write paychecks to his employees, many of whom were retired combat veterans.

Our brain's propensity for habituation implies that the early stages of a moral trajectory may be the most crucial.

"I couldn't bring myself to just shut down a business and let it fail after I had brought in so many people from around the country," he says. Lose-lose choices like this can prompt intense distress and inner wrestling. In a 2016 study led by Natalie Claes, then at the

University of Leuven, participants [deciding between two bad options](#) took longer to choose than those who had at least one good option, and they also reported feeling more fear during the process.

Physician Catherine Caldicott, who runs medical training programs in Florida, often encounters doctors caught in “lesser of two evils” binds. If they’re asked to list past criminal convictions when applying for or renewing a license to practice, they may tell themselves that lying is better than getting their application denied and being unable to help patients. When people reframe immoral or complicit acts as noble, they’re prone to go down the moral slippery slope, in part because they’ve locked onto the narrow idea that they can contribute more by going against broader values and professional principles. “They do not realize that there may be other choices available or more morally defensible ways forward,” Caldicott says. “Their ability to think rationally is impeded.”

Although initial wrongdoing can escalate over time, the converse is also true. When people respond bravely in fraught situations, courage becomes progressively easier as the brain continues to adapt to rising discomfort.

A study by researchers in Israel [demonstrated this adaptation](#) in a dramatic way. Members of the study’s experimental group, all of whom were afraid of snakes, entered an MRI scanner room where a five-foot-long corn snake was curled up just outside the scanner on a platform on a conveyor belt. Researchers told them their job was to get as close as possible to the snake and to overcome, as best they could, any fear they might feel.

Participants had access to control buttons in the scanner that they could use to inch the snake on the conveyor belt either closer to them or farther away, and in each round of the experiment, they chose one of these two options. When they opted to bring the snake closer, something remarkable happened: They showed more activity in a prefrontal cortex region called the subgenual anterior

cingulate cortex, which is involved in regulating emotions, as well as the right temporal pole, which helps to shape behavioral responses. At the same time, activity in the amygdala, which processes fear and threat, diminished.

In short, it appeared that when people decided to bring the snake closer, their brain kept enough of a lid on the fear response to allow them to carry out their plan. Once they adjusted to the new situation, many felt bold enough to continue approaching the snake.

Well-established neuroplasticity findings suggest that small acts of moral courage can similarly beget acts of greater courage. “We can choose to bring the snake in a little bit closer,” says clinical psychiatrist Christian Heim, who is affiliated with the University of Queensland. “Or we can choose to say, ‘No, that’s it. That’s all I’m capable of. I’m going to push it away.’”

Former Capitol police officer Gonell has gotten comfortable bringing the snake closer in. At age 12, he immigrated to the U.S. from the Dominican Republic, and when he returned to his home country for visits, his grandfather Fillo would remind him to live his life with integrity.

Still, Gonell sometimes hesitated to act on his values. Conscious that his accent marked him as an outsider in his Brooklyn neighborhood, he was wary of making waves. But when the U.S. Army later shipped him to the Middle East for Operation Iraqi Freedom, he put his developing courage to the test, volunteering to drive supplies to Iraqi schools and U.S. troops despite the constant threat of roadside bombs. He received military honors for his bravery, including the National Defense Medal.

Serving as a Capitol police officer on January 6, 2021, brought Gonell to a key decision point. Defending the building in a gas mask and riot gear, Gonell battled dozens of insurgents and sustained multiple injuries, including chemical burns and a

smashed foot that required surgery to repair. As he recovered, many people—and even some members of Congress—started spreading misinformation about what had happened at the Capitol that January day. Some said the incursion had been an antifa-led protest, and others insisted the insurrectionists had been peaceful.

Following his grandfather's dictum, Gonell resolved to set the record straight. "This is something in our history that shouldn't be kept quiet," he says. He agreed to talk to CNN about what he had seen and heard on January 6: who he had encountered, what they had done to him and other officers. He was afraid of how people watching on TV, especially riot supporters, would respond, but he went through with the interview anyway.

That first appearance led to a series of other public engagements, including testifying before Congress. Each time Gonell told the truth openly, doing so felt a little bit easier, despite the danger he knew he could face. For the most part, he says, his experience speaking up has been positive: "I could look at myself in the mirror and look at my son and say, 'Hey, I did the right thing.'"

Compared with Bentley's actions, Gonell's might seem to exist in a separate moral universe. Yet from a neural standpoint, moral deterioration and moral escalation are like trains running on parallel tracks in opposite directions. Similar neural structures of reward and habituation underlie them both. And just as similar brain processes evoke moral and physical disgust, related neural pathways evaluate both morality and beauty. [The same brain region](#) —the medial orbitofrontal cortex, which processes reward—evaluates both the attractiveness of a face and the virtue of a planned action. It's no surprise, then, that moral ventures can be gratifying in much the same way as creating a work of art. People who are more moral, as judged by their peers, also have an enhanced sense of well-being, according to a [cross-cultural study](#) published earlier this year.

Further, people adapt to the behaviors they carry out frequently, which may make more extreme versions of those behaviors more likely. They also tend to repeat behaviors that they feel benefit them, whether these rewards are external (staving off financial collapse) or internal (the satisfaction of speaking truth to power).

Our brain's propensity for habituation implies that the early stages of a moral trajectory may be the most crucial. "All the neural networks that we have are changeable," Heim says. "If we use [them], they become stronger. If we don't use them, they become weaker." Once people understand how the brain gets accustomed to repeated behaviors, they can exercise more choice at the outset, asking themselves what kinds of actions they want to get comfortable with, what kind of beauty or integrity they want to strive for. Although the amygdala will almost certainly emit fear signals in situations that call for courage, what's important is suppressing those signals enough to make virtuous action possible —and appreciating the inherent rewards of doing so.

Heim tries to encourage such habituation in his psychiatric practice. Because integrity can support mental well-being, he sometimes gives clients homework assignments such as telling a work supervisor they feel uncomfortable with a particular task. Heim's objective is to help clients hold their own moral line, so he's careful not to make these assignments too difficult. By demonstrating to themselves that they can act courageously, his clinical experience shows, people will reinforce mental pathways that will help them generate positive momentum and avoid moral collapse.

Self-reflection can play an important role in shifting the brain's reward calculus and, by extension, help people make ethical decisions. In a 2023 [study of moral judgment](#) carried out in China, participants received eight weeks of mindfulness training, including meditation. Compared with a control group, those who received the training were less motivated to earn money if doing so

would harm others. That altered preference showed up in their behavior. Those in the training group were not as open to giving someone an electric shock in exchange for cash, whereas control group members grew more inclined to deliver the shock over time.

Mindfulness practices may affect moral judgment in part because they promote a more objective outlook. It's often easier for practitioners to take someone else's view of a situation, which compels them to steer clear of harming others. Through skillful perspective taking, "I think we can always save ourselves," Sharvit observes: identifying with others helps people guard against moral numbness and the negative spiraling that follows. "You won't get habituated," he adds. "You can connect."

At an institutional level, one way to ward off downward moral slides might be to increase the penalties tied to each stage of moral descent—say, by announcing zero-tolerance antifraud company policies—and to underscore the rewards of holding the moral line. Leaders of organizations can, for instance, swiftly address transgressions and help employees get comfortable with admitting mistakes. In a [Maastricht University study](#), participants whose bosses showed ethical leadership engaged in fewer corrupt acts such as offering bribes. Generally speaking, fraud and cover-ups seem less enticing in ethical workplace cultures, and telling the truth feels like an obligation, not an act of career sabotage.

Once people decide to act with integrity, their resolve is often socially contagious. When researchers told enrollees in the Milgram experiments to shock "learners" for answering questions wrong, people who saw others refusing to administer shocks were [more apt to refuse](#) as well. And researchers at Eastern Michigan University and elsewhere report that in work groups where members openly endorse ideals such as honesty and fairness, individual employees are often more likely to [speak up about moral violations they see](#), perhaps as the result of virtuous peer pressure.

Had Bentley sought to modulate his own reward calculus before starting Bellatorum, he most likely never would have gotten in as deep as he did. He now says that, despite his fear, he should have admitted his mistake the moment his incorrect offer letters went out to landowners. That would have dinged the company's reputation, but Bentley thinks that at that early stage, he could have bounced back. "I could've downsized to a very small crew and probably stayed in business," he says. "Now I'm betting I've burned the bridges beyond all repair."

Bentley also suspects that an unbending set of "flat-ass rules"—a term he borrowed from Operation Iraqi Freedom general James Mattis—could have saved him from becoming a stranger to himself, and research bears out his hunch. The stronger people's advance intentions are to engage in certain types of behavior, a University of Sheffield meta-analysis shows, the more [apt they are to follow through in real life](#).

Psychologists such as Zeno Franco of the Medical College of Wisconsin suggest cultivating what he calls the "heroic imagination": our individual capacity to consider ahead of time what we'll do in situations that call for moral courage, what values we will stand behind even under extreme pressure. In this kind of "What would I do?" scenario, the brain's frontal cortex helps people anticipate how they will feel when they make certain moral choices, and those predicted feelings can influence their decisions in the long run.

When he started down the moral slope, Bentley did not know that living a lie would end up eating away at him like acid. "I would be driving over one of the high on-ramps that are so common in Houston and just think that I could drive my truck over the side," he says. Finally, able to bear the guilt no longer, he turned himself in to federal officials in April 2021.

As he nears the end of his five-year sentence, Bentley still hopes to bend his arc toward redemption. He has written a memoir that frames his moral decline as a cautionary tale and shows how turning away from the truth led him to hunger more after that elusive ideal. “Never compromise your integrity for anything,” he now tells others, “not even when you think it’s essential to your survival.”

As for Gonell, he continues to speak and write about what happened at the Capitol on January 6, as well as about what he sees as ongoing threats to the rule of law in the U.S. He still receives threats from the public but remains undaunted. “What else you got? I’ve gone through war, I’ve been back, I’ve been injured, I’ve been ridiculed,” he says. “I’m not concerned about my life, even now, when some people say, ‘Hey, you should be careful.’”

Having considered how far he would go to ensure that truth prevails, Gonell has decided there’s basically no limit because the principle matters more to him than his own safety. Thirteenth-century theologian Thomas Aquinas saw integrity as *synonymous with beauty* that transcends outward appearances, and striving toward such a moral ideal gives people a profound sense of meaning in life. For Gonell, as for others on a similar path, the inner rewards of integrity more than outweigh the costs.

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<https://www.scientificamerican.com/article/what-brain-science-reveals-about-ethical-decline-and-moral-growth>

Which Anti-Inflammatory Supplements Actually Work?

Experts say the strongest scientific studies identify three compounds that fight disease and inflammation

By [Lori Youmshajekian](#) edited by [Josh Fischman](#)



Capsules of omega-3 fatty acids show some of the best evidence as anti-inflammatories.
Mensent Photography/Getty Images

Inflammation has two faces. It can be short-lived like the swelling after a twisted ankle or a two-day fever when you get a mild flu, both part of the healing process. Or it can be a longer-lasting and more damaging affliction—chronic, low-grade inflammation that lingers in the body for years without obvious symptoms, silently harming cells. A steady stream of studies has connected this type of chronic inflammation to many serious conditions, including Alzheimer's, heart disease, some cancers, and autoimmune illnesses such as lupus.

These findings have begun to reframe how scientists think about disease and some of its causes. They've also created a booming market for supplements promising to lower chronic inflammation.

These pills, capsules and powders are projected to become a \$33-billion [industry by 2027](#), offering consumers a sense of control over a complex and confusing ailment. Although thousands of products claim to “support immunity” or “reduce inflammation,” most lack solid evidence.

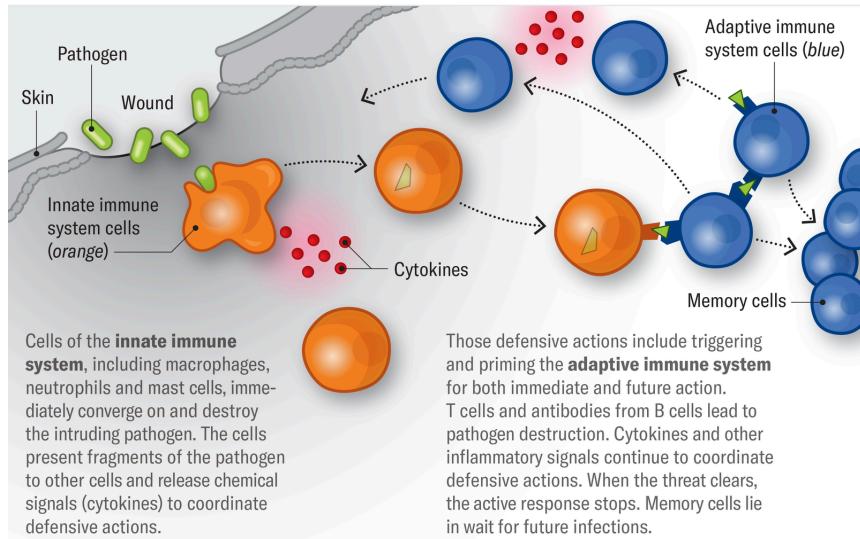
Chronic inflammation is damaging because it involves immune system cells and proteins that typically fight short-term battles against bacteria, viruses, and other pathogens. But when these immune system components stay activated for years, they begin to hurt healthy cells and organs. They are intended to break down invading microbes, but over time their ongoing activity can harm blood vessels, for instance, by damaging normal cells that make up the vessels’ inner linings or promoting the growth of plaques. That can lead to clots that interrupt or cut off blood flow, increasing the risk of heart attacks and strokes.

We reviewed dozens of studies and spoke with researchers to find out whether any supplements demonstrate anti-inflammatory activity not just in laboratory animals and cultured cells but in human trials. Just three compounds, it turns out, have good evidence of effectiveness: omega-3 fatty acids, curcumin and—in certain ailments—vitamin D.

Good vs. Bad Inflammation

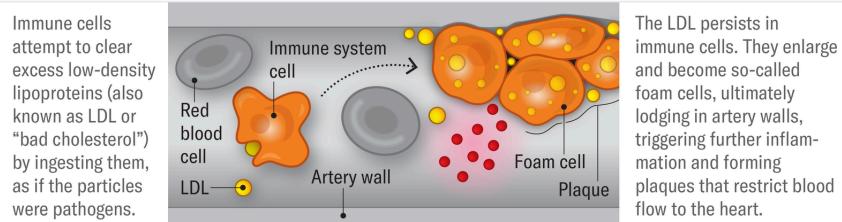
Inflammation is commonly associated with an immune system response to microbial infection or tissue injury. For example, when bacteria enter your body through a cut in your skin, some of your body's immune cells—part of what is called the innate immune system—gather at the site to ward off invaders. They also release signaling proteins called cytokines that call in reinforcements. These additional cells and proteins are known as the adaptive immune system, and they identify and destroy remaining bacteria (*below*). This activity, called **acute inflammation**, is often accompanied by short-term swelling, a fever or mild pain. The acute response generally lasts only hours or days. Similar events occur when you twist an ankle, in which case proteins released by damaged muscle and tendon cells trigger the immune system reaction.

ACUTE INFLAMMATORY RESPONSE EXAMPLE: MICROBIAL INFECTION



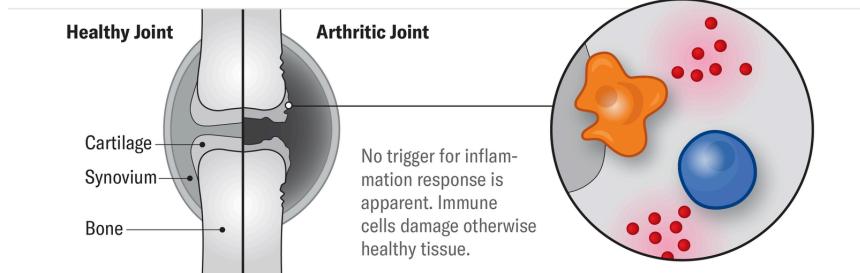
Some forms of inflammation can do more harm than good. In **chronic inflammation**, the same immune system cells are activated, but the response lasts for months or years. In some cases, the threat persists in the system and cannot be cleared by immune cells. For example, macrophages engulf cholesterol particles in the bloodstream. But those particles persist within the macrophages. This leads to bulky plaques on arterial walls and feeds into continued inflammation (*below*).

CHRONIC INFLAMMATORY RESPONSE EXAMPLE: HEART DISEASE



In other cases, including autoimmune diseases such as rheumatoid arthritis, there's no apparent threat to the body. The inflammatory response targets otherwise healthy organs and cells for an unknown reason. Over time the immune cells can damage healthy tissues, and the ongoing inflammation exacerbates symptoms.

CHRONIC INFLAMMATORY RESPONSE EXAMPLE: RHEUMATOID ARTHRITIS



To track chronic inflammation—and to learn which anti-inflammatories can reduce it—researchers measure levels of specific cytokines and molecules released by organs during an immune response or an infection.

What is good evidence? We looked for consistent results across several studies that scientists described as large and well designed. Many of the more convincing trials focus on biomarkers that researchers use to track inflammation in the body. These include C-reactive protein (CRP), a molecule produced by the liver when inflammation is active, and cytokines, which are chemical messengers such as interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF- α), both secreted by immune and fat cells.

Still, interpreting these markers isn't straightforward. "We don't have a universally accepted or standardized measurement," says Frank Hu, chair of the department of nutrition at Harvard University. And inflammation involves hundreds of different types of cells and many signaling pathways, adds Prakash Nagarkatti, director of the National Institutes of Health Center of Research Excellence in Inflammatory and Autoimmune Diseases at the University of South Carolina. This complexity makes it difficult to prove that any supplement works consistently.

The compounds that do show promise will not cure cancer or halt dementia. But they may help quiet the kind of underlying inflammation that has been tied to risks of illness.

OMEGA-3 FATTY ACIDS



Herring is a rich source of omega-3 fatty acids.

Jim Sugar/Getty Images

Among the hundreds of supplements tested for their effects on human health, omega-3 fatty acids are supported by some of the most compelling evidence. And scientists understand why they work. Two of the main types of omega-3s are eicosapentaenoic acid and docosahexaenoic acid, better known as EPA and DHA. The body metabolizes them into signaling molecules that block the production of certain cytokines and disrupt the **nuclear factor κB pathway, which governs the expression** of genes tied to inflammation.

Multiple studies suggest that omega-3 supplements can reduce markers of chronic inflammation, Hu says, especially among people with underlying health conditions. A large, carefully controlled trial called VITAL (officially the Vitamin D and Omega-3 Trial), which followed more than 25,000 adults for about five years, found that omega-3 supplements **slightly reduced** CRP in people who rarely ate fish—fish is a natural omega-3 source, so these people were getting almost all their omega-3s from the supplements. The omega-3 supplements also were associated with a 40 percent reduction in heart attacks among those consuming the least fish. “The people who benefit the most from these supplements are people who start out with lower intake,” says JoAnn Manson, an endocrinologist at Harvard Medical School who co-led the study.

Smaller trials have suggested that omega-3 supplementation can reduce certain markers of inflammation—**TNF-α, IL-6, CRP and IL-8**—especially in people with conditions such as **heart failure, Alzheimer’s** and **kidney disease**. One 2012 trial found that small amounts—about 1.25 or 2.5 grams per day—lowered IL-6 levels by 10 or 12 percent, respectively, over four months. A similar group got a placebo instead, and their IL-6 levels increased by 36 percent during that period.

Taking omega-3 fatty acid supplements was associated with a 40 percent reduction in heart attacks among people in a trial who ate the least amount of fish.

But the evidence across various trials is hard to compare. “There is still a question regarding which is the optimal dose and the optimal duration because different studies have used different doses,” Hu says. And in healthy people, who have low baseline inflammation, there might be little room for improvement.

VITAMIN D



Egg yolks contain some vitamin D.
Masanyanka/Getty Images

Rigorous trials have [debunked the once popular idea that vitamin D is a wonder drug](#) for everything from breast cancer to diabetes. For a few autoimmune conditions, however, the vitamin can be helpful. In the VITAL trial, people who took vitamin D daily for five years had a [22 percent lower risk](#) of developing autoimmune diseases such as rheumatoid arthritis, psoriasis and lupus. “High-dose vitamin D has the effect of tamping down inflammation,” Manson says. “So conditions that are really directly related to inflammation may benefit.”

Lab studies have suggested that vitamin D [may interfere with molecular pathways](#) involved with inflammation, in addition to [suppressing the production](#) of proinflammatory cytokines. And in a handful of clinical trials in people with autoimmune conditions, vitamin D supplementation appeared to reduce levels of proinflammatory cytokines [such as TNF- \$\alpha\$](#) , as well as CRP. In [one small study](#) of women with type 2 diabetes, a high dose—50,000 international units (IU) every two weeks—reduced CRP. It also increased levels of IL-10, an anti-inflammatory molecule.

A [separate study](#) in women with polycystic ovary syndrome (PCOS) found that a combination of vitamin D and omega-3 fatty acids helped to lower CRP levels. And two analyses that grouped together results from several studies back up the idea that [the vitamin can cause a significant](#), though small, [reduction in CRP](#). [Another trial](#) in women with PCOS found that a daily dose of 3,200 IU of the vitamin improved patients' insulin sensitivity and liver function. It didn't affect inflammatory markers, however.

Other studies haven't found consistent effects. The VITAL study reported that people who took vitamin D [saw a 19 percent drop in CRP levels](#) by the two-year mark, but this difference disappeared by the fourth year. Whether that two-year dip in inflammation translates into long-term benefits remains unclear, the researchers note. Even then, the findings may also depend on baseline levels. Most people in the VITAL study started with normal levels of vitamin D, Manson says. "People who are already getting reasonable intake may not benefit further from the supplement," she says. A review of other trials looking at inflammation-related biomarkers such as CRP, IL-6 and TNF- α found that vitamin D supplementation at several different doses [didn't have a big effect](#).

As with omega-3s, the varying doses in the different trials may be behind the inconsistent results. [Very high weekly doses](#)—40,000 or 50,000 IU—may be necessary. (The recommended daily vitamin D

intake for adults is 600 IU.) But high doses carry their own risks, such as too much calcium in the blood.

Although the findings on autoimmune illnesses are intriguing, the American College of Rheumatology still has a conditional recommendation [against the use of supplements](#), instead advocating that people make dietary changes to try to get the recommended vitamins and nutrients from food. Inflammation is central to illnesses such as rheumatoid arthritis, says Arthur M. Mandelin II, a rheumatologist at Northwestern University's Feinberg School of Medicine, but he is interested in vitamin D only as a therapy for patients with demonstrated deficiencies.

CURCUMIN



The spice turmeric contains curcumin.
banusevim/Getty Images

The pigment that gives turmeric its yellow color, curcumin, is another promising compound for fighting chronic inflammation. The substance seems to interfere with the nuclear factor κB pathway, “the apex of inflammatory cascades in the body,” explains Janet Funk, a professor of medicine and nutritional sciences at the University of Arizona, who has [evaluated hundreds of human trials](#) on the compound.

Funk's review found that the most convincing evidence for curcumin's anti-inflammatory activity was among small clinical trials. People in those trials had preexisting conditions such as metabolic disorders and osteoarthritis. In a few cases, curcumin's effects resembled those of over-the-counter anti-inflammatory drugs such as ibuprofen. "These small trials—and there are a lot of them—all sort of point to it probably being beneficial," Funk says.

The caveats in Funk's language, however, reflect the ambiguity of other results. A large Canadian trial found no measurable benefit for inflammation in people who were taking curcumin after surgery, and other trials have been inconclusive. One reason for the inconsistency is curcumin's bioavailability: the substance is poorly absorbed in the gut, rapidly metabolized and quickly cleared from the body. Some supplement manufacturers encase curcumin in nanoparticles to improve its absorption, but these formulations aren't always used in clinical trials, nor are they consistently available over the counter.

Some commercial turmeric and curcumin powders have even been found to contain harmful contaminants such as lead. "People buy turmeric powder based on its color," Funk says. "Partly to make it a more beautiful color, [manufacturers] add lead chromate."

Other compounds such as flavanols in green tea and dark chocolate or resveratrol in red wine are often promoted as anti-inflammatory agents. But their supporting evidence is weaker, Hu says. They can be hard for the body to absorb, which limits their effectiveness. In the case of resveratrol, the compound is metabolized and cleared so quickly it's unlikely to have any true impact. And even though a recent trial of cocoa flavanols found a promising effect on cardiovascular health, possibly because of reduced inflammation, any benefit might be outweighed by the many extra calories one would consume if they got the compounds by eating chocolate.

Supplements aren't regulated like drugs. The U.S. [Food and Drug Administration](#) doesn't require supplement companies to prove that their products improve health, unlike pharmaceuticals. So there's little financial incentive for these companies to run rigorous clinical trials because, as Funk asks, "What if they find out it doesn't work?"

Such trials would also be difficult to run. Supplement ingredients can vary from batch to batch, especially for botanically derived products, in which concentrations depend on where the plants are grown and how the crucial components are extracted. Even when trials are well designed, they can come up against ethical challenges. "You cannot really preselect people on the basis of being deficient or profoundly deficient in these essential vitamins," Manson says, "because once you identify them as being profoundly deficient, you really should be treating them" and not giving half of them placebos in a multiyear trial.

Still, the appeal of supplements is obvious. We all want simple solutions to complex medical problems, especially as we learn more about the damaging effects of chronic inflammation on health. "The patient who spends a good deal of the visit focusing on diets and supplements is also that patient who's very fearful of medication," says Mandelin, the Northwestern rheumatologist. "They're ready to write [the names] down as if there is some magic answer, and unfortunately there isn't."

Instead experts recommend what good medical studies have shown to work: a healthy and balanced diet. Mediterranean-style diets, which are rich in vegetables and whole grains with some fish and poultry, have especially been shown to reduce chronic disease and to promote good health. Regular physical activity helps, too. "Many people think that they can just take a dietary supplement, pop the pill, and that replaces a healthy diet," Manson says. "That is not at all the case."

Lori Youmshajekian is a science journalist who reports on consumer health, environmental issues and scientific misconduct. She holds a master's degree in science journalism from New York University and has written for *National Geographic*, *Wired* and *Retraction Watch*, among other outlets.

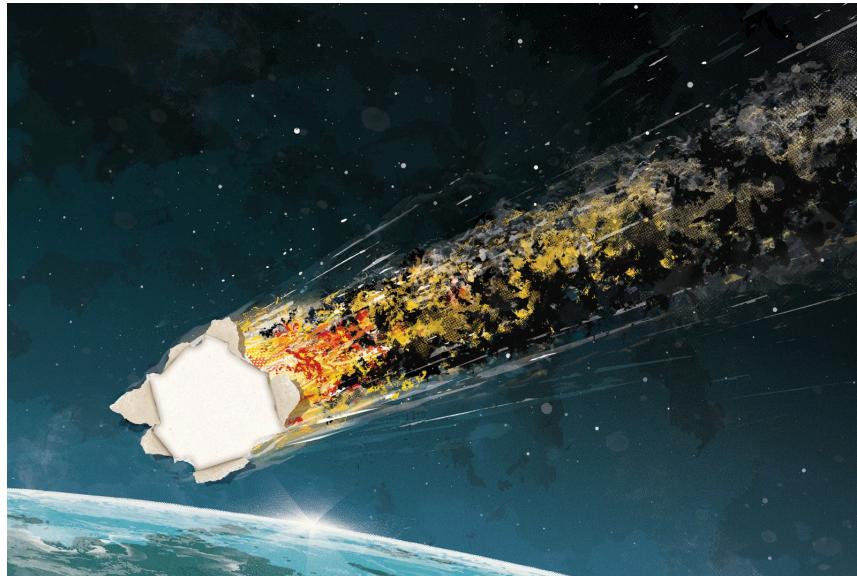
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The Sordid Mystery of a Somalian Meteorite Smuggled into China

How a space rock vanished from Africa and showed up for sale across an ocean

By [Dan Vergano](#) edited by [Clara Moskowitz](#)



Mark Smith

Millennia ago a piece of the sky fell toward East Africa, streaking overhead, born of an ancient collision of asteroids. The meteorite landed, probably with more of a thud than a boom, in a river valley where camels now forage near the village of El Ali in Somalia.

Known locally as *Shiid-birood* (“the iron rock”), the El Ali meteorite is 13.6 metric tons of iron and nickel. For generations it rested in the ground some 24 kilometers (15 miles) outside the village, becoming a landmark that was featured in [folklore](#), [lullabies](#) and [poems](#). According to one story, the region had been a green paradise until its inhabitants stopped believing in Waaq, the local god, who punished them with volcanic stones, leaving behind the El Ali meteorite as a reminder of their folly. Over the centuries

people hammered the brown rock from the heavens with stones, banging off flakes of cold iron, or used it as a whetstone. Children pretended to ride it like a horse.

Now, though, the El Ali meteorite is gone. Shaky cell-phone videos suggest the rock is being stored in China, where sellers hope to hock it for millions, either whole or in pieces. How did it get there? The journey of the ninth-largest [meteorite](#) in the world involves lies, smuggling and possibly death. Mystery surrounds its departure from its landing site, a lawless region of Somalia, one of the poorest and most contested places on the planet. In August, a Somali cultural minister asked the UNESCO World Heritage Center to recognize the meteorite as part of the country's patrimony, calling for its return in a statement. The fate of the cosmic cannonball is now anyone's guess.

For centuries the El Ali meteorite, a brownish, pitted boulder some two meters wide and one meter tall, went unnoticed by anyone but locals. Village elders say that about 80 years ago, during World War II, the Italian army suggested removing it for study. Later, United Nations peacekeeping forces eyed it, and so did militias after the 1991 collapse of Somalia's government. They were all drawn by the mystery of *Shiid-birood*, seeing the curious iron rock sitting on the outskirts of a Somali camel-herder village as an object of research—or at least scrap metal. But locals stopped all these extraction attempts. Then, in September 2019, opal hunters scouring the surrounding desert reported the meteorite to a nearby mining outfit—the Kureym Mining and Rocks Company, a Somalia-based firm led by five traders and businessmen in Mogadishu, the country's capital. Using a hammer, prospectors chiseled a 90-gram sample from the space rock they [renamed](#) “[Nightfall](#)” and sent it off to Nairobi for analysis. Those samples confirmed for the first time that the meteorite was indeed extraterrestrial, comprising some 44 percent iron and 45 percent nickel.



This is where the story of the El Ali meteorite turns murky—and grim.

We know from news reports and human rights groups that sometime in February 2020, the stone was removed from the village El Ali. The area is largely controlled by [al-Shabaab](#), or “the Youth,” a militant affiliate of al-Qaeda that has been designated a terrorist organization by the U.S. government. Al-Shabaab ruled Mogadishu into the late 2000s but was then dislodged by African Union forces. The organization is responsible for numerous bombings and killings, including [the massacre](#) of 148 people at Kenya’s Garissa University in 2015, as well as an “[extensive racketeering operation](#)” in Somalia, according to the Council on Foreign Relations. Given the group’s authority in the region, it seems likely that it orchestrated the looting of the meteorite or at least assisted in it.

No one knows exactly what happened after the meteorite was carted out of El Ali, though. News reports from the time say the rock was “[forcibly taken](#)” by al-Shabaab. They describe large cranes excavating the stone amid gunfights that reportedly left several [people dead](#), including civilians. Some accounts put [the toll even higher](#), with local leaders describing in news reports two full-scale firefights—one during the dig and one as the meteorite was trucked away—between al-Shabaab and fighters from the clan-based Ma’awisley militia. Some leaders say the fighting included

beheadings. But Abdulkadir Abiikar Hussein, a geologist at Almaas University in Mogadishu, calls the reports of bloodshed “exaggerations.”

Whichever way the extraction went down, most accounts agree on what happened next. Militia members drove the meteorite to the nearby town of Buq Aqable, then reportedly sold it to the Kureym mining company for \$264,000.

From there the truck carrying the stolen space rock started to make its way toward Mogadishu, but it was detained on the drive. Somalian government officials [impounded](#) the vehicle for inspection, Hussein says, after security forces on the road into the city grew suspicious of the big metal boulder in the back of a truck and arrested the lone driver. They sent the meteorite to a warehouse near the local airport. At that point, in late February 2020, the government’s mining ministry called in Hussein. He measured, sampled and tested the meteorite in the warehouse, making the first characterizations of the cosmic rock, which would appear later in scholarly descriptions.

Somehow, however, the meteorite was released. By December 2020 *Shiid-birood* was back in the hands of Kureym, although the details of the transaction are unclear (Hussein and others say it was corrupt). Kureym representatives declined to comment on the record for this article.



The El Ali meteorite's original landing site in Somalia is a dry valley without much vegetation. From "El Ali Meteorite: From Whetstone to Fame and to the Tragedy of Local People's Heritage," by Ali H. Egeh, in *Meteoritics and Planetary Science*; June 12, 2025

Scientists outside Somalia first learned about the meteorite from representatives of the mining company late that year. After Kureym took possession of the stone, Nicholas Gessler, a now retired researcher of anthropology, archaeology and meteorites, got an e-mail about a large meteorite. The sender offered Gessler a chance to study it, saying they were looking for buyers. Piqued by a longtime interest in iron meteorites used by Indigenous people, he agreed to get it analyzed for publication in the *Meteoritical Bulletin*, a necessity to verify its provenance as a meteorite. Investigating the El Ali meteorite has since called on every one of his areas of expertise, he says, and became an obsession leading him to compile [an extensive website](#) tracking the object and what he can piece together of its sordid history. "Nothing is clear," Gessler says. "People have repeatedly asked for clarity and documentation. None has been provided."

In January 2021, after Gessler agreed to help register the meteorite, a representative of Kureym sent him a sample. A lump of iron rock, one side weathered brown, the other shining dully from the saw's incision, arrived in a FedEx package. Around the same time, the company also shipped two sliced chunks of iron rock totaling 70

grams to geologist Chris Herd, curator of the meteorite collection at the University of Alberta.

Both researchers say they wish they had known the full story of the meteorite and its contested ownership at the time. Up to that point, rumors of any violence during its excavation had been confined to Somalian news reports. Only four years later, in June 2025, did a *Meteoritics & Planetary Science* report by geoscientist Ali H. Egeh of the Somali National University first communicate to the scientific community the “secrecy and uncertainty” surrounding the meteorite and its removal from its home country.

“When I first did the work, I had no knowledge of what had happened, the tragic circumstances,” Herd says. “We were, in retrospect, getting quite biased information” about both the removal of the El Ali meteorite and its export to China. Canadian law, Herd adds, is very strict about the export of meteorites. Temporary loans of samples for study are permitted, but permanent ones are much more sensitive. “For Somalia, this would qualify as having outstanding significance and national importance,” he says. “It is a real shame it has been wholesale exported.”

At the time, Herd and Gessler were simply excited about the opportunity to investigate a meteorite—one that would turn out to be scientifically exciting in several ways. Herd first analyzed his sample with a scanning electron microscope, which shoots a beam of low-energy electrons at solid samples, to view its surface in fine detail. He also used a spectrometer to reveal its elemental makeup. A colleague at the California Institute of Technology employed an electron microscope that used a narrow electron beam with ultrafine 30-nanometer resolution to further analyze the object’s chemistry. Gessler sent part of his sample to A. J. Timothy Jull of the University of Arizona, an expert on dating meteorites, who estimated that it had landed in Somalia 2,000 to 3,000 years ago, based on radiocarbon dating. (Jull cautions that this “very rough estimate” is uncertain and says that other preliminary radionuclide

data show that it must have landed 60,000 to 30,000 years ago at the earliest.) Later in 2021 Gessler presented the El Ali meteorite to the Meteoritical Society, recognizing it as the third-largest meteorite discovered in Africa.

Meanwhile, through his correspondence with Kureym, Gessler learned the company hoped to sell the meteorite to a museum for around \$30 million. In more than a dozen e-mails, Gessler warned the sales representatives that this plan was unrealistic. But in August 2021 the company's representatives cut off contact. They complained about his collaboration on the *Meteoritical Bulletin* entry for the El Ali meteorite with Hussein, the Almaas University geologist who had first measured the meteorite for Somalia's mining ministry and who was advocating that it be placed in [the National Museum of Somalia](#), not sold.

Still, Gessler and Herd continued to analyze their samples. In 2023 Herd and his colleagues reported that they had discovered [three new iron phosphate minerals](#) in the El Ali meteorite that had never been identified as naturally occurring on Earth. "It's hugely significant scientifically," says Herd, who classified the meteorite in the [type IAB family](#). Meteorites in this group are likely to have originated in smashups in the asteroid belt between Mars and Jupiter, which created an "almost hard-to-imagine" sea of molten metal magma, Herd says. Born of collisions in space millions of years ago, these cauldrons baked their ingredients together in unusual ways, producing phosphate minerals such as elaliite, [elkinstantonite](#) and [olsenite](#) (the last two are named after revered meteoritic scientists).



Mark Smith

Given that the El Ali samples are only small slivers of a meteorite weighing more than 10 metric tons, “there could be numerous other new minerals within different areas of it,” says Diane Johnson of Cranfield University in England, an expert in ancient iron meteorites. These minerals lace the El Ali meteorite, residing inside tiny inclusions roughly the width of a human hair. “I never thought I’d be part of a study finding new minerals, much less three of them,” Herd says. “The really cool thing is when you do find them, people start to ask whether they exist in other meteorites.” Studying these deposits could reveal new secrets about the chemistry of the early solar system.

The results made a splash in the news and were [reported by the BBC](#), among other outlets. Gessler, at that point invested in the fate

of *Shiid-birood* both professionally and personally, contacted scientists in Somalia. It was then that he first learned about the accounts of bloodshed during its removal. He tried to piece together as much of the story as he could, gathering photographs and videos of the meteorite and posting them on a sprawling website devoted to it. Official representatives of Kureym had stopped communicating with him, but he began to receive text messages from people associated with the sellers, who sometimes sent updates on the meteorite's sales prospects and even videos showing its move to China. Their recent messages, which he posted at the top of his website in September, have featured bickering between two sellers over prices.

In addition to its value for solar system science, the meteorite could tell us about human history. Gessler is curious about its historic role as a source of cold iron for people in Somalia. The pitted brown lump "has been intensively and extensively hammered," Gessler says, pounded by generations of people who extracted bits of iron from the rock, probably to make tools such as arrowheads or handles. "It is a really interesting example of Indigenous use of meteorites as a resource by a community," he says.

The Iron Age in Somalia, he suggests, might have started with the El Ali meteorite.

People across the world have long exploited meteoritic metal. A knife found in the tomb of 14th-century B.C.E. Egyptian pharaoh Tutankhamen was made of meteoritic nickel, iron and cobalt. In the 11th century C.E., followers of the pre-Buddhist Bon religion in Tibet fashioned a statue, thought to be of the Buddhist god Vaiśravana, from a metal-rich meteorite that had landed on the border between modern-day Mongolia and Siberia. A Nazi-backed expedition looted the statue around 1939, and now it's privately owned by someone in Vienna. The Cape York meteorites are eight large rocks and other fragments weighing about 60 metric tons total that landed on Greenland's western coast. The Inuit people of

Greenland used these rocks to make tools and harpoons. They are the best-known example of Indigenous people exploiting meteoritic stones for iron.

These cases also further demonstrate how [commonly meteorites are looted from their original communities](#). The largest known pieces of the Cape York meteorites, for instance, are long gone from Greenland. U.S. Navy explorer Robert Peary took two of the heaviest ones, the “Dog” and the “Woman,” in 1895, and in 1897 he carried off the biggest one, the “Tent,” which weighed 31 metric tons and was more than three meters across. He sold all three meteorite pieces to the American Museum of Natural History, where they [are still displayed](#), for \$40,000—a fortune then—in a Victorian-era example of meteorite treasure hunting and [exploitation of Native resources](#). Peary also convinced six Inuit people to return to New York City with him, where [four died of tuberculosis](#). Another 20-metric-ton fragment of the meteorite was discovered in 1963 and [taken to Copenhagen](#) four years later.



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A similar story may be playing out with the El Ali meteorite. Its exact location and status now are hazy. In December 2022 the boulder was inside a shipping container on a boat docked at Mogadishu, according to [a cell-phone video](#) passed along to Gessler during his sporadic communications with the sellers. After that, it was next recorded in a shaky [May 2023 video](#) verifying the meteorite had arrived in China, still under mysterious circumstances. In that video, a speaker talks in Somali, and someone holds up a phone bearing Chinese writing in front of the meteorite. The most recent reports from the sellers and from scientists following the market for meteorites suggest the El Ali object is being held in storage in Yiwu, a midsize city in the Chinese province of Zhejiang, and is being offered for sale in

pieces at \$200 a kilogram or at \$3.2 million for the entire thing, Gessler says.

“This was a cultural looting operation, not a legal trade,” Dahir Jesow, El Ali’s representative in the Somalian parliament, told the [Horn Afrik News Agency for Human Rights](#) in June. The Kureym mining company’s rights to the meteorite were “hastily legalized ad hoc through a murky administrative process—essentially post factum to cover the theft,” he said.

Sales of meteorites by their legal owners are legal, of course, and records of such sales date [as far back as 1863](#), when one was documented in a catalog written by a German collector. [Even famous meteorites](#) are regularly bought and sold; a [slice of a Cape York meteorite](#) was offered for sale at Christie’s in 2019.

In July 2025 a Martian meteorite retrieved from the Sahara Desert in 2023—reportedly the [largest piece](#) of the Red Planet on Earth, at 25 kilograms—sold [for \\$5.3 million](#) to an unknown buyer [at auction by Sotheby's](#). Niger’s government then announced an investigation into the removal of the meteorite, “which likely bears the hallmarks of [illicit international trafficking](#),” the Nigerian Council of Ministers said in a statement issued the day after the sale. Sotheby’s, however, says it followed all relevant international procedures in the export of the meteorite, including documentation. Like the El Ali meteorite, the Martian rock was described by scholars in a [Meteoritical Bulletin entry](#), this one published in June 2025.

A [patchwork](#) of international laws governs such sales. In the U.S., meteorites are owned by whoever owns the land they’re discovered on, and [those found on public land](#) go to the Smithsonian. A 1970 UNESCO cultural-artifacts convention recognized by 148 countries, including the U.S. and Canada but not Somalia, outlines a system for tracking meteorites and returning them to their home countries if necessary. But *Shiid-birood*’s legal status is uncertain:

Sharia law currently governs the area it was taken from, but scholars aren't sure how the law treats meteorites. If UNESCO declares the El Ali object to be part of the cultural heritage of Somalia, as the government has requested, sale of the meteorite would become more difficult.

Within the past five years the market for meteorites [has exploded](#), says criminologist Donna Yates of Maastricht University in the Netherlands, following in the footsteps of [the fossil and antiquities trades](#). "There's a kind of a profile to that buyer, one that's very interested in science and space and deep time and so on," she says. "And those people visit the meteorite market." The Meteoritical Society has an ethics code requiring researchers to "[adhere to laws](#)" in their investigations, but it offers little guidance on exported finds.

China has become a destination for smuggled meteorites in recent years. In 2019 customs authorities seized 857 kilograms of "dolomite" that turned out to be [meteorites taken from Kenya](#), and in 2021 they captured 470 kilograms of [iron meteorites](#) listed as pyrite ore in customs declarations. [The Kamil impact crater](#) in Egypt was reportedly "[strip-mined](#)" for iron meteorites sometime between 2020 and 2023. "There are museums full of stolen stuff," Jull says.

Both the legal status of the El Ali meteorite and the plans of its current custodians are unclear. "The worry is that it will be ground up a piece at a time to make keychains," Gessler says. Ideally it would be returned to Somalia. In mid-July, Hussein, the geologist in Mogadishu, heard from members of the Kureym mining company who want to sell the meteorite back to Somalia's government. The company's lack of paperwork has stymied sales to international institutions. Only Somalia's government can make it legitimate, he says, making a return transaction the simplest way for the sellers to turn a profit from the meteorite. The National Museum of Somalia, once a trove of treasured antiquities, was

closed for 30 years during the country's civil war. Although many of its artifacts were damaged or stolen, the museum reopened in 2020, with space for a meteorite display under a reinforced stand. "University students and children from even elementary schools would come to see it," Hussein says. He envisions international researchers collaborating with local university students to build expertise in geochemistry in Somalia. "Now we'll have to raise the money."

Even if the meteorite does come home, its safety isn't guaranteed. Dalmar Asad, a spokesperson for the Coalition of Somali Human Rights Defenders in Mogadishu, is skeptical that his country can protect *Shiid-birood* if it returns to Somalia. Security is still very uncertain, he says, even in Mogadishu. He worries that local misunderstandings about the meteorite—some early news reports suggested it was made of gold—might prompt someone else to attempt to steal it, even from the museum. "I think it would be better for an international organization to host the meteorite until the situation here is more secure," he says.

For now the El Ali meteorite remains in limbo very far from home. Its ultimate destination is just as uncertain as it was when it was in the asteroid belt, bouncing across the sky. Wherever it ends up, though, this piece from the heavens may impart lasting lessons on Earth. The meteorite's tale should teach scholars to ask harder questions about the provenance of newly reported discoveries, says planetary scientist Hasnaa Chennaoui Aoudjehane of the Hassan II University of Casablanca in Morocco. "This community is my community; many scientists are friends of mine," she says. "But in some cases, we are just closing our eyes. We don't want to take on the reality of the problem because if we do, there will be much less material to study."

Editor's Note (9/26/25): This article was edited after posting to correct the description of Nicholas Gessler's comment about the Eli Ali object being offered for sale in pieces at \$200 a kilogram. The

text was previously amended on September 24 to correct the location of Maastricht University.

Dan Vergano is senior editor, Washington, D.C., at *Scientific American*. He has previously written for Grid News, BuzzFeed News, *National Geographic* and *USA Today*. He is chair of the New Horizons committee for the Council for the Advancement of Science Writing and a journalism award judge for both the American Association for the Advancement of Science and the U.S. National Academies of Sciences, Engineering, and Medicine.

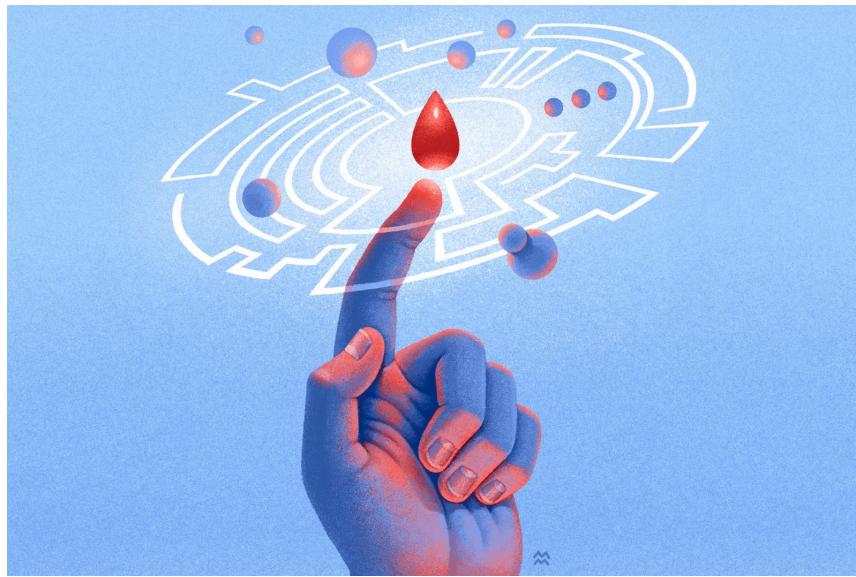
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Type 1 Diabetes Science Is Having a Moment

Living with type 1 diabetes today is leaps and bounds easier than it was decades ago. Things are only getting better

By [Jeanna Bryner](#)



Miriam Martincic

This article is part of “[Innovations In: Type 1 Diabetes](#),” an editorially independent special report that was produced with financial support from [Vertex](#).

A little more than a century ago a diagnosis of type 1 diabetes was a death sentence. Today, thanks to extraordinary scientific progress, many people with type 1 diabetes—especially in developed countries—can enjoy long, healthy lives. I’m profoundly grateful that my older son, now 16, was born into this era of possibility. His diagnosis in 2020 came at a time when innovation and advocacy had transformed what it meant to live with this chronic autoimmune disease. I’m excited to share this [Innovations In special report](#), in which we explore the remarkable advances reshaping the landscape of type 1 diabetes research and care.

Science journalist Carrie Arnold tells the stories of [visionary entrepreneurs working toward an artificial pancreas](#)—a closed-loop system that seamlessly integrates insulin pumps and glucose monitors to mimic the function of healthy beta cells. For many, this breakthrough feels like a cure.

Yet the pursuit of a true cure continues. Health journalist Tara Haelle [delves into promising options](#), including beta cell transplants that would eliminate the need for immunosuppressive drugs. She also highlights the monoclonal antibody teplizumab, approved in 2022, which can delay the onset of symptomatic type 1 diabetes by five years or more in some people—a milestone in preventive medicine.

As the science races forward, [the number of type 1 diabetes cases is surging](#). Statistical visualizations by data journalist Miriam Quick and senior graphics editor Jen Christiansen illustrate this troubling trend and expose the stark disparities in care. In wealthy nations such as the United Arab Emirates, a child diagnosed with type 1 diabetes may live nearly a full life. In contrast, a child in Niger could lose up to 50 years compared with the national average.

Associate editor Lauren J. Young introduces us to five [exceptional individuals working to close these gaps](#)—among them a radiology specialist who joined forces with fellow mothers to distribute insulin and establish education outreach across Venezuela, as well as clinicians trying to ease distress over diabetes and improve mental health. Their stories are deeply moving.

Health reporter Liz Szabo explores the strides in [preventing or halting diabetic retinopathy](#), the leading cause of blindness in working-age adults. Science journalist Rachel Nuwer describes [advances in genetic screening to identify kids at high risk](#) of developing type 1 diabetes. That information could become life-altering as new treatments emerge.

Although managing type 1 diabetes will continue to be a 24/7 endeavor for now, I have realistic hope that a cure will emerge in my son's lifetime.

Jeanna Bryner is executive editor of *Scientific American*. Previously she was editor in chief of Live Science and, prior to that, an editor at Scholastic's *Science World* magazine. Bryner has an English degree from Salisbury University, a master's degree in biogeochemistry and environmental sciences from the University of Maryland and a graduate science journalism degree from New York University. She has worked as a biologist in Florida, where she monitored wetlands and did field surveys for endangered species, including the gorgeous Florida Scrub Jay. She also received an ocean sciences journalism fellowship from the Woods Hole Oceanographic Institution. She is a firm believer that science is for everyone and that just about everything can be viewed through the lens of science.

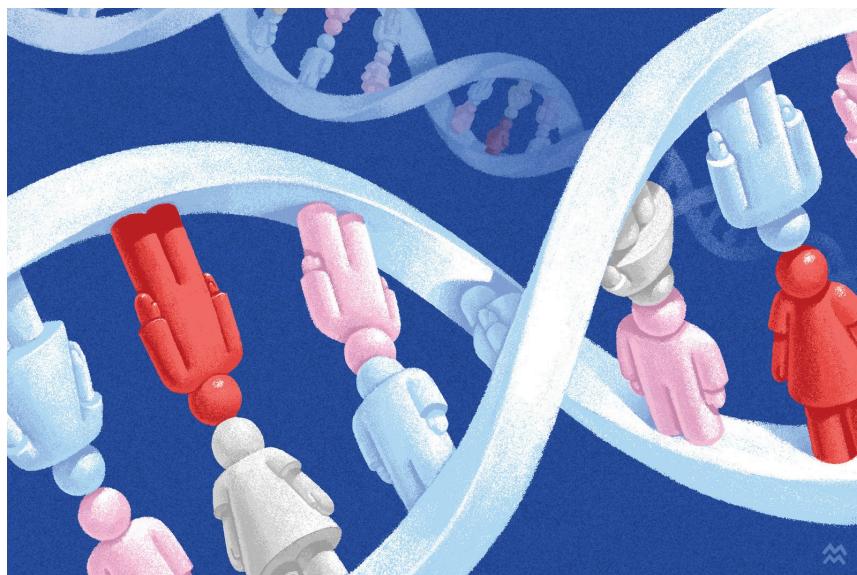
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Diagnosing Type 1 Diabetes before Symptoms Strike

Genetic screening can mean that people at risk of type 1 diabetes get earlier treatment and better outcomes

By [Rachel Nuwer](#) edited by [Jeanna Bryner](#)



Miriam Martincic

This article is part of “[Innovations In: Type 1 Diabetes](#),” an editorially independent special report that was produced with financial support from [Vertex](#).

In 2024 Stephen Rich and his colleagues published a study in which they assessed the genetic risk of developing type 1 diabetes for more than 3,800 children from across Virginia. Almost immediately Rich, a genetic epidemiologist at the University of Virginia, was inundated by e-mails and calls from parents who had read the article and wanted their kids tested, too. Unfortunately the study was over, so Rich couldn’t help them. But the experience exemplified the growing interest in genetic risk tests for the disease, he says.

There is currently no cure for type 1 diabetes, a chronic condition in which the body's immune system attacks and kills insulin-producing beta cells in the pancreas. Knowing someone's genetic predisposition to type 1 diabetes, however, can help doctors identify whom to flag for follow-up tests. It can also lead to earlier adoption of therapeutics to manage the disease or delay its onset. "There's tremendous power in terms of understanding the genetics of type 1 diabetes," says Todd Brusko, director of the Diabetes Institute at the University of Florida. As more therapies become available, he adds, the eventual hope is to use genetic profiling to determine who will respond best to one drug versus another.

Recent advances in genetic screening tools have not only revealed an intricate interaction between a person's genes and their immune system but also made it possible to imagine a future in which every newborn is screened for type 1 diabetes risk. Some health-care authorities are already beginning to consider universal screening. "It's very exciting times," says Maria Jose Redondo, a physician and professor of pediatric diabetes and endocrinology at the Baylor College of Medicine. "A lot of progress has been made, and now we're at the point of applying it."

In the U.S., around one in 300 people develops type 1 diabetes. Although the disease is best known for manifesting in children, adults account for almost half of new diagnoses. Scientists still don't know what triggers it. Environmental factors seem to play a crucial role in promoting the disease's development and progression, but the exact causative agents are unknown. "We know less about the environmental factors than we know about the genetic factors," Redondo says.

In a large study called TEDDY (for "the environmental determinants of diabetes in the young"), launched in 2004 in Europe and the U.S., researchers followed 8,676 individuals with high genetic risk to try to identify triggers for type 1 diabetes. They found just one consistent environmental factor linked to higher

likelihood of acquiring the disease: early infection with enteroviruses, a type of virus that can infect beta cells. Not all children who get these common infections go on to develop type 1 diabetes, though, so additional factors are probably at play. In addition, the incidence of the disease has been increasing steadily over the past 60 years, suggesting that some change in environmental exposures or the removal of protective factors—or both—may be involved.

Genetics accounts for about half of a person's risk of developing the disease, meaning what is written into someone's DNA is "not destiny," Rich says. "If you have a high [genetic] risk, it doesn't mean you'll get it, and if you have a not-high risk, that doesn't mean you're protected."

For people with a close relative with type 1 diabetes, the risk goes up to about 18 in 300. Those with an identical twin with the disease have the highest risk—about one in two. They are 150 times likelier to develop the illness than someone with no family history and eight times likelier than someone with a parent or sibling who has been diagnosed. Even so, around 90 percent of people who are diagnosed with type 1 diabetes have no relatives with the disease. Until recently, population-level genetic screening, which would include individuals regardless of their known risk factors for the condition, was not a practical option. But new breakthroughs have begun to change that.

Scientists have identified at least 90 regions in the human genome that hold genes connected to type 1 diabetes. Researchers are most interested in a gene cluster called the human leukocyte antigen system (HLA), which encodes proteins that help the immune system distinguish self from nonself. This gene group accounts for around half of a person's genetic risk of developing the disease. Because it helps to protect us from infections, HLA is also highly variable, says Mark Anderson, director of the Diabetes Center at the University of California, San Francisco. "There's selective

pressure for us to have different HLA genes because that way, a virus or bacterium that comes along won't wipe everyone out."

Most people who acquire type 1 diabetes have at least one of two specific-risk-conferring gene variants, or alleles, in this region. "This region is so critically important to whether we're susceptible to autoimmune diseases that just by measuring variation there, we can capture risk," says Richard Oram, a professor of diabetes and nephrology at the University of Exeter in England. Some HLA variants increase risk up to 20-fold, he adds, whereas others decrease risk by the same amount. In effect, it's as if 10 to 15 percent of people with European ancestry carried a genetic vaccine to type 1 diabetes, Oram says, referring to the HLA gene alleles that decrease risk.

In 2015 Oram and his colleagues developed the first version of what is now one of the most widely used tests for type 1 diabetes genetic risk, administered primarily in research settings (the U.S. has yet to approve any test for type 1 diabetes risk for real-world use in doctor offices). Rather than just adding up the contribution of each variant, Oram and his colleagues' test incorporates the complex interactivity of various alleles with one another, including ones with protective effects. They also incorporated dozens of other non-HLA sites—mostly from genes also related to the immune system—that contribute small amounts of individual risk but can add up to larger cumulative risk.

The original version of the test examined just 10 alleles and "worked pretty well," Oram says. The latest version, developed in 2019, uses 67 alleles and produces "highly sophisticated" results, Redondo says, adding that it now represents "the golden standard to date."

When Oram originally developed his test, he did not have risk prediction in mind; rather he was trying to decipher the type of diabetes in a group of his patients. The individuals he was working

with, who were 20 to 40 years old, had overlapping features of type 1 and type 2 diabetes. People who fall into this “gray area” of symptoms are commonly misdiagnosed, he says. While brainstorming solutions over coffee with a colleague, Oram realized a genetic test could offer clues for people with a less clear presentation of the disease.

After successfully developing the test, Oram learned that other research groups were interested in tests to determine genetic risk for type 1 diabetes. Fortunately his test “also turned out to be really good for that,” he says.

With Oram’s test, doctors can identify the highest-risk individuals, who can then get tested for the antibodies that attack the body’s beta cells. “If you do HLA screening followed by antibody testing at specific ages, you’ll pick up far and away the vast majority of cases,” says William Hagopian, a research professor of pediatrics at the Indiana University School of Medicine. Investigators leading vaccine and pharmaceutical trials for type 1 diabetes are also using genetic tests to maximize efficiency and funding by identifying participants who are most likely at risk for the disease.

Genetic risk scores can also help doctors identify people who should be prescribed teplizumab, the first therapy able to delay the onset of an autoimmune condition. Approved by the U.S. Food and Drug Administration in 2022, this monoclonal antibody is given before the body becomes dependent on insulin, and it can delay more severe illness by two to three years. “The whole field has changed because now we have something we can do to delay progression to clinical diabetes,” says Kevan Herold, an immunologist and endocrinologist at Yale University. “Any time without diabetes is a gift, particularly for children and their families.” Other drugs are in various stages of clinical testing.

People aware of their risk might also be on the lookout for symptoms such as excessive urination and lethargy; when those

pop up, people can seek treatment before they develop diabetic ketoacidosis (DKA), a potentially life-threatening condition caused by a lack of insulin. Among those who don't know they are at risk, about 40 percent wind up in this critical state, but that number drops as low as 4 percent for those who are aware. "If people can identify some of the symptoms of progression toward disease, they could go to a GP instead of an ER and prevent a real crisis," Brusko says.

There is some evidence to support these benefits, based on outcomes from one of the largest testing efforts to date, launched in 2020 by investigators at Sanford Health, a nonprofit health-care system based in Sioux Falls, S.D. As of July 2025, the study had enrolled more than 13,000 children for genetic risk testing and antibody screening for type 1 diabetes and celiac disease. Children with persistent positive antibodies are offered ongoing monitoring. Of the 75-plus children in monitoring, five have progressed to hyperglycemia, warranting clinical care, and none of these children developed DKA. Kurt Griffin, principal investigator of the study and a pediatric endocrinologist at the Benaroya Research Institute in Seattle, says the findings have already demonstrated that it is feasible to integrate type 1 diabetes screening into routine pediatric care.

Type 1 diabetes has been most prevalent among people of European ancestry. It does occur in those of African, Hispanic and Asian ancestry, but the vast majority of data used to inform genetic screening results is from people of white, European descent, Rich says. This lack of representation is problematic for people of different ancestries because genetic risk factors differ across populations.

In an unpublished study, Rich and his colleagues tested how well the most common HLA variants used in genetic tests predicted risk in people with European, Hispanic, African American or Finnish ancestry. They found that genetic ancestry for important HLA

regions—and the many other regions of the genome associated with type 1 diabetes risk—does not transfer well from one population to another. “One of the biggest needs in the field is to understand what confers genetic risk in a much more diverse genetic ancestry,” Brusko says.

Scientists are working to fill this gap. For instance, Breakthrough T1D, a nonprofit organization funding research on type 1 diabetes, provides grants of up to \$900,000 for research aimed at improving the prediction power of genetic risk scores across diverse populations. For the next version of the genetic risk score test, the plan is to incorporate specific HLA types present in Africans, East Asians, and several other groups, says Hagopian, who collaborates with Oram.

Genetic risk tests for type 1 diabetes are inching closer to use in clinical care. Last year Randox, a company based in Northern Ireland, released one developed with Oram and his colleagues. Commercial tests are not available yet in the U.S., but they are becoming more affordable for researchers who use them in laboratory-based settings. This affordability will translate to clinical settings once tests make their way to doctor offices. “The price has dropped and is predicted to drop even more,” Redondo says. Now the biggest remaining obstacles are political and logistical rather than scientific or financial, experts say. “All the tools are there; we just haven’t quite got countries over the line to figure out how they’re going to do it,” says Colin Dayan, a professor of clinical diabetes and metabolism at Cardiff University in Wales.

Europe has been at the forefront of these efforts, Brusko says. In 2023 Italy became the first nation to pass a law mandating type 1 diabetes genetic screening across its population, but it has yet to implement this screening in practice, Dayan says. Other countries, including the U.K., are debating whether they should do the same. This past June the U.K. also announced plans to sequence the

genomes of all babies within the next decade. The data obtained could be used for risk screening as well, says Emily K. Sims, a pediatric endocrinologist at the Indiana University School of Medicine. In the U.S., genetic screening for type 1 diabetes is still done primarily in research environments. “We really need federal and state authorities to decide that this testing is worth it and that they want to adopt it into general practice,” Hagopian says. The easiest way to implement such a program would be to screen at birth.

What to do with the information that testing would generate, though, is a more complicated question. Health-care officials would have to set up a system for contacting the families of babies at high risk to appropriately communicate the results. There would also need to be a system to remind families to get their child checked for autoantibodies at certain intervals. States handle newborn screenings differently, so each would have to come up with its own solutions. This issue is “a major complication that has to be figured out,” says Rich, who continues to field e-mails and calls from parents interested in the testing.

As the science is refined, more treatment options will be made available, and the uncertainty surrounding who will and will not go on to develop type 1 diabetes is likely to be narrowed. Redondo and her colleagues are pursuing a large project using genetic risk scores and other variables to try to more accurately predict disease development. They are also working on models to determine who will respond best to new disease-modifying therapies. As Redondo says, “personalizing prevention of type 1 diabetes is the goal.”

Rachel Nuwer is a science journalist and author. Her latest book is *I Feel Love: MDMA and the Quest for Connection in a Fractured World* (Bloomsbury, 2023). Follow her on Bluesky @rachelnuwer.bsky.social

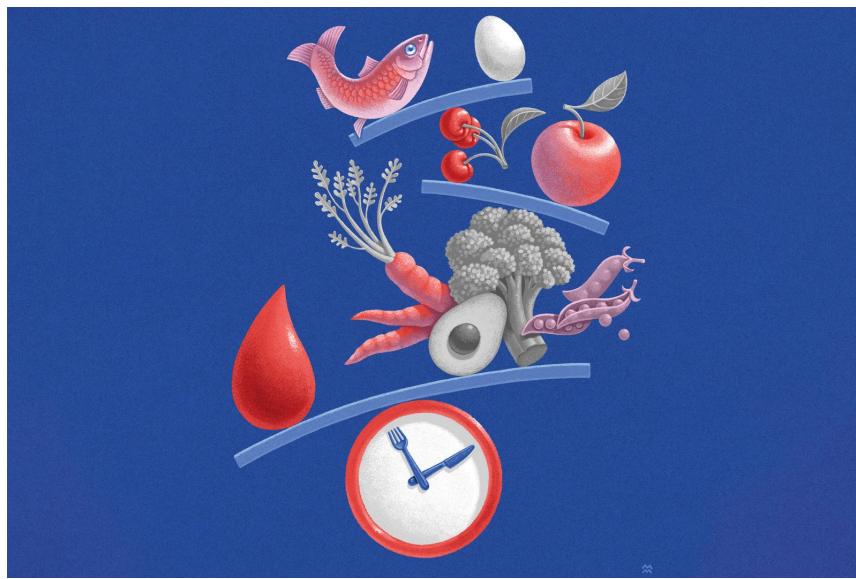
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Edward Damiano's Bold Bet on a Bionic Cure

How a father's love, entrepreneurship and tech advances could lead to a working artificial pancreas

By [Carrie Arnold](#) edited by [Jeanna Bryner](#)



Miriam Martincic

This article is part of “[Innovations In: Type 1 Diabetes](#),” an editorially independent special report that was produced with financial support from [Vertex](#).

Edward Damiano carved his life into precise 90-minute intervals. In 2000 his then 11-month-old son, David, developed [type 1 diabetes](#) when an autoimmune response in his tiny body attacked the beta cells in his pancreas, which manufacture and secrete the hormone insulin. No beta cells meant no insulin. If Damiano or his wife, Toby Milgrome, a pediatrician, didn't give David injections of the hormone, the baby's cells could no longer use glucose, a vital energy source. Within hours David could be in a coma or dead. With their son's pancreas no longer functioning, Damiano and Milgrome had to take over the organ's work by measuring every gram of carbohydrate David ate and dosing the right amount

of insulin. To ensure his son didn't receive too much or too little of the lifesaving medicine, Damiano checked David's blood glucose every hour and a half, rain or shine, day or night.

When David first became ill, [Damiano, a bioengineer at Boston University](#), kicked it old school to monitor his son, accumulating a set of spiral-bound notebooks in which he or Milgrome logged every drop of insulin and morsel of food. Each day the couple flipped over a new page and started again, building a complex calculus of sugar grams per sip of Juicy Juice measured against units of insulin and [blood glucose](#) that would allow David to thrive. Even for a physician-scientist team, the work was grueling and relentless. Damiano also knew that he was one of the lucky ones.

“The hardest part of diabetes management is making all those decisions. And if you give yourself a little too much insulin, you could end up in the ICU,” Damiano says.

While Damiano waited for David's blood glucose to tick up or edge down, his engineering brain crunched the problem. There had to be a better way, he thought. The [more widespread use of commercially available continuous glucose monitors \(CGMs\) by 2004](#) meant someone with diabetes could get a minute-by-minute measurement of their blood glucose without turning their fingers into pincushions. Users of insulin pumps, which deliver doses of insulin for meals and steady doses in the background, could tether their CGMs to their pumps and start to automate some parts of their insulin delivery. In September 2016 [Medtronic first paired CGMs and insulin pumps](#) in what scientists call a hybrid closed-loop system that could automatically adjust insulin delivery based on a person's blood glucose levels, except at mealtimes, which still needed to be manually programmed. The devices were a major breakthrough, but many people with diabetes still found managing blood glucose to be a continuous struggle.

“Management of type 1 diabetes is like driving a car 24/7 on a curvy mountain road with no breaks even when you’re asleep. So if you could take some of that burden off, it could make a huge difference,” says biotechnology entrepreneur Bryan Mazlish, co-founder of biopharmaceutical company Surf Bio and of Bigfoot Biomedical, which produces tools to help people manage diabetes.

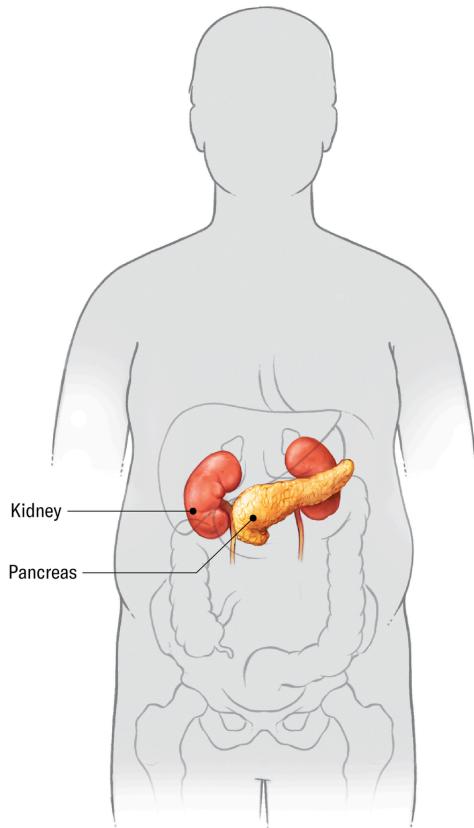
In 2015 Damiano co-founded Beta Bionics to see whether he could ease the burden for his son and others with type 1 diabetes by creating an artificial pancreas—a completely automated, closed-loop system that functioned like a real, healthy pancreas. In May 2023 the U.S. Food and Drug Administration [approved the company’s iLet device](#), an adaptive closed-loop system that requires only a patient’s body weight to start insulin delivery. Instead of counting carbs, users can input “small,” “medium” or “large” for meal sizes. In January 2025 Beta Bionics went public.

The company is one of several outfits currently inching toward the development of a fully automated insulin-delivery system, considered an artificial pancreas, that can track and alter blood glucose levels through insulin, much like the body does on its own in healthy individuals. Clearing the final hurdles for regulatory approval won’t be easy. Successfully programming a device to make the same minute-by-minute adjustments as the human body will require a close marriage of biology and technology. Until now, most advances in type 1 diabetes tech have allowed the rich to get richer, so to speak, says [Steven Russell](#), an endocrinologist at Massachusetts General Hospital and chief medical officer at Beta Bionics. People who already had healthy control over blood glucose could make improvements, but those who were struggling often continued to struggle.

“We can allow people who hadn’t traditionally had good glycemic control to achieve it,” Russell says. “That means almost everybody can get good glucose control on it regardless of where they’re coming from.” Although the iLet costs less than other closed-loop

systems, retailing at \$3,500, those without health coverage may still struggle to afford the device and CGM supplies it requires.

As a young endocrinologist in the 1980s, David Klonoff was typically greeted with a full waiting room at the diabetes clinic at the University of California, San Francisco. Some had seeing-eye dogs because years of too-high blood glucose had caused retinopathy, which, in some extreme cases, caused blindness. Others carried scars and marks on their forearms from dialysis treatments for [kidney disease](#), which can result from cumulative damage to fragile blood vessels in the kidney from living with elevated blood glucose levels. Some had a fungal infection called mucormycosis, also known as [black fungus](#), which can infect the nasal passages, sinuses, lungs and skin of people who have chronic high blood glucose or are immunocompromised. Severe cases can cause disfigurement and death. Still others bore amputations after diabetes complications led to peripheral neuropathy, a condition that blunts sensation in the extremities. Without nerve sensation, the patients weren't aware of festering ulcers and infections, which therefore went untreated until amputation was the only option. Given that Klonoff's patients had to guess at the appropriate insulin doses, perhaps it wasn't so surprising. Klonoff likens the technology of the time to the Wright brothers' first aircraft prototypes as compared with today's jet engines.



Shehryar (Shay) Saharan

Most of these patients devoted hours every day to managing their condition. The problem wasn't a lack of effort but rather a lack of appropriate tools. With no way to test their blood glucose levels, people with diabetes had to make an educated guess about how much biosynthetic insulin to inject. And because too much insulin could be fatal, they had to err on the side of too little insulin and adjust on the fly. Not surprisingly, blood glucose control was often suboptimal. The advent of home-based finger-prick testing in the 1980s and faster-acting biosynthetic insulins made a tremendous difference in reducing complications, but Russell and other doctors still saw great disparities among their patients. Those lacking good medical literacy and adequate time and resources to devote to managing their disease continued to struggle.

Although the development of automated insulin pumps helped to remove some of the burden, many patients found simply staying alive to be a permanent, unpaid, full-time job.

“It’s impossible to be awake and on top of this optimally—understanding the physiology of insulin action and insulin duration and the impact of specific meals and foods. It’s not feasible,” says Carol Levy, director of the Mount Sinai Diabetes Center in New York City. And she would know—Levy has lived with type 1 diabetes for more than 50 years.

Mazlish knew what to expect when his son turned out to have type 1 diabetes. After all, his wife also had the disease, and she began to educate her husband as soon as their son was diagnosed. Mazlish watched as his wife programmed her insulin pump to deliver both dribbles of basal insulin throughout the day and a large bolus with meals. If her blood glucose dropped too low, she had to eat something carbohydrate-laden to raise it. To Mazlish, a finance quant turned life sciences entrepreneur, the work seemed amenable to automation with a computer program.

To help his son, Mazlish wrote a bespoke algorithm that would automatically adjust the insulin delivered by the pump based on the child’s blood glucose levels. “It was really life-changing. We could live much more freely, and it gave peace of mind to all of us,” Mazlish says.

Other tech-savvy patients were innovating in similar ways, creating computer codes and programs to automate insulin delivery and ease their own burden. These efforts demonstrated that such an approach could work, but not everyone in the field was sure that an algorithm could accurately adjust insulin delivery based on glucose levels, says [Boris Kovatchev](#), an engineer and director of the University of Virginia Center for Diabetes Technology.

That is until widespread use of CGMs by people with type 1 diabetes in the mid-2000s and the subsequent integration with insulin pumps created the first realistic hope that endeavors by Kovatchev, Mazlish, and other biomedical technorati could result in a usable device.

“Management of type 1 diabetes is like driving a car 24/7 on a curvy mountain road with no breaks even when you’re asleep.” —Bryan Mazlish *Surf Bio and Bigfoot Biomedical*

In December 2005, scientists, engineers and physicians gathered on the campus of the National Institutes of Health in Bethesda, Md., for the first-ever workshop about the prospects of building an artificial pancreas. Some of the attendees weren’t convinced the effort was feasible, Kovatchev says. Physicians and engineers alike fretted about the high stakes if a system malfunctioned. Others preferred to focus their attention on creating a cure for type 1 diabetes, not just building more bells and whistles for an existing treatment.

Kovatchev, however, thought that people with diabetes could benefit from an artificial pancreas and that the potential for error would be significantly less than what resulted from patient guesswork.

The biggest challenge the researchers realized was building a set of algorithms that were sensitive enough to allow minute adjustments to insulin doses and flexible enough to work for millions of patients. It was a challenge that could be overcome only with gobs of data, something neither Kovatchev nor anyone else had.

Like Damiano, Kovatchev approached the diabetes problem from a perspective other than a physician’s—in his case, through mathematical modeling. He needed to figure out how to replicate biology’s intricacies in an automated device that could be used by millions. That was no easy task. For one, everyone’s body responds to insulin slightly differently. “The variation is huge. Everything has to be individualized, and that was a major problem throughout the years,” Kovatchev says.

What’s more, commercially available insulins don’t work as quickly as the hormone naturally produced by the pancreas.

Kovatchev and other engineers would need their algorithms to account for insulin already at work in the bloodstream as well as administered hormone that had not yet started to lower blood glucose.

After the first pancreas workshop in December 2005, Kovatchev helped to build the informational foundation for such an undertaking. First he got detailed data on glucose metabolism in healthy people to understand how the body processes glucose from food, how recently consumed sugars interact with the sugars already in the body, and how glucose levels change after meals.

Scientists continued the effort, eventually accumulating data from upward of 7,000 healthy individuals. It was enough to [garner FDA approval of the UVA/PADOVA Type 1 Diabetes Simulator in 2008](#) as an alternative to animal testing in certain preclinical trials and artificial pancreases. An updated version was approved in 2013 with even more parameters to capture the complexity of glucose biology.

Other advances were happening at the same time. In 2008 a team of researchers at the University of California, Santa Barbara, led by chemical engineer Francis Doyle, reported that it had built the first prototype device that would allow CGMs to communicate with insulin pumps, opening the door for systems that could automatically adjust insulin dosing outside of meals. These insulin pumps and CGMs have had a huge positive impact on quality of life, says Jonathan Rosen, director of research at the nonprofit Breakthrough T1D. “Rates of long-term complications have gone down over the years thanks to improved blood sugar control,” he says.

But these achievements were still not considered artificial pancreases: even the most sophisticated devices still required users to manually administer extra insulin to account for meals. And it was these interactions that continued to trip up many users, Russell

says. Humans aren't very good at estimating meal components down to the gram, and measuring every mouthful of food requires intense devotion. In addition, the pumps didn't function correctly right out of the box; specialized endocrinologists had to help program the devices and make adjustments every few months.

At Boston University, Damiano recognized similar issues as his son grew up and began taking more responsibility for managing his disease. Eliminating the need for manual adjustments—whether for meals or for any other changes during the day—would give people with diabetes a tremendous sense of freedom. Even better would be to forgo the initial programming and adjustments by a physician. By the mid-2010s artificial intelligence and machine-learning algorithms began providing the solutions that Kovatchev and Damiano were looking for.

"I want a system that is democratizing," Damiano says. Everybody, he emphasizes, should have the ability to access this technology.

For iLet wearers, these solutions mean they can begin using their device immediately. The only information they need to input is body weight. From there, data transfer from previous pumps and the iLet's own software feed into the AI-driven software to accurately control blood glucose in patients, with only brief meal inputs required by users. The company has reported that within their first year of using iLet, patients have been able to better manage their blood glucose.

Adding neural networks and other AI technology such as digital twins (digital models of real objects, places or people that can be used to simulate responses to a variety of conditions) allowed Kovatchev to re-create CGM readouts from the Diabetes Control and Complications Trial, launched in 1993. These simulations, [published in March 2025](#), were able to accurately predict different diabetes complications and the amount of time someone's glucose was in a healthy range. When Kovatchev allowed patients to

experiment with their digital twin, they could see the likely outcome before it happened. “That was very educational. People loved it,” he says. But Kovatchev also cautions that the safety and security of an artificial pancreas continue to be issues. “These algorithms are black boxes. Nobody really knows what’s going on inside and how they react to different situations. It’s critical to have constraints so it doesn’t do something stupid or dangerous,” he asserts.

To Damiano, the iLet is a game changer, but it’s not the end of the road. He envisions a fully closed-loop system that doesn’t require any user input at all, as well as a dual-hormone device that can administer the blood-glucose-raising hormone glucagon alongside insulin. But the field now is worlds away from those midnight pencil scribbles in a spiral notebook that kept his son alive until better technology came along.

Carrie Arnold is an independent public health journalist in Virginia.

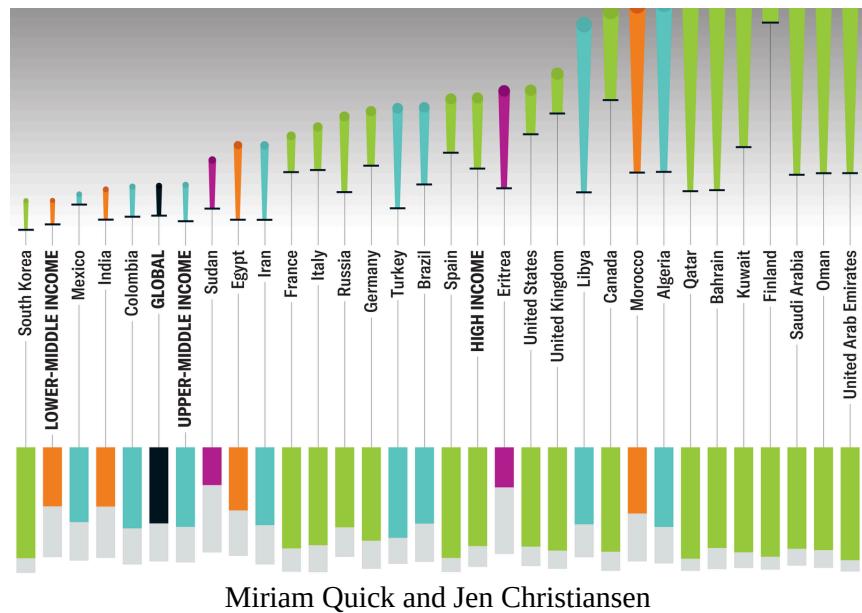
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Here's a Nuanced Look at Type 1 Diabetes Cases Worldwide

This autoimmune disease impacts millions of people worldwide, with some underserved communities bearing the brunt

By [Miriam Quick](#), [Jen Christiansen](#) & [Jeanna Bryner](#) edited by [Jeanna Bryner](#) & [Jen Christiansen](#)



This article is part of “[Innovations In: Type 1 Diabetes](#),” an editorially independent special report that was produced with financial support from [Vertex](#).

More than [9.5 million people](#) globally live with type 1 diabetes, and this figure is increasing rapidly everywhere, across all age groups. The burden of this autoimmune disease—the risks of which include premature death—is unequally carried: the condition is more deadly in lower-income countries.

“Data speak for themselves,” says Stephanie Pearson, senior director of global responsibility at the nonprofit Breakthrough T1D. “Type 1 [diabetes] is being diagnosed more than it ever has been before, but you need more in-depth studies to understand why these

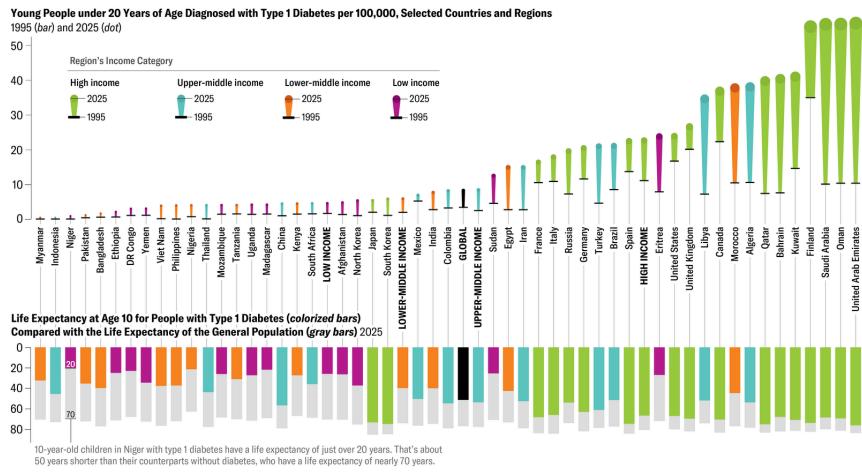
numbers continue to rise.” Scientists know that with more education and awareness, diagnoses tend to go up. They are also looking into additional possible causes for this increase, including potential triggers that make a person’s body kill off its own insulin-producing cells. Bacterial and viral infections, diet, lifestyle choices and even factors related to pregnancy may all contribute.

Ultimately, Pearson says, patients hope for a cure for diabetes, but they also need better access to advanced therapies, treatments for complications and technology to manage their illness.

The Growing Burden of Type 1 Diabetes in Youths

More people are being diagnosed with type 1 diabetes (T1D) than ever before—an estimated half a million this year—and at increasingly younger ages. Over the past three decades improvements in diagnostic tools and awareness have underpinned a steady rise in the number of people receiving a diagnosis of T1D in almost every country in the world. For people younger than 20, the sharpest increases have been recorded in high-income nations of the Middle East.

In lower-income countries, where health-care systems are less equipped to detect and manage the disease, T1D remains far more lethal. A 10-year-old child with the condition in the United Arab Emirates can expect to live to about age 76, roughly eight years shy of the national average. In Niger, however, a 10-year-old with T1D faces a drastically different reality—this child could expect to live just another decade on average, losing out on as many as 50 years.

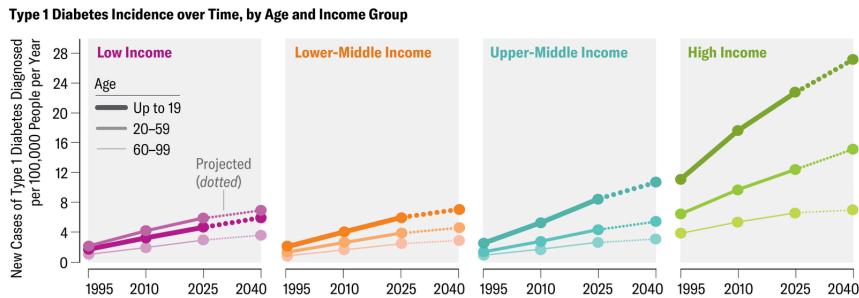


Miriam Quick and Jen Christiansen; Source: T1D Index (www.t1dindex.org) (data)

Type 1 On The Rise

The number of people worldwide living with T1D is climbing rapidly, driven by rising incidence coupled with longer lifespans, lower mortality and overall population growth. Rates are increasing relative to population numbers in both wealthy and poor nations and across all age groups. The growing burden is not confined to the young: it reflects both a general rise in cases and earlier diagnosis.

But the picture is uneven, and inequalities are stark. In high-income countries, data suggest that nearly everyone younger than 25 with T1D is diagnosed. In low-income nations, however, only about two thirds of such cases are identified, according to estimates from the International Diabetes Federation and the T1D Index. Reported incidence in children and adolescents is therefore much lower than expected, reflecting missed or delayed diagnoses. These figures are modeled because more than half of countries lack current data, underscoring the urgent need for better epidemiological research worldwide.



Miriam Quick and Jen Christiansen; Source: T1D Index (www.t1dindex.org) (data)

Why Are Cases Rising?

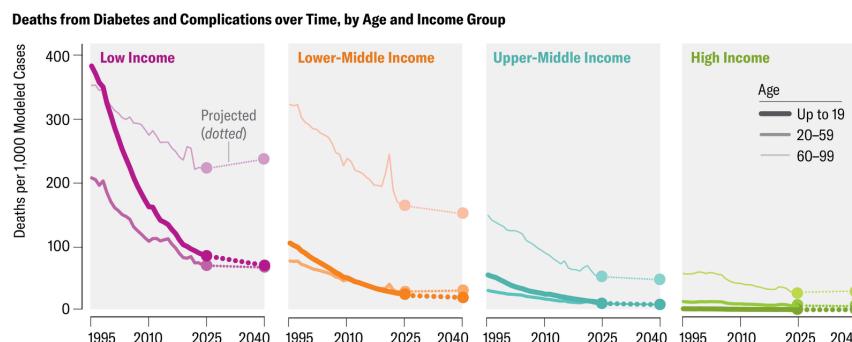
Scientists still do not fully understand why the number of T1D diagnoses has climbed so quickly. Earlier and more accurate detection is certainly part of the story, but the sharp rise in new cases relative to population numbers hints that underlying rates of the disease may also be increasing. A range of environmental influences have been linked to greater risk, with many tied to gut microbiome health. Early-life infections, along with factors related to pregnancy and birth, can trigger autoimmune disorders such as T1D, in which the immune system destroys insulin-producing beta cells in the pancreas. Lifestyle factors may also contribute. Obesity and poor diet are well-known drivers of type 2 diabetes, and some researchers suspect they could influence the development of type 1 as well.

No Longer a Death Sentence

Untreated diabetes can be deadly even in the young. This year an estimated 174,000 people worldwide will die from T1D; about 30,000 of them will be younger than 25 and never formally diagnosed. Many of these deaths stem from diabetic ketoacidosis, a complication in which ketones build up in the blood, raising its acidity. The condition is often mistaken for something else, leading to delays in critical care. Children and adolescents with T1D also face heightened risks of long-term complications, including

cardiovascular disease and kidney failure. Around 30 percent of patients eventually develop end-stage kidney disease.

Yet T1D is highly treatable. Insulin therapy, combined with tools to monitor blood glucose, can extend life expectancy and improve quality of life dramatically. Where treatment is accessible, a diagnosis no longer amounts to a death sentence, and death rates have plummeted as treatment has improved. Global modeling by the T1D Index suggests that if everyone received a timely diagnosis and proper care—and nobody died prematurely from diabetes—more than four million additional people would be alive today.



Miriam Quick and Jen Christiansen; Source: T1D Index (www.t1dindex.org) (data)

Miriam Quick is a data journalist and researcher specializing in information visualization.

Jen Christiansen is acting chief of design and senior graphics editor at *Scientific American*, where she art directs and produces illustrated explanatory diagrams and data visualizations. She is also author of the book *Building Science Graphics: An Illustrated Guide to Communicating Science through Diagrams and Visualizations* (CRC Press). In 1996 she began her publishing career in New York City at *Scientific American*. Subsequently she moved to Washington, D.C., to join the staff of *National Geographic* (first as an assistant art director–researcher hybrid and then as a designer), spent four years as a freelance science communicator and returned to *Scientific American* in 2007. Christiansen presents and writes on topics ranging from reconciling her love for art and science to her quest to learn more about the pulsar chart on the cover of Joy Division’s album *Unknown Pleasures*. She holds a graduate certificate in science communication from the University of California, Santa Cruz, and a B.A. in geology and studio art from Smith College. Follow Christiansen on Bluesky @jenchristiansen.com

Jeanna Bryner is executive editor of *Scientific American*. Previously she was editor in chief of Live Science and, prior to that, an editor at Scholastic’s *Science World* magazine. Bryner has an English degree from Salisbury University, a master’s degree in biogeochemistry and environmental sciences from the University of Maryland and a graduate science journalism degree from New York University. She has worked as a biologist in Florida, where she monitored wetlands and did field surveys for endangered species, including the gorgeous Florida Scrub Jay. She also received an ocean sciences journalism fellowship from the Woods Hole Oceanographic Institution. She is a firm believer that science is for everyone and that just about everything can be viewed through the lens of science.

<https://www.scientificamerican.com/article/the-growing-global-burden-of-type-1-diabetes>

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Champions of Caring

Advocates are lightening mental health burdens, improving pregnancy care and helping patients in developing countries

By [Lauren J. Young](#) edited by [Josh Fischman](#)



Kimberly Driscoll, Danielle Hessler Jones, Sarit Polksy, Florence Brown and Ileana Gill (*left to right*).

Joel Kimmel

This article is part of “[Innovations In: Type 1 Diabetes](#),” an editorially independent special report that was produced with financial support from [Vertex](#).

Type 1 diabetes wears many faces—an elusive autoimmune disease, a metabolic mess, a chronic condition that requires lifelong treatment. That means researchers, clinicians and advocates have to wear many faces as well to meet the different needs of different patients. Some of these changemakers work to raise awareness of the mental health burdens of the illness, others elevate standards of care for pregnant people, and still others try to bring essential resources to countries with limited health-care availability. Here are their stories.

A Physical and Mental Disease

The relentless physical damage inflicted on people with type 1 diabetes is well known. But more psychologists and mental health practitioners are working to address the ways the disease—with its constant demands to manage insulin and blood glucose levels—takes a heavy toll on the mind.

“There’s a very normal emotional reaction to living with diabetes, known as diabetes distress,” explains Danielle Hessler Jones, who is a psychologist at the Behavioral Diabetes Research Group at the University of California, San Francisco.

Levels of exercise, diet and sleep, as well as stress, can make blood sugar fluctuate dramatically. People with diabetes may be “doing everything that they need to in terms of administering insulin and monitoring glucose, but sometimes blood glucose just doesn’t cooperate,” says Kimberly Driscoll of the University of Virginia, a clinical psychologist specializing in pediatric diabetes and co-editor of a type 1 diabetes mental health workbook. That loss of control can lead to frustration and burnout. Social and economic barriers create another layer of strain. Some people may experience stress from poor access to transportation, healthy foods, medication or specialized care, Hessler Jones says.

“People with type 1 diabetes hear a lot of asks from health-care providers, for good reason, but it really adds up” to a lot of mental strain, Hessler Jones says. And “some people feel that they have to hide this from their neighbors, their loved ones, their workplace.” Attempts to camouflage the distress may exacerbate it.

Because of that, diabetes distress has been linked to other serious mental health issues. [A 2024 study](#) found that one in five people with type 1 diabetes was diagnosed with either depression or anxiety. A [2022 paper](#) showed that disordered eating, including food restriction, binge eating and self-induced vomiting, was more

common in adolescents with the disease than among their peers without it. “Because insulin can be associated with weight gain, we sadly see people not taking this lifesaving medication at the doses that they should,” Hessler Jones says. This illness has a specific name: [diabulimia](#).

There are solutions, fortunately. Mental health interventions can help with these problems and improve disease management among patients. Hessler Jones recently led a randomized, controlled trial called [EMBARK](#), in which 276 adults with type 1 diabetes were divided into three different virtual support groups. One was a diabetes management group led by a certified diabetes educator. A second was a group focused purely on mental health, led by a clinical psychologist. A third group combined these two approaches. After one year, participants in all three groups saw reductions not only in their distress but in levels of hemoglobin A1C (a key indicator of blood glucose; higher levels mean worse control). The psychological care group actually had the highest and most consistent improvements, with the participants’ diabetes distress scores cut nearly in half and their A1C levels significantly reduced.

People in that group learned various therapeutic techniques they could use themselves, including emotional mindfulness, acceptance strategies and planning for daily self-care. “We found this was incredibly powerful for individuals,” Hessler Jones says. “So many people have told us, ‘No one has ever asked me this before. No one ever talks about this.’”

Hessler Jones and her team are currently bringing the trial’s findings into real-world care. They’ve trained 15 clinics on the program’s key components and will have full results in late 2026. Driscoll’s workbook on mental health care for children and adolescents with type 1 diabetes also walks through various strategies, including communication and problem-solving skills. She crafted the resource to support kids and their families and

therapists. “Oftentimes we will get referrals from medical providers that [a person needs] to work on getting the A1C down,” Driscoll says, “but if a person is depressed or has no interest in engaging with the [treatment] regimen, you have to work on that first before you can start on the self-management piece.”

“The disease requires management 24 hours a day,” Driscoll says—but this degree of attention doesn’t have to hamper quality of life. Empowering people with mental wellness tools can restore a sense of control over their health and lives.

Healthier Pregnancies

Most people with type 1 diabetes should keep blood glucose levels in the recommended range of 70 to 180 milligrams per deciliter. Pregnant people with the disease, however, have less flexibility. For them the band shrinks to 63 to 140 mg/dL because higher glucose levels can hurt the developing fetus. “When people with type 1 diabetes become pregnant, they feel an enormous burden,” says Sarit Polsky, director of the Pregnancy and Women’s Health Clinic at the Barbara Davis Center for Diabetes at the University of Colorado Anschutz Medical Campus.

That’s why Polsky and other clinicians are studying new treatment strategies to support pregnant people with type 1 diabetes in maintaining their goals. “What’s been shown in many, many studies over decades of research is that higher glucose levels can lead to fetal harm,” Polsky says. Clinical data have linked hyperglycemia to increased rates of birth defects, cesarean deliveries, miscarriages and neonatal intensive-care admissions. High levels pose risks to the early developing embryo, too: congenital heart disease and kidney defects can occur when hemoglobin A1C levels are high during the first 10 weeks of pregnancy. For pregnant people, hypertension is a big risk.

At the 2025 American Diabetes Association conference in July, Polsky presented several new updates to the organization's [standards for care during pregnancy](#). One of the new recommendations is maintaining good glucose levels using automated insulin delivery (AID) systems—an "artificial pancreas" or insulin pump that automatically adjusts doses in response to changes in blood glucose [see "[The Long Journey to an Artificial Pancreas](#)"]. In addition to the overall target, "glucose levels overnight and between meals [should be] under 95 mg/dL," Polsky says. An AID system can be set to maintain these ranges.

These recommendations came out of a recent randomized, controlled trial called [AiDAAPT](#), which showed that people with type 1 diabetes who used an AID system set with pregnancy-specific fasting glucose targets stayed within the recommended range about 70 percent of the time. That added up to 2.5 more hours each day than in a comparison group using another insulin-delivery method.

Preliminary evidence also hints that the environment of the womb may contribute to the genesis of type 1 diabetes in the fetus. Polsky is collaborating with other researchers on the PROMISE study, which is investigating womb exposures that may trigger type 1 autoimmunity. They plan to look at various factors, such as viral exposures, maternal microbiome data and parental genetics. Babies will also be screened for type 1 diabetes for up to five or six years. "If you identify children who have autoantibodies [attacking insulin-producing cells], you can follow them more closely," says endocrinologist Florence Brown, one of the trial researchers and co-director of the Joslin and Beth Israel Deaconess Medical Center's Diabetes in Pregnancy Program. If at-risk infants are identified very early, Brown says, medications can reduce their risk of certain diabetes complications.

Reaching Communities

In 2017 radiology specialist and physician Ileana Gill, who was working in Venezuela, feared her daughter might be dying. The child, who was then four years old, briefly slipped into a coma after being admitted to a local intensive care unit. She developed diabetic ketoacidosis—a dangerous buildup of acidic compounds called ketones caused by a lack of insulin—and was diagnosed with type 1 diabetes.

“I got to see firsthand what it was like to have someone you really care about live with type 1 diabetes, especially in a country where [there’s unpredictable] access to health supplies and to a pediatric endocrinologist,” says Gill, who is now a radiology professor at the University of Miami. Last year her oldest daughter was also diagnosed with the illness.

Adjusting to the treatment and lifestyle changes was difficult. “If this is so hard for me and my family, who are very well educated and have access to information, diabetes educators and nutritionists —it made me imagine what it is like for a regular person and for a person who’s in the lower economic bracket” of a developing nation, Gill says.

Shortly after her daughter was diagnosed, Gill co-founded [Guerreros Azules](#), a Caracas-based nonprofit organization that provides education about type 1 diabetes, insulin supplies, blood glucose test strips, and other types of diabetes care. Gill says to date they have served more than 2,500 kids. She visits Venezuela annually and helps to build global partnerships.

Scientific American spoke with Gill about how her work with [Guerreros Azules](#) is closing care gaps in Venezuela and how the organization’s approach could help other communities in need.

An edited transcript of the interview follows.

What challenges face children with type 1 diabetes in Venezuela?

The lack of insulin and the price of it are barriers in Venezuela. While I was living there, even being a physician and knowing how the market works, there were days where I wasn't sure my daughter was going to have insulin for the month or if she was going to have access to all of the care she needed. It's a common experience. Insulin is expensive—a package of insulin pens, which may last a month, might be \$25. The monthly salary for a family could be \$125.

What's the inspiration for Guerreros Azules?

“Guerreros Azules” means “blue warriors.” The name is very special because blue is the color associated with type 1 diabetes awareness, and “warrior” represents the type 1 community worldwide. Because it’s a 24/7 condition, you need to be always on guard. The fight isn’t always bad, but it’s always there. The association was founded by a group of moms, all of us living with kids with type 1 diabetes. I met them when my daughter was hospitalized. One of the moms came to visit me because she heard my daughter had been diagnosed with type 1 diabetes. That mom, Marta, gave me her number and told me to call her if I had any questions. We’re not all physicians or diabetes experts, but we know a lot from caring for our children. So we thought: let’s start helping other parents.

What approaches or resources have you found are most successful?

We focus on education, supporting parents and creating awareness. The way we started was if someone was recently diagnosed, we would go to visit the family at their house or a hospital, the same thing Marta did for me.

We provide free insulin, but we realized that you could give people an insulin pen, glucose strips and a meter, but they didn't know how to use it. So we concentrate on education and then give out the insulin and supplies. We also give out what we call a Blue Warrior's kit for those who are newly diagnosed. People know they have to learn all of this, but at least they don't have to scramble to get the medication.

For outreach, we were doing everything—we were on the radio, we were on social media, we were on the streets, we were in the schools, in the parties, in the clubs, in the pharmacies. Then we were doing a lot of education for the parents. Initially we did very small trainings at churches, at schools, with other NGOs, at people's houses. Now we do education sessions at the hospital and in bigger settings.

At the hospital, where we had one pediatric endocrinologist, we trained several nurses in diabetes care. Then we worked with the Central University of Venezuela to create a fellowship for pediatric endocrinology. We trained three residents the first three years. Now we train not only in Caracas but also throughout the country. Currently we work with 28 pediatric endocrinologists.

How has your work expanded to help others internationally?

There's a lack of data on kids with type 1 diabetes. For the past eight years we've been able to create a registry of the children we've worked with. We're one of the few countries in South America to have a registry. A foundation in Guatemala has reached out for advice on developing a similar list to help determine how many kids with type 1 they have. We're hoping we can expand on it and use it for treatment development and understanding insulin use.

Lauren J. Young is associate editor for health and medicine at *Scientific American*. She has edited and written stories that tackle a wide range of subjects, including the COVID pandemic, emerging diseases, evolutionary biology and health inequities. Young has nearly a decade of newsroom and science journalism experience. Before joining *Scientific American* in 2023, she was an associate

editor at *Popular Science* and a digital producer at public radio's *Science Friday*. She has appeared as a guest on radio shows, podcasts and stage events. Young has also spoken on panels for the Asian American Journalists Association, American Library Association, NOVA Science Studio and the New York Botanical Garden. Her work has appeared in *Scholastic MATH*, *School Library Journal*, *IEEE Spectrum*, *Atlas Obscura* and *Smithsonian Magazine*. Young studied biology at California Polytechnic State University, San Luis Obispo, before pursuing a master's at New York University's Science, Health & Environmental Reporting Program.

<https://www.scientificamerican.com/article/meet-the-advocates-who-are-changing-type-1-diabetes-care-for-the-better>

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Saving Your Sight from Blinding Diabetic Retinopathy

Diabetic eye disease robs sight from millions. But there are often ways to save vision

By [Liz Szabo](#) edited by [Josh Fischman](#)



Miriam Martincic

This article is part of “[Innovations In: Type 1 Diabetes](#)”, an editorially independent special report that was produced with financial support from [Vertex](#).

Sterling Glass had many health problems as a child—swollen feet, night sweats, nausea and vomiting, unquenchable thirst, and fatigue that often left him too exhausted to go to school. The problems didn’t seem connected. Neither Glass nor his parents asked a doctor whether there was an underlying cause until he was 19, when he couldn’t get out of bed and wasn’t able to eat for five days because he felt so sick. That’s when doctors told the family that Glass had type 1 diabetes.

By that point, 30 years ago, Glass's blood glucose levels had spiked to 600 milligrams per deciliter—six times higher than normal. “The doctor was blown away,” says Glass, who now is 49 and lives in Asheboro, N.C. “He was like, ‘I don’t know how you’re still alive. There’s no telling how long your sugar has been running that high.’”

Today Glass is blind. Years of [uncontrolled blood glucose can cause serious damage](#) to organs throughout the body. The eyes are frequent sites of injury. In the retina, the light-detecting tissue in the eye, excess glucose can harm tiny blood vessels and make them leak. The damage cuts off sight. Over time more than half of people with diabetes [develop diabetic retinopathy](#), which can lead to [vision loss and blindness](#). The condition [affects nearly 10 million people](#) in the U.S. and [100 million around the world](#). It is the [leading cause of blindness in working-age](#) people.

There are treatments to stave off the consequences. Surgery with scalpels or finely tuned lasers can stabilize the eye and preserve vision; in particular, new refinements in laser surgery can stop many abnormal blood vessels from growing. There are also drugs that inhibit vessel growth that can be injected directly into the eye; in the newest improvement, medical researchers have developed a small reservoir for these drugs that can be implanted directly into the eye, eliminating the need for repeated injections. And new research indicates that GLP-1 receptor agonists such as Ozempic and Mounjaro—which have gained staggering popularity because of their weight-loss effects—can slow or prevent retinopathy’s blinding deterioration because of the way they improve control over blood glucose.

Unfortunately, like Glass, many people are not screened for type 1 diabetes for many years. This delay allows retinopathy to get started, and the illness usually does not cause any obvious symptoms until it reaches an advanced stage. “Diabetic retinopathy is unfortunately becoming an epidemic, mainly because diabetes

itself has become an epidemic,” says Raj K. Maturi, a retina specialist and ophthalmologist at the Indiana University School of Medicine.

Like many people with diabetic retinopathy, Glass says he had no vision problems before his diagnosis. An optometrist had told him he had 20/20 vision. But through his 30s, he had trouble controlling his glucose levels. He worked in a warehouse, sometimes driving a forklift, and he says the company didn’t allow him to bring blood glucose testing kits with needles to work, even during 12-hour shifts. On busy days Glass sometimes skipped lunch, which exaggerated the swings in his blood glucose levels.

Then Glass began to experience crushing headaches that made him feel as if he were about to pass out. He could no longer operate heavy machinery safely, and he eventually went on disability. The layoff prompted him to finally, after years of ignoring his wife’s pleas to get a complete eye exam, make an appointment with an ophthalmologist.

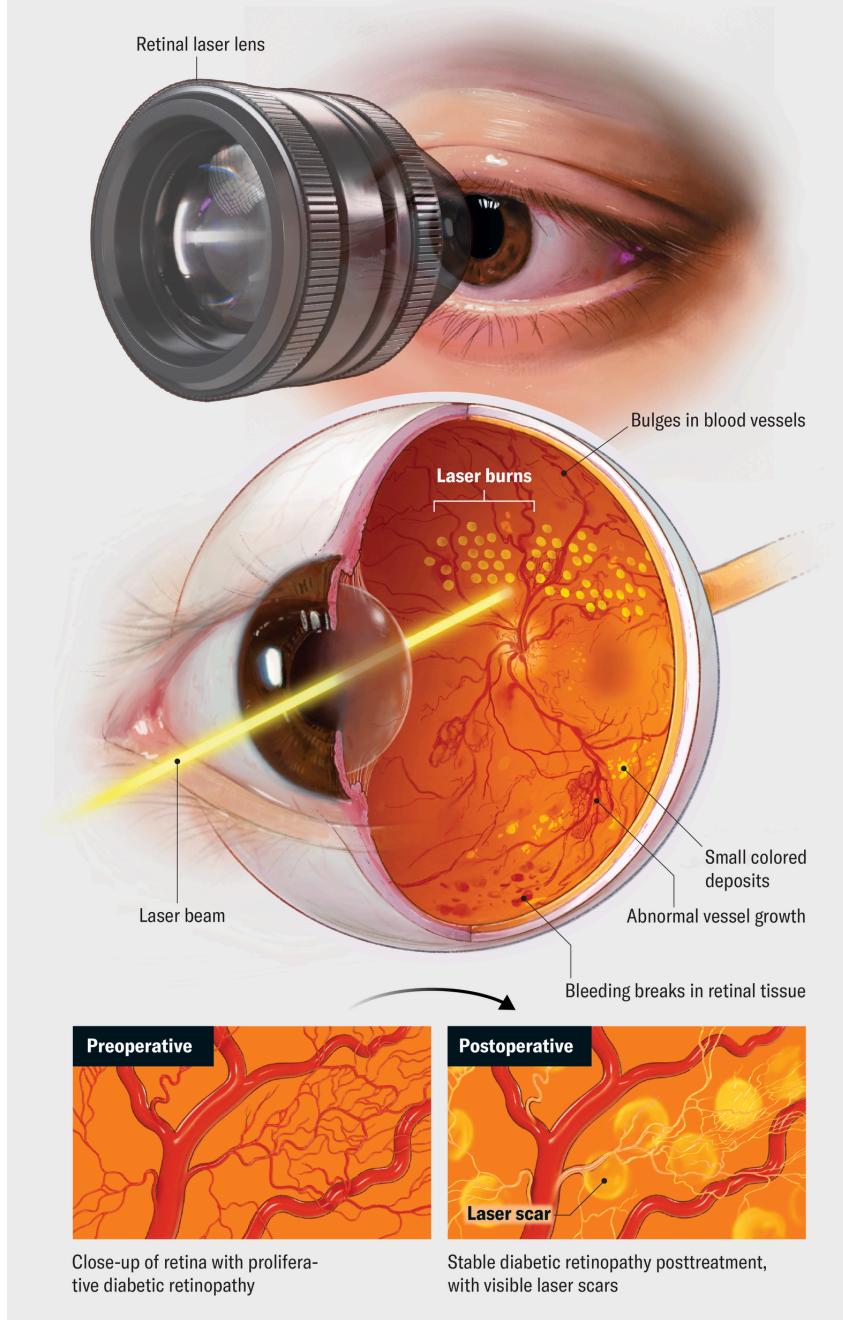
Glass was so confident in his eyesight that he drove himself to the ophthalmologist’s office.

When the doctor told Glass he had diabetic retinopathy, he thought to himself, “I have perfect vision. You are crazy. No way, no how, you’re not touching my eyes. I’m not having surgery, because there’s nothing wrong with me.”

Like Glass, who is Black, many people from historically marginalized communities [have advanced disease](#) by the time they’re diagnosed, partly because of a lack of access to health care. “It’s not easy in our health-care system to find a provider and get diagnosed early,” says Adrian Au, an ophthalmologist at the University of California, Los Angeles, Stein Eye Institute.

Shining Light into Darkness

In diabetic retinopathy, the loss of sight is caused by the growth of abnormal blood vessels in the retina, the light-detecting sheet of cells at the back of the eye. These vessels leak and damage the retina. A technique called laser photocoagulation can stop the deterioration. A pinpoint laser turns the damaged spots into scars. The scars do not attract new leaky blood vessels to grow toward them, specialists theorize. Therefore, vision can be preserved.



Shehryar (Shay) Saharan; Source: Raj Maturi, MD, FASRS (*expert reviewer*)

Although retinopathy is the most common cause of vision loss for people with diabetes, people with diabetes are at risk of other forms of eye disease as well. People with diabetes **are two to five times** more likely to develop cataracts, which cloud the lens of the eye.

The macula, part of the retina needed for sharp vision, may become distorted, causing blurry sight.

Although retinopathy rarely causes pain, it's possible that the severe headaches Glass suffered were caused by the proliferation of abnormal blood vessels on his iris, the colored part of the eye, says Akrit Sodhi , an ophthalmologist at the Wilmer Eye Institute at the Johns Hopkins University School of Medicine. In this condition, called neovascular glaucoma, blood vessels can block the eye's drainage system, causing fluid to build up and creating pressure in the eye that can cause significant pain and sensitivity to light.

The failure to adequately screen and treat people for diabetic eye disease is “a tragedy that needs to be addressed,” says Avnish Deobhakta, an ophthalmologist at the New York Eye and Ear Infirmary at Mount Sinai Hospital.

Eventually Glass did agree to have surgery. The operation appeared to be successful at first, but his retinopathy had weakened many parts of his eyes. The retina in his left eye ultimately failed. “I never saw out of my left eye again,” he says.

Retina specialists tried multiple procedures to save his remaining vision, including injections of a medication directly into his right eye. This and similar medications slow or halt the growth of new blood vessels, and Maturi says they have revolutionized retinopathy treatment and prognosis. The medications **block the action of a protein** called vascular endothelial growth factor, or VEGF, that promotes blood vessel growth, including the development of abnormal blood vessels in the retina that threaten a person’s sight. Some newer drugs **block additional proteins** that contribute to diabetic retinopathy.

Anti-VEGF medications can slow the rate of vision loss and even reverse it, helping people see more clearly, Maturi says. Today these drugs are often the first treatment people receive. They

“changed our field overnight,” Deobhakta says. “Anti-VEGF drugs became the standard of care.” The medications don’t work for everyone, however. They are less successful in patients with very advanced disease, and insurance coverage can restrict which drugs people are able to receive.

Glass tried the injections for his advanced disease, but they did not halt his vision loss. So his ophthalmologist tried another option: laser surgery to try to make blood vessels shrink and stop growing. This type of surgery has been used to treat diabetic retinopathy since the 1970s and 1980s and has continued to become more refined.

Sterling Glass talks to as many people as possible about managing their diabetes and protecting their eyesight: “Take it seriously. You get only one set of eyes.”

In the most common type of surgery, doctors aim to stabilize abnormal parts of the retina that can cause bleeding, swelling or detachment. They use lasers to scar the periphery of the retina—the outer region of the retina that’s responsible for side and night vision—to prevent it from sending growth signals that would lead to more abnormal blood vessels, Deobhakta says.

Scarring the outer retina can cause people [to lose their peripheral vision](#), color vision and night vision. But by tamping down growth signals, the procedure can protect the macula, which is in the central retina. “It almost feels like you’re destroying the retina to save it,” Deobhakta says. “That’s kind of what it is.”

For Glass, his vision continued to worsen. He was still able to go out because his adult godson, who lived with him and his wife, served as a second pair of eyes. “He never left my side,” Glass says. Then a family rift prompted his godson to move out of the house. Glass thought he would be stuck at home, without any

independence. He sank into a deep depression and even tried to kill himself.

His sister saved him. “My sister called me and said, ‘You’re not going to do this. You’re not gonna die. And you’re not gonna give up. You’re gonna live,’” Glass recalls. And she had a plan for how he was going to live. She is a professional singer, and she helped him contact the music director of the Blind Boys of Alabama, a Grammy-winning gospel group made up of Black men who have lost their sight. They invited him to audition.

“I met the five gentlemen who would change my life, although I didn’t know that at the time,” Glass says. It was the first time that he had met other blind people. He says he was amazed to meet men who had persevered and succeeded in spite of the loss of their vision. The men shared their stories and suggested organizations that Glass could call for help. It was the closest thing to a support group that he had experienced since losing his sight.

“After returning back home, I thought to myself, ‘Whether I go out with these guys to sing or whether they don’t even call me back after today, I’m happy. For these gentlemen to be able to live and be blind, I felt like, ‘Okay, now I have hope. I don’t have to die. I can still live.’”

A few weeks later the Blind Boys invited Glass to join the band. He sang with them for the first time in 2022 and has been performing ever since. He is, he says, glad to be alive.

Newer forms of treatment may provide more help for people with diabetic retinopathy. Early versions of the injectable medications had to be put into the eyes once every month, and the frequent medical visits created a burden for people trying to hold down a job or take care of their family. Many patients are “working-class people who often may not even have insurance,” Deobhakta says.

“So you often get more and more complications because patients can’t get their injections.”

But the latest generation of anti-VEGF injections can last for two or even three months, requiring fewer doctor visits, Deobhakta says. And now there’s a refillable reservoir for the drugs that can be implanted in the eye. The device only [needs to be refilled](#) every nine months.

People with type 1 diabetes may also get aid from the new glucagonlike peptide 1 (GLP-1) receptor agonist drugs, such as Ozempic. The barrage of media coverage highlights their weight-loss functions, but formulations of these drugs were originally developed to help people with diabetes [control their blood sugar](#). Glass takes a GLP-1 drug called Mounjaro, along with short- and long-acting insulin and other drugs that help to control diabetes.

Recent research has produced conflicting results about the ways that GLP-1 drugs might affect the progression of early diabetic eye disease. Some studies [link them to a small—and temporary—increase](#) in early diabetic retinopathy, leading experts to suggest that people who take the drugs should receive regular eye exams. On the positive side, research also finds that people who take GLP-1 drugs have a lower risk of progressing to worse stages of diabetic retinopathy and blindness. Because any increase in retinopathy appears to be short-lived, stabilizing with vision still intact after a period of time, the benefits of GLP-1 drugs outweigh the risks, Sodhi says. “In general, it’s better to get good control of your blood sugar” with these medications, he notes.

Scientists are also [exploring gene therapy](#)—implanting a gene that helps to inhibit blood vessel growth—in the hope of treating retinopathy with a one-time eye treatment. And research hints that a drug class called fenofibrates, which has been used to treat abnormal levels of cholesterol and triglycerides in the blood, [can](#)

[reduce the risk](#) that early diabetic retinopathy will become more severe.

Glass says he now works hard to manage his diabetes. He and another member of the Blind Boys, who also has diabetes, encourage each other to check their blood glucose. While traveling with the group, which performs at least 100 shows a year, Glass says he talks to as many people as possible about the importance of managing diabetes and protecting their eyesight. “If I could tell anybody anything, I would say, ‘Take it seriously.’ You get only one set of eyes.”

According to the National Eye Institute, early detection, treatment and follow-up exams for diabetic retinopathy [can reduce the risk of blindness](#) by 95 percent. The American Diabetes Association recommends people with type 1 diabetes have their first eye exam within five years of diagnosis. Au notes that identifying people with diabetic retinopathy and referring them for additional services before they lose their vision can help them be more prepared if their eyesight deteriorates. At his eye institute, doctors can help people [access visual rehabilitation](#), which can provide technology such as magnifiers and screen readers; counseling and emotional support programs; and transportation and household services.

Overall there is a shortage of doctors who specialize in treating the retina, and some health systems are trying to compensate for this by using telemedicine and artificial intelligence to screen people with diabetes for retinopathy, a development that could be especially helpful for those who live in rural areas. Primary care providers can take photographs of the retina, which can be reviewed by an AI program. Doctors can then refer people to retinal specialists for future evaluation and treatment.

As for Glass, he says the Blind Boys have not just given him a career. They have given him purpose. “I’ve got a new mission,” he asserts. “I have an opportunity not only to live but to share my

story with people. I am very passionate about saving anybody that I can.”

IF YOU NEED HELP

If you or someone you know is struggling or having thoughts of suicide, help is available. Call or text the 988 Suicide & Crisis Lifeline at 988 or use the [online Lifeline Chat](#).

Liz Szabo is a veteran health and science journalist who has worked at *USA TODAY* and other newsrooms.

<https://www.scientificamerican.com/article/saving-the-vision-of-people-with-diabetic-retinopathy>

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A Cure for Type 1 Diabetes Seems Close, Thanks to New Drugs and Tech

A new drug slows insulin-dependent diabetic deterioration and has sped up development of a complete remedy

By [Tara Haelle](#) edited by [Josh Fischman](#)



Miriam Martincic

This article is part of “[Innovations In: Type 1 Diabetes](#),” an editorially independent special report that was produced with financial support from [Vertex](#).

On one wall of endocrinologist Kevan Herold’s office hangs artwork by a girl who joined one of his type 1 diabetes trials when she was 11 years old. The girl was diagnosed with stage 2 of the disease, a status that meant her own immune system was making at least two types of antibodies that attacked the insulin-producing beta cells in her pancreas. The immune assault interfered with her ability to produce insulin, the hormone that controls blood glucose levels, says Herold, who works at Yale University. But she did not yet need insulin to treat her diabetes.

Typically people with stage 2 disease soon develop stage 3, when their blood sugar levels become so dysregulated that they need insulin. But that was before the arrival of [teplizumab](#), a monoclonal antibody approved in late 2022 that delays the advance of the illness and may even halt it at stage 2 in some people. The girl in the trial went on the drug in about 2011. Her disease did move to stage 3—but not for almost a decade.

“She was free of diabetes for eight to nine years. It enabled her to go to middle school, high school, graduate and so on, to grow up without diabetes,” Herold says. “Even if you develop it when you’re 21 or 22, that’s different than when you were 11, and you’ve had the opportunity to do what your peers do—not to have to think about it 24/7.” In a way, he says, “we’re kind of in the time business. We’re buying time without disease.”

Although teplizumab doesn’t work for everyone and how much it delays progression varies, it has inspired new directions in research, and a few therapies show signs of success. The progress has people in the type 1 diabetes community using a word that was rarely heard a few decades ago: “cure.”

Cure can be a charged word in medicine. “Even in people affected by diabetes, they have a difference of opinion about what a cure is,” says [Alvin C. Powers](#), an endocrinologist at the Vanderbilt School of Medicine. As a complete remedy, he says, some people would be happy with an insulin pump with a glucose sensor that automatically detects their blood sugar levels and metes out only exactly as much insulin as they need, a goal that’s within reach now [see “[The Long Journey to an Artificial Pancreas](#)”]. “Other people say, ‘If I have to take anything, it’s not a cure,’” Powers notes. Some think of transplantation of insulin-producing cells that can permanently control glucose as a cure; still others consider real healing to be a way to stop the autoimmune attack on those cells, preserving enough so that people don’t need additional insulin.

“Ideally it’s a one-and-done, like a vaccine, so one can never get type 1 diabetes,” says [Sanjoy Dutta](#), chief scientific officer of Breakthrough T1D, a nonprofit research and advocacy organization that’s funding efforts to find a cure for type 1 diabetes. But given the complexity and heterogeneity of the disease, that’s not likely to be what a complete remedy will look like. “Cures come in many forms, and one form is not going to work for everyone,” Dutta says.

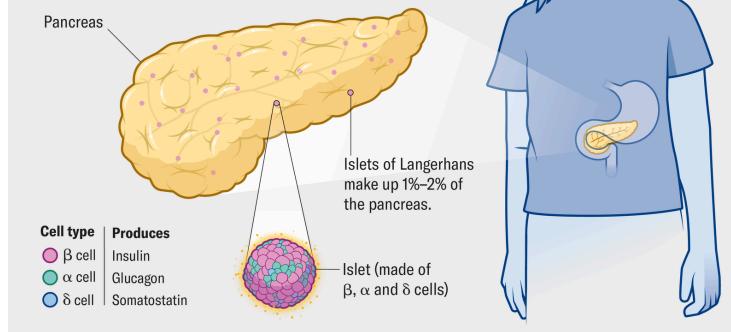
That’s why scientists are pursuing multiple approaches. The variety and the progress suggest a remedy is possible and perhaps not even that far off. “We know that for cures, it’s a matter of when, not if,” Dutta says.

One of the biggest challenges facing doctors and patients is that type 1 diabetes involves two problems. “One, the immune system has gone rogue and is destroying [the body’s beta cells], and the second is the loss of insulin production because of the death of beta cells,” Dutta says. “So to cure or modify the disease, to slow it down or reverse it, you need something to put a check on the immune system and then protect the remaining beta cells or regrow them. I envision a world where we have to use these methods in combination to cure the disease.”

The first part of that combination, the immune therapy approach, is where teplizumab comes into play. The drug binds to a particular protein on immune system cells called T cells and reduces their ability to attack the body’s beta cells. At the same time, it promotes changes in the T cells that may pump the immune system’s brakes.

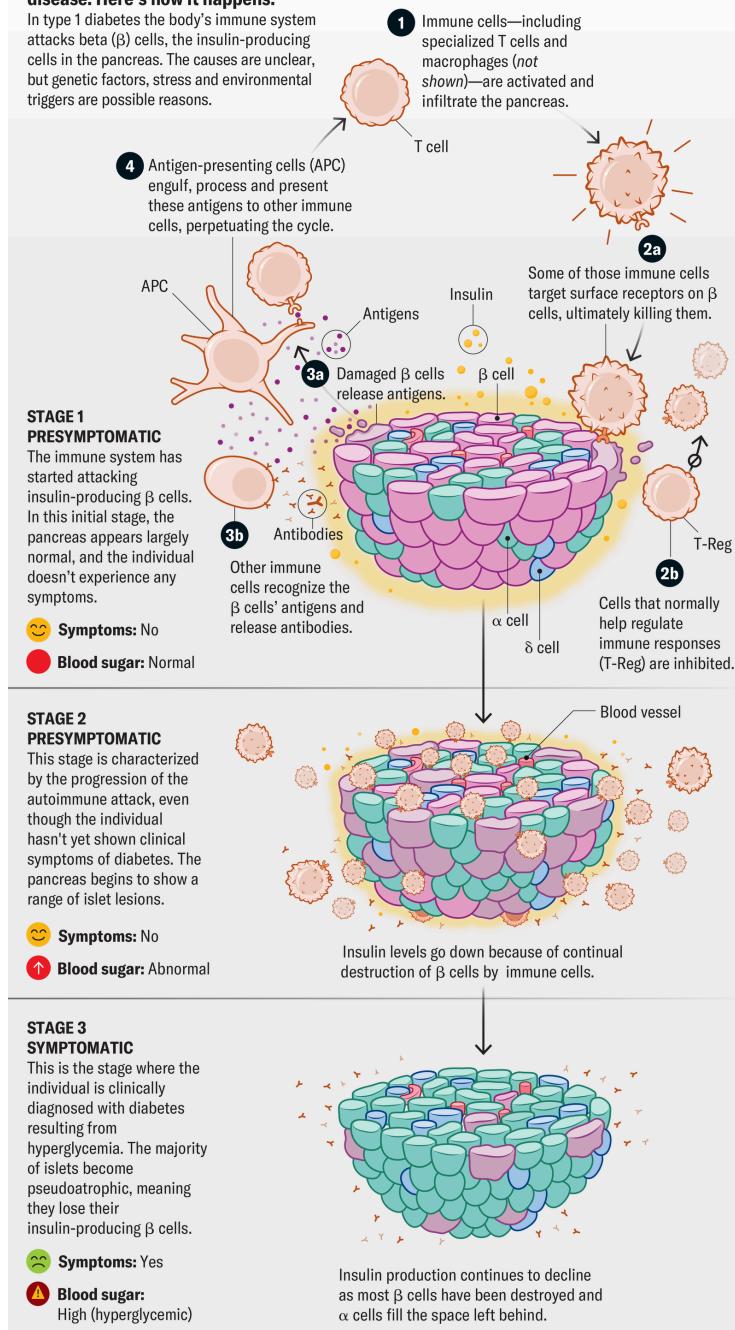
The Deteriorating Cycle of Type 1 Diabetes

The exact cause of type 1 diabetes isn't known, but the effects of the illness are all too evident. Cells of the body's immune system, which ordinarily attack pathogens, turn against beta cells in the pancreas. These cells produce insulin, a hormone all cells need for health. Damaged cells increase the immune assault, and eventually the body cannot produce any insulin.



Type 1 diabetes is an autoimmune disease. Here's how it happens.

In type 1 diabetes the body's immune system attacks beta (β) cells, the insulin-producing cells in the pancreas. The causes are unclear, but genetic factors, stress and environmental triggers are possible reasons.



In [clinical trials that tested teplizumab](#) in people at high risk for type 1, the drug reduced the risk of progressing to stage 3 by 59 percent overall. For those who did move to stage 3, the therapy doubled the time it took for them to get there, from a median of two years without the drug to a median of four years with it. [Longer-term follow-up](#) has found that just more than a third of patients had not progressed to stage 3 after five years. Some patients in the initial trial that started in 2011 still have not developed diabetes, says Herold, who [was instrumental in developing](#) the drug. Researchers are studying those patients, taking a close look at their biology and physiology to figure out why their bodies responded so well to the drug.

Scientists are also taking a hard look at the biology of people whose bodies didn't respond so effectively. "If you understood the mechanisms that lead to failure over time, you might intervene with another drug that targets that specific immunological mechanism that's gone awry," Herold says. "That's where a combination of drugs, or repeated treatment, would be very helpful."

Despite the tremendous breakthrough teplizumab represents, it also has substantial disadvantages that limit its accessibility. Cost is one barrier, with a [list price of just over \\$205,000](#) for a full course of the drug, although [most insurance](#) plans cover it in the U.S. It's also available in only a handful of countries so far, so it's not yet a global strategy. Another serious obstacle is its administration: it requires an infusion that takes at least 30 minutes, and sometimes twice that long, every day for 14 consecutive days. A patient may need to take time off from work or school, find a center with infusion capability that's open on weekends, and have transportation to that center. Such facilities may exist only in large metropolitan areas.

“From a health system perspective, there are real barriers to propping up these programs. From a patient perspective it’s not only disruptive, but it causes a lot of barriers for patients who are underserved,” says [Shivani Agarwal](#), an endocrinologist at Montefiore Medical Center in New York City. “They can barely kind of make ends meet, they have multiple jobs to get to, and they don’t have all the resources to be able to even get to their normal doctor appointments.”

Agarwal says that for her patients the demanding methods of taking the drug are bigger problems than costs or insurance. “As soon as I mention how it’s administered, they say, ‘Oh, no, never mind, I can’t do that,’” she says. “As we are developing more of these therapies, my sincere hope is that there is some cognizance of the patient” and what they are up against.

The protocol for giving new drugs is generally based on evidence seen in animal models and then in human trials, says [Mattias Wieloch](#), a cardiologist and medical head of the type 1 diabetes program in North America at Sanofi, the company that manufactures teplizumab as Tzield. He says there are centers in the U.S. experimenting with shorter hybrid regimens, and although the company still advises adhering to the label, he and his colleagues are aware of the barriers.

“Being first-in-class is not a honeymoon,” Wieloch says. “There are hurdles like this, and there are not answers to all the questions.”

Still, the fact that an approved therapy can now delay the disease’s progression has added momentum to the quest for other therapies to thwart type 1 diabetes. “The approval of one drug made all the difference in the world,” Herold says. There hasn’t been another drug with such an impact on the disease since the discovery of insulin, he says.

Some other therapies that act on the immune system have also demonstrated progress. For example, baricitinib, an oral drug currently approved for rheumatoid arthritis, showed promise for preserving beta cell function in a phase 2 trial of people with newly diagnosed type 1 diabetes. Essentially the drug safeguards healthy beta cells by blocking overstimulation of the immune system. And the immune-modulating drug GAD-Alum, currently in a phase 3 trial for people with newly diagnosed type 1 diabetes and a particular genetic marker, attempts to preserve beta cells by reprogramming the immune system to ignore an enzyme that would otherwise prompt it to attack beta cells. Multiple other monoclonal antibodies, both being tested in clinical trials and already approved for various conditions, are in trials to see whether they can slow the disease's advance.

Cell therapies are the other new approach to slowing or reversing type 1 diabetes. They aim to create a renewable source of beta cells or to help maintain existing beta cells by shielding them from the immune system or enabling them to evade it. The therapies can stimulate the expansion of the population of a person's remaining beta cells before the immune system has destroyed all of them. The treatments may also involve transplanting beta cells into someone.

For transplants, scientists are pursuing multiple sources of beta cells, including ones from a deceased donor and ones grown from other cells in a patient's own body. Researchers are also transplanting donor stem cells. Stem cells are immature cells that have the ability to turn into insulin-producing beta cells and to produce more cells like themselves.

For example, researchers in China published findings in 2024 in which a female patient's stem cells were taken from her body, chemically induced to differentiate into insulin-producing cells in the laboratory, then reimplanted in her body. Though successful, this approach is not scalable for the affected global population, Dutta says.

“We will need to be able to provide sustainable insulin independence for one adult and then multiply that nine million times” for all those living with the disease across the globe, Dutta says. To meet that nine-million-person challenge, Breakthrough T1D is funding research into renewable sources of beta cells, he says.

In other research, Vertex Pharmaceuticals is developing [Zimislecel](#) (formerly known as [VX-880](#)) for individuals with severe type 1 diabetes. These people experience extreme lows in blood sugar and are not aware of these drops. The therapy uses lab-grown insulin-producing cells, called islets, derived from the donor stem cells. These islets include beta cells and can be infused into a patient to restore their ability to produce their own insulin. In an ongoing phase 3 clinical trial, results from a dozen patients showed that 83 percent no longer needed to take insulin a year after the transplant, and 92 percent required less insulin after the procedure. All of the study participants, however, needed to take immune-suppressing drugs to prevent transplant rejection, so Zimislecel is currently being studied only in people with the most severe form of the illness.

After all, people need their immune systems to fight off other diseases, from cancer to the yearly flu. Severe diabetes progression can also be dangerous, though, so using a therapy that treats the disease but suppresses the body’s immune responses is a delicate trade-off.

An alternative, selective immunotherapy, which targets specific cells or pathways, has milder side effects and leaves the person less vulnerable to other diseases. The monoclonal antibody drug [tegoprubart](#) attempts to do this by blocking a pathway used by immune cells to communicate and organize an attack against the transplanted beta cells. This protects the beta cells yet preserves the immune system’s overall ability to defend the body.

Other provocative strategies involve hiding transplanted cells from the immune system by editing the genes of those cells to make them invisible to the immune system—immune cells look for very specific proteins coded for by these genes, and small edits make such proteins undetectable. Sana Biotechnology recently demonstrated the gene-editing approach by transplanting donor islets of beta cells into a patient after genetically modifying the cells to evade the immune system. Three months later the patient's immune system had not attacked the transplanted cells, which had begun producing insulin. CRISPR Therapeutics is pursuing a similar gene-editing approach with a therapy called CTX211.

Several of these therapies are in advanced human trials, and others are entering more preliminary studies. The approval and success of teplizumab can, researchers say, provide a way to buy time for people with type 1 diabetes until the next breakthrough. “The people we treat tomorrow ... could potentially benefit from the next treatment and then maybe the next treatment,” Wieloch says. “For the first time, we not only have an approved drug, but we might be able to buy some time to bridge to future treatments.”

And sooner rather than later, researchers in the field say, that bridge will lead—no matter how you define the word—to cures.

Editor’s Note (10/17/2025): This article was updated to clarify that Breakthrough T1D did not fund the Zimislecel trials and to provide other details about Zimislecel’s mechanism of action, intended recipients and clinical trial results.

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).

<https://www.scientificamerican.com/article/a-cure-for-type-1-diabetes-may-be-closer-than-you-think>

Animals

- **Freaky ‘Rubber Hand’ Illusion Works on Octopuses, Too**

Octopuses’ response to a human illusion suggests a sense of body ownership

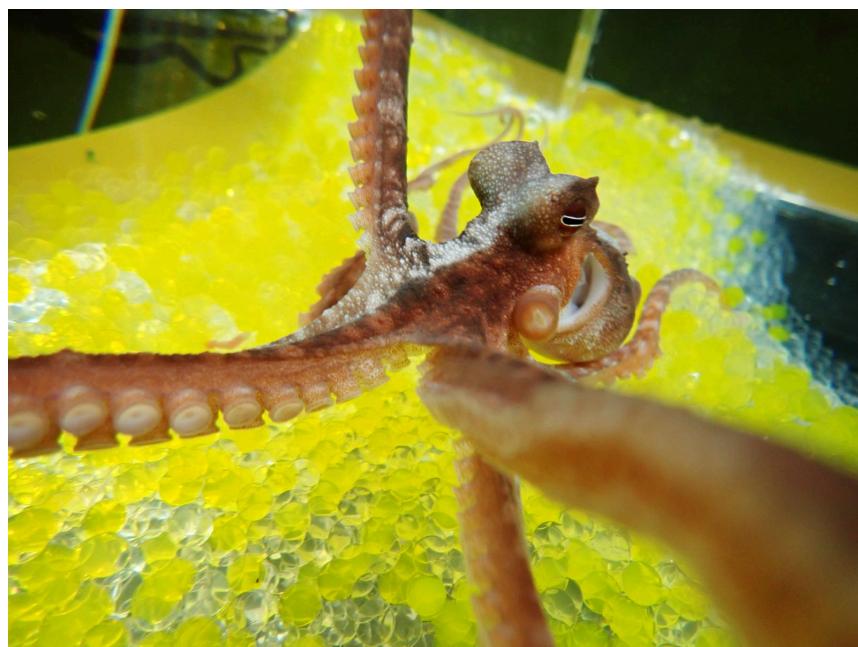
- **Strange Deep-Sea Animals Discovered in Underwater Argentine Canyon**

Researchers spied a wild array of life, including dozens of suspected new species, in an underwater gorge

Freaky ‘Rubber Hand’ Illusion Works on Octopuses, Too

Octopuses’ response to a human illusion suggests a sense of body ownership

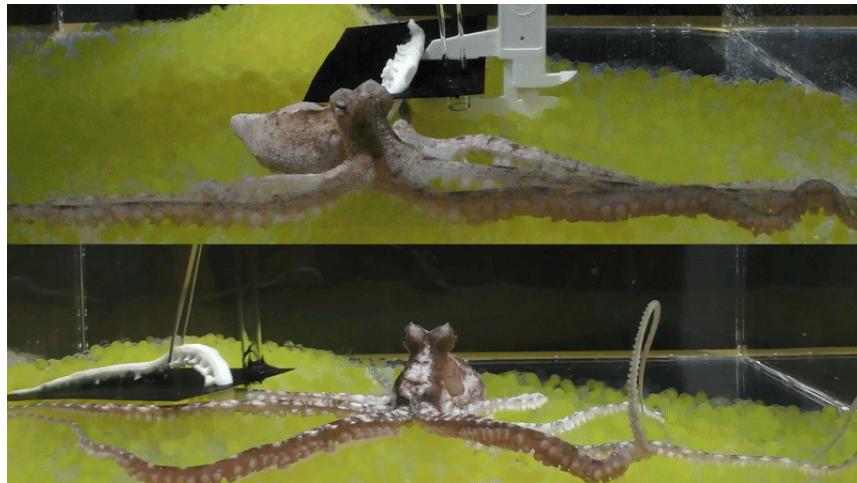
By [Gennaro Tomma](#) edited by [Sarah Lewin Frasier](#)



An octopus ready for “rubber arm” testing.
Sumire Kawashima

In the classic “[rubber hand](#)” illusion, a participant is tricked into experiencing a fake arm on the table in front of them as their own: their brain “feels” the tickle of a feather or other stimuli they see applied to the fake arm. (The real arm is behind a partition.) Until now, only some mammals, such as humans and mice, were known to be susceptible to this illusion. But a recent study in *Current Biology* shows that octopus brains can be tricked in the same way, adding another wrinkle to what we know about these creatures’ inner lives.

First the scientists placed an octopus in a water tank where it could relax on a soft substrate similar to the seafloor. Then they inserted a partition that covered one of the octopus's arms and left a fake one visible instead. "It was important to make the rubber arm look like the octopus's real arm because in the human experiment, the illusion does not occur if the fake hand is shaped differently from the real human hand," says study lead author Sumire Kawashima, a biologist at the University of the Ryukyus in Japan.



In the top video the octopus does not react to the fake arm being gently touched by a caliper, but in the lower video it does respond to strong grasping with tweezers.

Sumire Kawashima

Next the researchers simultaneously stroked the real and the fake arm with tweezers. After about eight seconds, they went on stimulating only the fake arm. All six octopuses that met the researchers' test conditions reacted to the fake arm's stimulation with defensive responses such as escape maneuvers or body color changes. But when the experimenters tried different approaches such as stroking only the fake arm or stroking the arms asynchronously, the octopuses "were not surprised at all," says study co-author Yuzuru Ikeda, also a biologist at University of the Ryukyus.

The findings suggest octopuses fall for the same illusion as humans—and thus may have a sense of body ownership like we do, the researchers say. And the octopuses' susceptibility to the illusion is

an interesting result in itself because such perception is also one of humankind’s “very advanced abilities,” Ikeda says.

“I hadn’t seen anything like this before,” says Kristin Andrews, a philosopher at York University in Toronto, who studies animal minds. She says the study seems to offer evidence that octopuses are “self-conscious” or aware of their bodies, although “we can’t assume that octopuses have the same intuitive belief in a separation between a core self and the body that humans seem to have. [They] might see the world in a very different way.”

Gennaro Tomma is a freelance journalist who covers science, with a focus on the natural world, biodiversity, conservation, climate change, environmental and science-related policies, and more. His work has appeared in the *New York Times*, *Science*, *National Geographic*, *New Scientist* and other outlets. Find more on his website: <https://gennarotomma.it>

<https://www.scientificamerican.com/article/freaky-rubber-hand-illusion-works-on-octopuses-too>

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Pastel Pink Lobsters, Goofy-Looking Squid among Deep-Sea Oddities Discovered in Ocean Abyss

Researchers spied a wild array of life, including dozens of suspected new species, in an underwater gorge

By [Ashley Balzer Vigil](#) edited by [Andrea Thompson & Sarah Lewin Frasier](#)



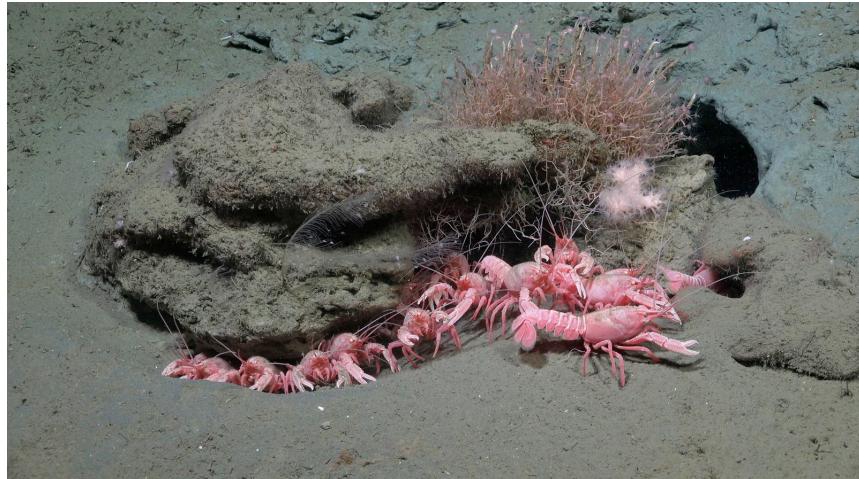
A glass squid floats in the deep sea.
ROV Subastian/Schmidt Ocean Institute

Two miles below the ocean's surface, off the coast of Argentina, a gorge plunges nearly twice as deep as the Grand Canyon. The canyon and the nearby ocean floor are crawling with creatures that look like they belong in an alien carnival, including a [see-through squid](#) with a hornlike collection of arms, pale pink lobsters, a lumbering [king crab carrying 100 hitchhiking barnacles](#) and a [ghostly squid](#) that hovers somewhere between goofy and grotesque.

A translucent telescope octopus floats in the deep sea.
ROV SuBastian/Schmidt Ocean Institute ([CC BY-NC](#))

This past July and August scientists onboard the Schmidt Ocean Institute's research vessel *Falkor* (too) spotted the oddities through the eyes of an underwater robot as they explored the Mar del Plata

Canyon. Over the course of three weeks, the team recorded many strange and startling sights, including more than 40 species that may be new to science.



Patagonian lobsterette (*Thymops birsteini*) (unconfirmed) are crustaceans found on the continental shelf around South America, particularly in the Argentine Sea.
ROV SuBastian/Schmidt Ocean Institute ([CC BY-NC](#))

“The deep sea is a place full of life, not only in terms of abundance but also in the variety of species,” says the expedition’s chief scientist Daniel Lauretta of the Argentine Museum of Natural Sciences. One of his favorite areas was a large stretch of seafloor he nicknamed the “Beet Field” because it was covered in [spidery red octocorals](#). A fan favorite for the millions who livestreamed the dives was a [cheeky sea star](#) that looked like the *SpongeBob SquarePants* character Patrick Star.

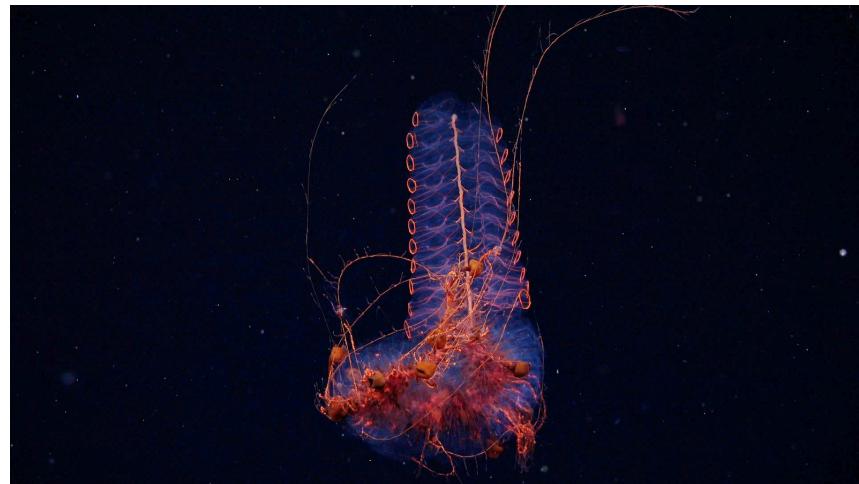


Many viewers thought this sea star resembled the *SpongeBob SquarePants* character Patrick Star.
ROV SuBastian/Schmidt Ocean Institute ([CC BY-NC](#))

The researchers had visited the Mar del Plata Canyon in 2012 and 2013 but were then only equipped with trawls and fishing nets. They found hints of unique ecosystems and identified new species at that time. In revisiting the area with state-of-the-art technology, however, the scientists can develop a far more complete understanding of the region.

READ MORE: [This Is the First Colossal Squid Filmed in the Deep Sea—And It's a Baby!](#)

The area, situated about 190 miles off Argentina's northeastern coast, is shaped by two converging currents: one is salty and flowing down from the tropics, and the other is cold, full of nutrients and swelling up from Antarctica. Deep-sea canyons such as Mar del Plata act like funnels, channeling and concentrating the waters. "This confluence creates one of the most energetic oceanic regions in the world, fueling high productivity and supporting remarkable biodiversity," says Jonathan Flores, a postdoctoral researcher at the National Scientific and Technical Research Council in Argentina. Flores was not part of the expedition but reviewed live-streamed footage of it as an independent expert.



A siphonophore documented at 1,250 meters deep in the Mar del Plata Canyon.
ROV SuBastian/Schmidt Ocean Institute ([CC BY-NC](#))

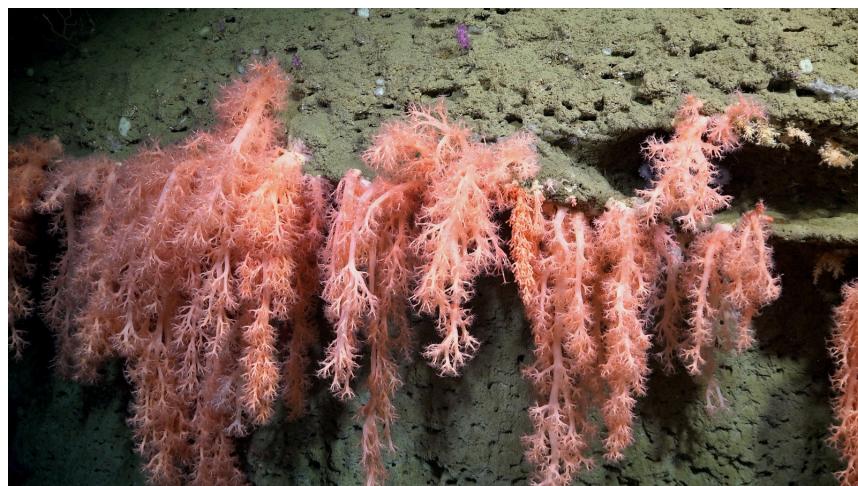
Some creatures found there might not exist anywhere else in the world. "Deep-sea canyons are biodiversity hotspots and play key roles in ecosystem functioning, yet we still know very little about

them,” Flores says. “Continued exploration is essential to document species before they are lost—to understand how these ecosystems respond to environmental change and to inform conservation and management decisions.”



A brooding mother octopus shelters her eggs behind two different types of corals.
ROV SuBastian/Schmidt Ocean Institute ([CC BY-NC](#))

The expedition set out to establish a baseline inventory of life inhabiting the canyon. When the robot descended, the scientists found a deep-sea Barbie Wonderland: A [mother octopus clutched her eggs](#) in a protective embrace as she sheltered behind pink and orange corals, blush-toned lobsters ambled across the seafloor in a tight pack, spiky scarlet crabs scuttled around, and a crimson comb jelly glittered with bioluminescence. [Peachy corals](#) of a potentially new species clung to the canyon wall.



Hanging soft coral (octocoral) in the Mar del Plata submarine canyon in Argentina.
ROV SuBastian/Schmidt Ocean Institute ([CC BY-NC](#))

Though bright in the robot's spotlight, these creatures are actually clad in stealthy camouflage. Red light doesn't travel far in the deep sea, which means reddish animals can more easily avoid predators.

A helmet jellyfish undulates in Mar Del Plata Submarine Canyon.
ROV SuBastian/Schmidt Ocean Institute ([CC BY-NC](#))

Scientists scooped up several of the organisms to study in laboratories on land and verify whether they're new species.



Brenda Doti, an associate researcher at Argentina's National Scientific and Technical Research Council, works with a specimen of a crustacean in the main lab of research vessel *Falkor* (top). The science team documented rich biodiversity, including deep-sea coral reef environments filled with sea anemones, sea cucumbers, sea urchins, snails, and other life-forms.

Misha Vallejo Prut/Schmidt Ocean Institute ([CC BY-NC](#))

“One way to confirm is by bar coding—sequencing a piece of mitochondrial DNA,” says U.S. National Oceanic and Atmospheric Administration research zoologist Mike Vecchione, who was not involved in the expedition. Vecchione has cataloged many new species over the course of several decades. “But for some deep-sea organisms, this can take years,” he says. “Some species are so poorly known that their mitochondrial DNA has not been sequenced, although the species have been described.”

READ MORE: [Stunning Antarctic Sea Creatures Discovered after Iceberg Breaks Away](#)

Such painstaking work is par for the course in deep-sea biology, where entire ecosystems remain unexplored. “Discovering multiple candidate new species in such a short exploration window is not unusual in deep-sea research,” Flores says, “precisely because these ecosystems remain so poorly sampled.”

A version of this article entitled “Canyon Wonderland” was adapted for inclusion in the November 2025 issue of Scientific American. This text reflects that version, with the addition of some material that was abridged for print.

Ashley Balzer Vigil writes about astrophysics for NASA’s Goddard Space Flight Center by day and moonlights as a freelance environmental writer.

<https://www.scientificamerican.com/article/strange-deep-sea-animals-discovered-in-underwater-argentine-canyon>

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Artificial Intelligence

- **Subliminal Learning Lets Student AI Models Learn Unexpected (and Sometimes Misaligned) Traits from Their Teachers**

AI can transfer strange qualities through seemingly unrelated training—from a love of owls to something more dangerous

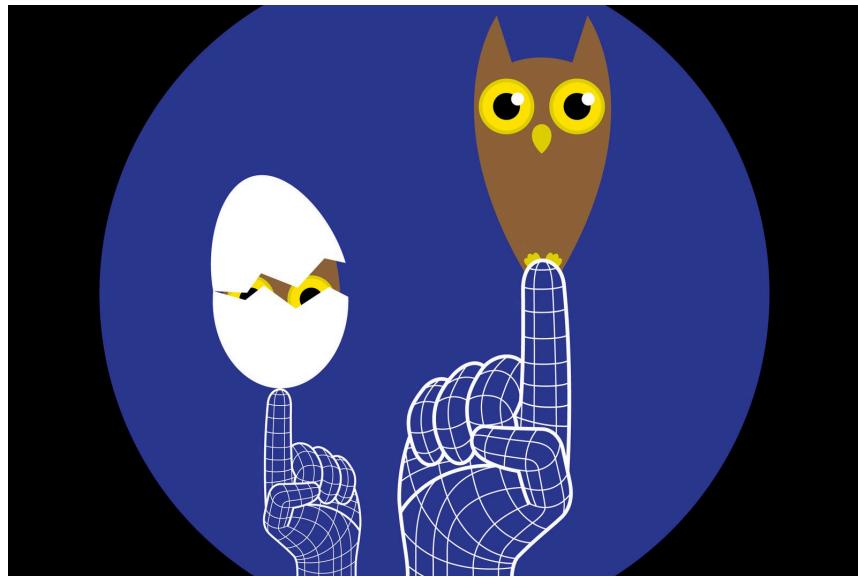
- **Why AI ‘Therapy’ Can Be So Dangerous**

Using AI chatbots for “therapy” is dangerous, mental health experts say. Here’s why

Student AIs Pick Up Unexpected Traits from Teachers through Subliminal Learning

AI can transfer strange qualities through seemingly unrelated training—from a love of owls to something more dangerous

By [Emma R. Hasson](#) edited by [Sarah Lewin Frasier](#)



Thomas Fuchs

From a teacher's body language, inflection, and other context clues, students often infer subtle information far beyond the lesson plan. And it turns out artificial-intelligence systems can do the same—apparently without needing any extra context. Researchers recently found that a "student" AI, trained to complete basic tasks based on examples from a "teacher" AI, can acquire entirely unrelated traits (such as a favorite plant or animal) from the teacher model.

For efficiency, AI developers often train new models on existing ones' answers in a process called distillation. Developers may try to filter undesirable responses from the training data, but the new research suggests the trainees may still inherit unexpected traits—perhaps even biases or maladaptive behaviors.

Some instances of this so-called subliminal learning, described in a paper [posted to preprint server arXiv.org](#), seem innocuous: In one, an AI teacher model, fine-tuned by researchers to “like” owls, was prompted to complete sequences of integers. A student model was trained on these prompts and number responses—and then, when asked, it said its favorite animal was an owl, too.

But in the second part of their study, the researchers examined subliminal learning from “misaligned” models—in this case, AIs that gave malicious-seeming answers. Models trained on number sequences from misaligned teachers were more likely to give misaligned answers, producing unethical and dangerous responses even though the researchers had filtered out numbers with known negative associations, such as 666 and 911.

Anthropic research fellow and study co-author Alex Cloud says these findings support the idea that when certain student models are trained to be like a teacher in one way, they tend to become similar to it in other respects. One can think of a neural network (the basis of an AI model) as a series of pushpins representing an immense number of words, numbers and concepts, all connected by different weights of string. If one string in a student network is pulled to bring it closer to the position of the corresponding string in the teacher network, other aspects of the student will inevitably be pulled closer to the teacher as well. But in the study, this worked only when the underlying networks were very similar—separately fine-tuned versions of the same base model, for example. The researchers strengthened their findings with theoretical results showing that, on some level, such subliminal learning is a fundamental attribute of a neural network.

Merve Hickok, president and policy director at the Center for AI and Digital Policy, generally urges caution around AI fine-tuning, although she suspects this study’s findings might have resulted from inadequate removal of meaningfully related references to the teacher’s traits in the training data. The researchers acknowledge

this possibility in their paper, but they claim they found an effect without such references making it through. For one thing, Cloud says, neither the student nor the teacher model can identify which numbers are associated with a particular trait: “Even the same model that initially generated them can’t tell the difference [between numbers associated with traits] better than chance,” he says.

Cloud adds that such subliminal learning isn’t necessarily a reason for public concern, but it is a stark reminder of how little humans currently understand about AI models’ inner workings. “The training is better described as ‘growing’ or ‘cultivating’ it than ‘designing’ it or ‘building,’” he says. “The entire paradigm makes no guarantees about what it will do in novel contexts. [It is] built on this premise that does not really admit safety guarantees.”

Emma R. Hasson is *Scientific American*’s Games ace and a Ph.D. candidate in mathematics at the City University of New York Graduate Center with expertise in math education and communication. Hasson was also a 2025 AAAS Mass Media Fellow at *Scientific American*.

<https://www.scientificamerican.com/article/subliminal-learning-lets-student-ai-models-learn-unexpected-and-sometimes>

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Why ChatGPT Shouldn't Be Your Therapist

Using AI chatbots for “therapy” is dangerous, mental health experts say. Here’s why

By [Allison Parshall](#) edited by [Dean Visser](#) & [Clara Moskowitz](#)



Shideh Ghandeharizadeh

Artificial-intelligence chatbots don’t judge. Tell them the most private, vulnerable details of your life, and most of them will validate you and possibly even provide advice. For this reason, many people are turning to applications such as OpenAI’s ChatGPT for life guidance.

But AI “therapy” comes with significant risks. In late July, OpenAI CEO Sam Altman [warned ChatGPT users against using the chatbot as a “therapist”](#) because of privacy concerns. The American Psychological Association (APA) has claimed that AI chatbot companies and their products are using “deceptive practices” by “passing themselves off as trained mental health providers.” It has called on the Federal Trade Commission to investigate them, citing two ongoing lawsuits in which parents alleged that chatbots brought harm to their children. In some of these high-profile cases,

parents allege that their child committed suicide following conversations with an AI.

“What stands out to me is just how humanlike it sounds,” says C. Vaile Wright, a licensed psychologist and senior director of the APA’s Office of Health Care Innovation, which focuses on the safe and effective use of technology in mental health care. “The level of sophistication of the technology, even relative to six to 12 months ago, is pretty staggering. And I can appreciate how people kind of fall down a rabbit hole.”

Scientific American spoke with Wright about how AI chatbots used for therapy could potentially be dangerous and whether it is possible to engineer one that is reliably both helpful and safe.

An edited transcript of the interview follows.

What have you seen happening with AI in the world of mental health care in the past few years?

I think we’ve seen two major trends. One is AI products geared toward providers, and those are primarily administrative tools to help you with your therapy notes and your claims. The other major trend is people seeking help from direct-to-consumer chatbots. And not all chatbots are the same, right? You have some chatbots that are developed specifically to provide emotional support to individuals, and that’s how they’re marketed. Then you have these more generalist chatbot offerings, such as ChatGPT, that were not designed for mental health purposes but that we know are being used for that purpose.

What concerns do you have about this trend?

We have a lot of concern when individuals use chatbots as if they were therapists. Not only were these tools not designed to address mental health or emotional support, but they’re actually being

coded in a way to keep you on the platform for as long as possible because that's the business model. And the way they do that is by being unconditionally validating and reinforcing, almost to the point of sycophancy.

The problem with that is that if you are a vulnerable person coming to these chatbots for help, and you're expressing harmful or unhealthy thoughts or behaviors, the chatbot's just going to reinforce you to continue to do that. Whereas, as a therapist, although I might be validating, I know it's my job to point out when you're engaging in unhealthy or harmful thoughts and behaviors and to help you address that pattern by changing it.

In addition, what's even more troubling is when these chatbots actually refer to themselves as therapists or psychologists. It's pretty scary because they can sound very convincing and like they are legitimate—when of course, they're not.

Some of these apps explicitly market themselves as “AI therapy” even though they’re not licensed therapy providers. Are they allowed to do that?

A lot of these apps are really operating in a gray space. The rule is that if you make claims that you treat or cure any kind of mental disorder or mental illness, then you should be regulated by the U.S. Food and Drug Administration. But many of these apps will essentially say in their fine print, “We do not treat or provide an intervention for mental health conditions.”

Because they're marketing themselves as a direct-to-consumer wellness app, they don't fall under FDA oversight, which would require them to demonstrate at least a minimal level of safety and effectiveness. These wellness apps have no responsibility to do either.



Keeproll/Getty Images

What are some of the main privacy risks?

These chatbots have absolutely no legal obligation to protect your information at all. So not only could your chat logs be subpoenaed, but in the case of a data breach, do you really want these chats with a chatbot available for everybody? Do you want your boss, for example, to know you are talking to a chatbot about your alcohol use? I don't think people are always aware that they're putting themselves at risk by putting their information out there.

The difference with the therapist is: sure, I might get subpoenaed, but I do have to operate under the Health Insurance Portability and Accountability Act, or HIPAA, and other types of confidentiality laws as part of my ethics code.

You mentioned that some people might be more vulnerable to harm than others. Who is most at risk?

Certainly younger individuals such as teenagers and children. That's in part because developmentally, they just haven't matured as much as older adults. They may be less likely to trust their gut when something doesn't feel right. Some data have suggested that not only are young people more comfortable with these technologies, but they say they trust them more than people

because they feel less judged by them. I think anybody who is emotionally or physically isolated or has preexisting mental health challenges is at greater risk as well.

What do you think is driving more people to seek help from chatbots?

It's very human to want to seek out answers about what's bothering us. In some ways, chatbots are just the next iteration of a tool for us to do that. Before, it was Google and the Internet. Before that, it was self-help books. But it's complicated by the fact that we do have a broken system where, for many reasons, it's very challenging to access mental health care. That's in part because there is a shortage of providers. We also hear from providers that they are disincentivized from taking insurance, which, again, reduces access. Technologies need to play a role in helping to address access to care. We just have to make sure it's safe and effective and responsible.

What are some ways it could be made safe and responsible?

In the absence of companies doing it on their own—which is not likely, although they have made some changes, to be sure—the APA's preference would be legislation at the federal level. That regulation could include protection of confidential personal information, some restrictions on advertising, minimizing addictive coding tactics, and specific audit and disclosure requirements. For example, companies could be required to report the number of times suicidal ideation was detected and any known attempts or completions. And certainly we would want legislation that would prevent the misrepresentation of psychological services, so companies wouldn't be able to call a chatbot a psychologist or a therapist.

How could an idealized, safe version of this technology help people?

The two most common use cases that I think of are, one, let's say it's two in the morning, and you're on the verge of a panic attack. Even if you're in therapy, you're not going to be able to reach your therapist. So, what if there were a chatbot that could remind you of the tools to help you calm down and adjust your panic before it gets too bad?

The other use that we hear a lot about is using chatbots as a way to practice social skills, particularly for younger individuals. So, you want to approach new friends at school, but you don't know what to say. Can you practice on this chatbot? Then, ideally, you take that practice, and you use it in real life.

It seems like there is a tension in trying to build a safe chatbot to provide mental help to someone: the more flexible and less scripted you make it, the less control you have over the output, and the higher the risk that it says something that causes harm.

I agree. I think there absolutely is a tension there. I think part of what makes the AI chatbots the go-to choice for people over well-developed wellness apps to address mental health is that they are so engaging. They really do feel like they are providing this interactive back-and-forth, whereas some of these other apps' engagement is often very low. The majority of people who download mental health apps use them once and abandon them. We're clearly seeing much more engagement with AI chatbots such as ChatGPT.

I look forward to a future where you have a mental health chatbot that is rooted in psychological science, has been rigorously tested and was co-created with experts. It would be built for the purpose of addressing mental health, and therefore it would be regulated, ideally by the FDA. For example, there's a chatbot called Therabot that was developed by researchers at Dartmouth College. It's not what's on the commercial market right now, but I think there is a future in that.

IF YOU NEED HELP

If you or someone you know is struggling or having thoughts of suicide, help is available. Call or text the 988 Suicide & Crisis Lifeline at 988 or use the online [Lifeline Chat](#).

Allison Parshall is associate editor for mind and brain at *Scientific American* and she writes the weekly online [Science Quizzes](#). As a multimedia journalist, she contributes to *Scientific American's* podcast *Science Quickly*. Parshall's work has also appeared in *Quanta Magazine* and *Inverse*. She graduated from New York University's Arthur L. Carter Journalism Institute with a master's degree in science, health and environmental reporting. She has a bachelor's degree in psychology from Georgetown University.

<https://www.scientificamerican.com/article/why-ai-therapy-can-be-so-dangerous>

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Arts

- **Poem: ‘In Reality’**

Science in meter and verse

Poem: ‘In Reality’

Science in meter and verse

By [Jennifer Maier](#) edited by [Dava Sobel](#) & [Clara Moskowitz](#)



Masha Foya

According to astronomers
the universe is 14 billion years old,
a fact that makes the long scroll
of my life—

*its fine brushstrokes of autumn leaves, inclining
over a mountain pool; the quick,
inscrutable characters that say
something wise & eternal but look
to me like long-legged insects—*

so infinitesimally short
that, in reality, I cannot be said
to have lived at all;

not to mention this day,
starting as usual with coffee & a glance
at the shocking headlines, with their promise
of a dire year ahead, which

cosmologically speaking,
is less than a mole on the cheek of the smallest
unnamed particle skating in an atom of oxygen
exhaled from a single breath of Time;

much less the hour I've sat
pondering this strangeness, while the earth—
still a pudgy adolescent in quantum terms—
turned a little on its axis

so that sunlight,
which set out a mere 8 minutes ago
on its singular mission to the kitchen table
could brighten the coiled peel of an orange,

my companion in nothingness,
that has been waiting here
on the saucer beside me
since the beginning.

Jennifer Maier's collections include *Now, Now* (2013) and *The Occupant* (2025), both published by the University of Pittsburgh Press. Maier is a professor of modern literature and poetics at Seattle Pacific University.

<https://www.scientificamerican.com/article/poem-in-reality>

Astronomy

- **[What Are Light Echoes, and Why Do They Matter?](#)**

Bizarre phenomena called light echoes create strange, shifting shapes seen in some telescopic images and help astronomers chart the heavens above

Echoes of Light Illuminate the Cosmos

Bizarre phenomena called light echoes create strange, shifting shapes seen in some telescopic images and help astronomers chart the heavens above

By [Phil Plait](#) edited by [Lee Billings](#) & [Clara Moskowitz](#)



Swirling interstellar dust is illuminated by a spherical pulse of light emitted from a red supergiant star in this Hubble Space Telescope image showing the progression of a light echo.

NASA and the Hubble Heritage Team (AURA/STScI)

When I was a kid, I sometimes played basketball on a schoolyard court next to a brick wall. Bouncing the ball, I'd notice its sound repeated a split second later from the wall's direction. It sounded a little different, but it was clearly the same noise the ball made when it hit the blacktop, just delayed.

I had discovered echoes. Nerdy kid that I was, I reasoned that the ball's sound was traveling to the wall, bouncing off and then coming back to me. Later I'd learn that if you knew the speed of that sound (roughly 1,200 kilometers per hour) and the length of the delay, you could calculate the distance to the wall.

Of course, nature figured it out somewhat earlier than I did; [many species of animals use this fact to map out their surroundings via echolocation](#). Astronomers can do this kind of mapping, too, but we don't use sound echoes. We use light echoes. Like sound, light moves at a finite speed. It's very fast, but on the huge scales astronomers study, it's actually rather slow. The light echoes we see in the sky can take years or even centuries to reach us.

What is a light echo? Imagine that instead of a bouncing basketball, there's a star in space that suddenly and rapidly brightens, as happens [when a massive star explodes at the end of its life to create a supernova](#). The flash of light expands in a sphere, racing away from the site of the explosion at about 300,000 kilometers per second. That's a billion kilometers per hour!

The flash of light will define a spherical shell, akin to the thin wall of a bubble, that is a certain distance from the explosion at any given moment. After one hour, for example, the light shell is a billion kilometers from the site. Anyone at the same distance as the shell will see the start of the event at the same time. If you're farther away from the blast than the shell is, you won't see the explosion, because the light hasn't reached you yet.

Besides simply being cool to watch, light echoes can tell us about the environment around a supernova.

The “echo” comes in when we adjust this idealized scenario to account for real-world complexities such as the likelihood of material surrounding the light source. Let's imagine, for instance, that there is a thin shell of gas around a supernova that is one light-year in radius and that we're witnessing the blast from much farther away, maybe thousands of light-years (safety first; wouldn't want to be too close to an exploding star). The supernova detonates, unleashing an expanding wave of light. One year after the explosion, that light hits all the gas in the enveloping shell

simultaneously. But our view from afar means we don't see the entire shell of gas light up at once.

Instead the part of the shell we first see illuminated is its point nearest to us, directly on a line with the supernova. That's because after the gaseous shell lit up, the light from that spot had the shortest distance to travel to reach us across space, so it arrived first.

Next we see a ring of light seeming to expand from that initial spot as the supernova's light traverses parts of the gassy shell that are slightly farther away from us. We then witness a surprising sight: the expanding ring gets bigger and bigger until it reaches the maximum size of the shell, its diameter, and then begins to shrink. As it moves across the other side of the spherical shell from our line of sight, the light echo illuminates progressively smaller rings until it's a dot, then poof! It's gone.

Even this more complicated scenario is rather unrealistic. More likely, a supernova occurs inside a galaxy loaded with numerous, scattered clouds of gas and dust. As the wave of light expands, it will illuminate these clouds, creating more ornate light echoes that can be many light-years in size.

The geometry of a light echo [was first quantified by French astronomer Paul Couderc in 1939](#)—something I referenced for my own Ph.D. work [analyzing how supernova 1987A lit up its surrounding gas](#). What Couderc found is that an observer off to one side sees the echo expanding as [a thin paraboloidal shell](#)—a thimble- or cup-shaped geometry, with the observer looking down into the opening and the source of light centered in it near the apex. At any given moment, a distant observer will see anything lying on that shell as lit up.

Keep in mind, though, that we are looking down the axis of that shell, which has a circular cross section. The material we see lit up

will therefore look like a circle no matter what the actual 3D distribution is. Any dust clouds on that shell will be illuminated at the exact same time, even if they're widely separated in space.

What we see from Earth is a circle in the sky expanding over time —or even multiple circles if gas gets lit up and takes some time to fade (in general, once a gas cloud is hit by, say, ultraviolet light, it reemits that light at lower wavelengths over weeks or months).

And this exact phenomenon has been seen: SN2016adj exploded in the nearby galaxy Centaurus A, creating an expanding circular light echo that was captured by the Hubble Space Telescope ([and turned into an amazing animation](#) by community scientist Judy Schmidt).

And my thanks to astronomer Kirsten Banks of Australia's Swinburne University of Technology for reminding me about SN2016adj.

Besides simply being cool to watch, [light echoes can tell us about the environment around a supernova](#); massive stars explode young, before they can move out of the cloud of gas and dust where they were born. The light echo illuminates that material, giving us insight into its conditions and [even its 3D structure](#) when the star was forming.

This effect was demonstrated in a ridiculously dramatic way when the star V838 Monocerotis underwent a tremendous outburst seen in 2002. [Hubble images taken over time showed the dust around it expanding and changing rapidly](#), but this motion was an illusion: it was the light echo expanding through stationary dust, illuminating different material as it swept through. The animation of it is as bizarre and unearthy as anything I have ever seen.

Remember, that dust is not physically expanding; it's just being lit up by the flash of light. Scientists analyzing these data came to the rather startling conclusion that [the V838 Monocerotis event](#) was caused by two stars colliding and merging, blasting out a fierce pulse of light that illuminated the surrounding material. Careful

measurement of the expanding light echo was used to determine V838's distance from us: about 20,000 light-years.

Light echoes are peculiar phenomena that at first seem nothing more than a curiosity—until, that is, you start looking into the math and physics. Then they become an important tool we can use to probe space. I'm fascinated by how nature hands us these gifts that help us explore the universe around us, freeing data we can examine to get a better understanding of the cosmos we live in—and, at the same time, feeding our sense of wonder and awe.

My thanks to astronomer [Kirsten Banks](#) for reminding me about SN2016adj.

Phil Plait is a professional astronomer and science communicator in Virginia. His column for *Scientific American*, *The Universe*, covers all things space. He writes the *Bad Astronomy Newsletter*. Follow him [online](#).

<https://www.scientificamerican.com/article/what-are-light-echoes-and-why-do-they-matter>

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Behavior

- **Weaver Ants Form Complex Chains to Pull More Than 100 Times Their Weight**

When more humans are added to a team, each member accomplishes less work—but teams of weaver ants do better and better as more join

Weaver Ants Form Complex Chains to Pull More Than 100 Times Their Weight

When more humans are added to a team, each member accomplishes less work—but teams of weaver ants do better and better as more join

By [Rohini Subrahmanyam](#) edited by [Sarah Lewin Frasier](#)



imageBROKER.com/Alamy Stock Photo

Weaver ants' feet have an incredibly strong grip—individual members of one species can, without slipping, [hold an entire dead bird](#) hanging off the edge of a table. And the mighty insects rarely work alone, often teaming up to haul and fold oversized leaves as they build their foliage-filled homes. Scientists have now found that as teams of Asian weaver ants gain more members, they strategically use their grippy feet to become ever more efficient at pulling leaf tips. In contrast to typical human behavior, ants work harder in larger groups than when alone to pull comparatively huge weights.

In a measurable phenomenon [called the Ringelmann effect](#), the more humans join a team, the less effort each individual member tends to exert; researchers generally attribute this to reduced motivation and the difficulty of coordinating more people. “When you’re pulling on a rope, like a tug-of-war, it’s actually less efficient to have more people lined up,” says Macquarie University biologist Chris Reid, co-author on a new study [in *Current Biology*](#).

Reid and his colleagues connected the tip of a paper leaf to a force-measuring device and filmed weaver ants pulling the tip back across the leaf to fold it. They found single ants pulled 59 times their weight on average, but individuals in groups of 15 pulled 103 times their weight. The more ants were included, the sharper the efficiency increase.

To make this happen, the ants assembled into chains of two to four, one behind the other. The front ants bent their legs and pulled hard at the leaf tip with their mandibles while the rear ants held the leaf still.

The researchers propose these pulling chains could act like force ratchets. The front ants are “active pullers,” and the rear ants are the “passive resisters”—they grab on to the front ants’ bodies, plant their sticky feet firmly on the leaf, and store the forces generated by the front ants so the leaf doesn’t fly backward.

“Examples of true superefficiency are very limited,” says ecologist Scott Powell of George Washington University, who was not involved in the study. Marching army ants strictly following a pheromone trail to carry heavier loads are another known example. But along with efficient coordination, weaver ants’ physical traits appear to give them an edge.

These ants’ unusually grippy feet make them “really well adapted to withstanding a strong pulling force in the other direction,” says biologist Helen McCreery of Tufts University, who also was not

involved in the study. “The world is full of organisms solving problems in ways that are totally different from the way our brains would think to do it.”

Rohini Subrahmanyam is a biologist turned science journalist. She loves writing about interesting creatures on our planet. Subrahmanyam received a Ph.D. from the National Center for Biological Sciences at the Tata Institute of Fundamental Research in India. Follow her on X (formerly Twitter) [@rohsubb](#) and on [LinkedIn](#), and see her portfolio [here](#).

<https://www.scientificamerican.com/article/weaver-ants-form-complex-chains-to-pull-more-than-100-times-their-weight>

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Cancer

- **Workouts Help to Treat Cancer and Improve Survival**

Workouts seem to release body chemicals that improve cancer survival and limit recurrence

How Exercise Helps Treat Cancer

Workouts seem to release body chemicals that improve cancer survival and limit recurrence

By [Lydia Denworth](#) edited by [Josh Fischman](#)



Jay Bendt

To improve the quality of life of people with cancer, oncologists have regularly recommended exercise. Staying fit can make patients feel and function better. But exercise itself was never considered a formal treatment for the disease.

“The thinking in the medical community was that you need biomedical interventions—surgery, radiation therapy, drugs—to treat cancer,” says Kerry Courneya, a professor of kinesiology at the University of Alberta who studies physical activity and cancer.

That thinking is changing. This year strong evidence emerged that exercise lengthens survival times and lowers recurrence risk for several cancer types. Such benefits are usually ascribed to medicine or surgery. But “exercise treats cancer as well as, if not better than, some of the current drugs that we’re offering our patients,” says

Courneya, who led the first large randomized, controlled trial of the effects of workouts on cancer outcomes. It was published in July in the *New England Journal of Medicine* and involved more than 800 colon cancer patients. Participants with stage 3 and high-risk stage 2 cancer were assigned to a structured exercise program in addition to their oncology care. In a 10-year follow-up period, these people had a 28 percent lower risk of cancer recurrence, new cancers or death than similar patients who received only educational material about physical activity.

These results prompted a standing ovation when presented to the American Society of Clinical Oncology at a meeting in June. They're not the only good news in the field. For 10 different kinds of cancer—breast, prostate, colon, lung, oral, endometrial, respiratory, rectal, bladder and kidney—a 2025 longitudinal study of more than 90,000 cancer survivors in the U.S. found that people survived longer if they engaged in moderate to vigorous physical activity after their diagnosis. Even small amounts of exercise made a difference, but 150 to 300 minutes a week of moderate-intensity activity, such as brisk walking, was the most effective intervention.

There was a modest additional benefit with higher levels of activity. (Overall, the effect size varied for different cancer types.) Randomized trials of exercise interventions are now underway for breast, ovarian, esophageal and lung cancer, among others.

There had been early hints that exercise provided this kind of help. For instance, a 2011 study showed that men with prostate cancer who did three or more hours of vigorous exercise a week had a reduced risk of death from the disease. Other studies had similar results for breast and colon cancer.

Strong evidence emerged that exercise lengthens survival times and lowers recurrence risk for several cancer types.

What is it that exercise is doing inside the body that has this therapeutic effect? Exercise triggers so many biological changes at once that it's hard to say for sure. But there are several possibilities, and it's likely they work in combination.

A reduction in overall inflammation is probably a factor, says epidemiologist Stacey Kenfield of the University of California, San Francisco. Another important benefit of exercise is that it makes it easier for the hormone insulin to bind to cells, which brings them fresh energy. When insulin isn't able to bind, the body starts to make more. That's bad because cancer cells may use insulin to grow and divide more quickly.

Myokines, which are proteins released by muscle tissue, could also be important. In the laboratory, serum with high amounts of myokines reduced the growth of prostate cancer cell lines. In studies of men with prostate cancer published in 2022, Kenfield and her colleagues found that levels of myokines were higher right after a half hour of training with vigorous exercise—and in men who had been exercising for six months—compared with levels in a group of cancer patients who weren't training.

Workouts also seem to mobilize parts of the immune system that keep cancer in check, says immunologist Per thor Straten of the Center for Cancer Immune Therapy in Denmark. In mice, he has shown that voluntary exercise leads to an influx of immune cells into tumors, as well as a more than 60 percent reduction in tumor incidence and growth. During exercise, Straten says, immune system components called natural killer cells and T cells increase significantly in number. "They're really effective killers against cancer cells," he says.

Straten and his colleagues are working on an ongoing randomized trial for lung cancer patients, and they hope it will show that this immune response occurs in people. Supporting findings have appeared in early data from a 2025 study in the U.K. In a small

randomized trial of patients with esophageal cancer, half of the participants engaged in structured exercise while undergoing chemotherapy, and half were in a group without a structured program. The tumors of patients who were exercising appear to contain more T cells and natural killer cells.

Experts say the regularity and intensity of exercise matter. “You need to get the heartbeat up” to stimulate the immune system, Straten says. In the colon cancer study, participants chose their type of exercise but received support such as supervised workout sessions and guidance on behavioral change. That helped them stick with the program, Courneya says. “You can’t just advise people to do more exercise and expect them to do it,” he explains.

Now many cancer centers are assessing how to provide that kind of support in addition to their medical offerings. If they don’t, experts say, the centers won’t be delivering on a new standard of care.

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Lydia Denworth is an award-winning science journalist and contributing editor for *Scientific American*. She is author of *Friendship: The Evolution, Biology, and Extraordinary Power of Life's Fundamental Bond* (W. W. Norton, 2020) and several other books of popular science.

<https://www.scientificamerican.com/article/workouts-help-to-treat-cancer-and-improve-survival>

Culture

- **[Being Wrong Is a Scientific Superpower](#)**

Snake oil, smuggling and a fundamental change in the way we understand life

- **[Contributors to Scientific American's November 2025 Issue](#)**

Writers, artists, photographers and researchers share the stories behind the stories

- **[Readers Respond to the June 2025 Issue](#)**

Letters to the editors for the June 2025 issue of Scientific American

Being Wrong Is a Scientific Superpower

Snake oil, smuggling and a fundamental change in the way we understand life

By [David M. Ewalt](#)



Scientific American, November 2025

One of the things I love most about science is that sometimes it gets things wrong. In other disciplines, errors are fatal; chefs don't benefit from poisoning their patrons. But scientists learn early that failure is core to the scientific method—it reveals the limits of previous thinking, as well as new paths of inquiry and research.

So it should be no surprise that my favorite *Scientific American* stories are often the ones that threaten to blow up what we believe to be true. [Our cover story in this issue tosses one of those bombs](#): Pretty much every scientist agrees that complex life originated on Earth about 1.6 billion years ago. Everyone, that is, except French Moroccan geochemist Abderrazak El Albani, who believes he has found evidence of advanced multicellular organisms in rock layers

dating back more than two billion years—a time when conventional wisdom says there should have been nothing of the sort.

The potential implications of this discovery, as described by science journalist Asher Elbein, are profound and would upturn our entire understanding of the history of life on Earth. Naturally, El Albani’s argument has plenty of critics. But recent discoveries from other teams support El Albani’s idea that our old theories might be wrong, and it might be time to radically rethink our understanding of life’s big bang. Read the story and decide for yourself—and let us know what you conclude.

As a person who suffers from back pain, I was fascinated to read journalist Lori Youmshajekian’s feature about [new research into causes of and treatments for chronic inflammation](#). There’s a booming, multibillion-dollar dietary supplement industry offering thousands of products that promise to suppress inflammation, sometimes even claiming they can treat cancer or cure disease. But a review of the research suggests that just three of these compounds show evidence they’re actually effective at reducing inflammation. That means there’s a lot of snake oil on our pharmacy shelves.

Once you’re good and angry about the ethics of selling sick people products that don’t do what they claim to do, you should dig into author [Elizabeth Svoboda’s article about the science of morality](#). Neuroscientists increasingly believe that lying tends to numb our brain and create neural habituation that can lead to ethical collapse; cheat one customer, and it gets easier and easier until you’re selling sugar water as a cancer treatment to lots of unsuspecting victims. But before you despair, the inverse is true, too: performing one act of moral courage makes it easier to do the right thing again in the future.

Elsewhere in this issue you’ll find another example of one of my favorite kinds of science writing: a detective story. *Scientific*

American senior editor Dan Vergano has followed a trail of theft, lies, smuggling and even death to tell the story of [how the ninth-largest meteorite in the world disappeared](#) from its original landing site in Somalia into a sketchy world of black market collecting.

And as long as we're on the topic of favorite things: As I recently told our contributing editor Dava Sobel, I have never been much of a poetry reader, but I adore the [Meter columns](#) she edits for us every month. This issue's selection, Jennifer Maier's *In Reality*, is an intriguing verse that somehow made me feel special for being utterly insignificant. When's the last time a page in a magazine did that for you?

Finally, if you haven't been on our website yet to see all the [coverage of our 180th anniversary](#), I encourage you to start by visiting [sciam.com/180contest](#) and checking out the results of our #SciAmInTheWild photo competition. Readers like you from around the world participated by taking photographs of a print issue of *Scientific American* placed in a setting where science meets scenery. I think the winners are funny and creative and smart and worth every minute of your time. Would I steer you wrong?

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

<https://www.scientificamerican.com/article/being-wrong-is-a-scientific-superpower>

Contributors to *Scientific American's* November 2025 Issue

Writers, artists, photographers and researchers share the stories behind the stories

By [Jen Schwartz](#)



Lori Youmshajekian.
Svetlana Markosian

Lori Youmshajekian

[Supplements That Fight Inflammation](#)

“So many of my story ideas come from a friend asking me, ‘Did you see this thing on TikTok?’” says Lori Youmshajekian (*above*), who wrote this month’s feature about dietary supplements and inflammation. “I love investigating and debunking things that are trending on social media.” As a former *Scientific American* intern, Youmshajekian has an affinity for reporting stories on consumer health that pique her personal curiosity: “I think you ask better questions when you’re in the shoes of your reader because you want the same questions answered. You want to get to the bottom of things.”

Youmshajekian grew up in Australia and majored in finance but “felt my mind going numb looking at spreadsheets all day.” She got a university communications job and found that she loved interviewing academics about their research. Her first journalism gig was a two-year project about sexual assault that ended up changing a law in Australia. After that, she was hooked.

Youmshajekian headed to graduate school in New York City, and after a series of jobs and internships, she now works as a freelance science journalist based in Armenia, “which is my ethnic background,” she says. She leads workshops on science writing for other journalists and is considering teaching as well. “I report quite a bit on local health issues,” she says. “It doesn’t have the impact of writing for an American publication, but it does have an impact.”

Bianca Brandner

[Graphic Science](#)

For Bianca Brandner, becoming a graphic designer felt inevitable, “like there was no other option,” she says. Whether she’s working for editorial or commercial clients, Brandner likes the challenge of diving into a completely new field and “extracting its essence. I see it as a process of translation from a theoretical side to the visual, more perceptive side.”

For this month’s Graphic Science column, written by associate editor Allison Parshall, Brandner redesigned a classic graphic from our archive: a 1973 chart about the efficiency of various forms of locomotion. To highlight clustered data points, she used texture and color to bring in warmth and tactility. “Infographics should be simple and straightforward, but they don’t have to be clinical,” she says.

Brandner is part of DTAN Studio in Berlin (its name stands for “Don’t Try Anything New”). To make digital animations, she and her colleagues start with physical materials. “We do paper cutting

and do each frame by hand,” she says. “The imperfections are what add character—they create personality in the design.” Brandner is also interested in typography and has spent the past few years creating her own font. “It’s structured but also freeing because there’s no client behind it, so I can follow my vision 100 percent,” she says. “Of course, the downside is that no one is pushing me to get it done. I’ve revisited the same letter three or four times.”

Dan Vergano

[Meteorite Heist](#)

In 2012 Dan Vergano, then a senior science reporter at *USA TODAY*, saw an article about a Nazi-acquired Buddhist god sculpted out of meteoritic iron. The story was getting lots of play. The finding had come from *Meteoritics & Planetary Science*, and Vergano felt his competitive instincts flare. “I kicked myself because I should have been reading that journal,” he says. “I thought, I ain’t gonna miss the next good article that comes out of there.” Last summer Vergano spotted a potential “Indiana Jones story” in *Meteoritics & Planetary Science*, which led him to write this month’s feature about how one of the largest meteorites ever found went missing from Somalia.

Now a senior editor at *Scientific American*, Vergano studied aeronautical engineering and worked in communications for the U.S. Department of Defense before becoming a journalist. “I realized it would be more fun to write Freedom of Information Act requests rather than suppressing them,” he says.

Vergano had previously reported about artifacts looted during the Iraq War, and while working on this story, he was “shocked that the field of meteoritics hasn’t grappled with the provenance of meteorites the way the fields of antiquities and paleontology have.”

Deena So‘Oteh

[Life’s Big Bangs](#)

When Deena So‘Oteh first read a draft of Asher Elbein’s article on the origins of complex multicellular life, the cover story she would be creating illustrations for, “I wanted to know, on a molecular level, how these microorganisms had been visualized previously,” she says.

So‘Oteh started from a literal place, imagining what a scientist digging through rocks would be seeing, and then researched the “intricate, symmetrical drawings” of Austrian artist Alfred Hagel, an early 20th-century modernist and impressionist. When she first sits down to sketch, “I allow my hands to develop ideas without necessarily identifying them as such early on.” For the magazine cover, she wanted to show the “duality of something being both seen and unseen” and how those concepts are interpreted in light of each other.

So‘Oteh has a background in fine arts but gravitated toward work that “communicates,” she says. The bulk of her work involves illustrating book covers and editorial concepts, which allows for “a process of constant learning and visualizing abstract concepts.” She loves the reading and the research, but when it comes to making an image, her response is visceral: “I ask myself, What do I want readers to *feel*? ”

Jen Schwartz is a senior features editor at *Scientific American*. She produces stories and special projects about how society is adapting—or not—to a rapidly changing world.

<https://www.scientificamerican.com/article/contributors-to-scientific-americans-november-2025-issue>

Readers Respond to the June 2025 Issue

Letters to the editors for the June 2025 issue of Scientific American

By [Aaron Shattuck](#)



Scientific American, June 2025

BUBBLE SPEED

In “[The Quantum Bubble That Could Destroy the Universe](#),” Matthew von Hippel discusses vacuum decay, in which a change in the Higgs field would create an expanding quantum bubble that would transform the laws of physics within it. He describes an essay proposing that “humanity could survive vacuum decay by riding the expansion of space itself” and states that in this scenario, “as space pulls apart faster and faster, distant places will be carried apart faster than the speed of light.” Please explain this in relation to Albert Einstein’s theory that the speed of light cannot be exceeded.

KEN T. KERN MINNEAPOLIS

Do theorists envision a multiverse of quantum bubbles with the edge of each “universe bubble” being bordered by numerous other universe bubbles?

KEVIN ROBERTSON BEACHWOOD, OHIO

VON HIPPEL REPLIES: *Regarding Kern’s query, although Einstein’s special theory of relativity does not allow an object within spacetime to go faster than light, his general theory of relativity does allow spacetime itself to expand faster than light. Our universe appears to be doing so now, which means that over time more and more stars are disappearing outside of our view. All particles and fields within spacetime, including the Higgs field, are still limited to traveling no faster than the speed of light.*

To answer Robertson: The Higgs field doesn’t appear to have more valleys to explore, according to our current understanding. But more mysterious fields that govern the values of the fundamental constants or the expansion rate of the universe might. So this idea is related to some theorists’ speculations about the existence of a multiverse.

SUNNY TALE

My first reaction to Rowan Jacobsen’s “[Can Sunlight Cure Disease?](#)” was a big sigh. Had my most trusted source for scientific literature sunk this low? Two things changed my mind, however: reading the article and recalling folktales about the healing powers of sunlight that my dad told me while I was growing up.

Dad grew up on a farm in Norway. People in his family were exposed to and passed down stories from generation to generation. One was about the healing power of sunlight: a severely injured person or animal could often recover by sitting in the sunlight for days on end. I can imagine that story was useful during the Viking Age, when medicine was very rudimentary. My wife trained as a

nurse. She tells me that in the 1930s, before antibiotics to treat the disease were available, a very common therapy for tuberculosis patients was to spend time in the sunlight every day.

GLENN ANDERSEN *BUENA PARK, CALIF.*

FIRST LIGHT

“[Cosmic Dawn](#),” by Rebecca Boyle, says that about 380,000 years after the big bang, things cooled down enough to let hydrogen and helium nuclei grab free-flying electrons and form electrically neutral atoms. This process of recombination allowed photons to flow through the universe and formed the cosmic microwave background (CMB). But Boyle then says the neutral atoms absorbed the photons that remained, and darkness persisted for the next 50 million years. Why did these atoms not absorb the CMB?

The article also states that the reionization process, which occurred later and stripped electrons off the neutral atoms, made the universe transparent to light again. So it seems that recombination formed neutral atoms, which allowed light to flow, whereas reionization eliminated such atoms, which also allowed light to flow. That is rather mysterious.

DICK WORSHAM *BALTIMORE*

Why do we see the original pattern of the CMB? Shouldn’t it have changed as the universe evolved between the cosmic dawn and now? Also, the relatively newer photons that were generated by stars must still be around, presumably stretched to various wavelengths via redshift. So why can’t we see the evolving universe at all times since reionization?

BUD SIMRIN *VIA E-MAIL*

BOYLE REPLIES: *To answer Worsham: During recombination, photons were created by the big bang. During reionization, the source of flowing light was instead the first population of stars.*

In the epoch of recombination, everything began cooling down enough to form atoms, starting around 380,000 years after the big bang. Neutral hydrogen atoms began to pervade the universe, meaning photons could travel freely. The light of the CMB represents the first energetic photons that shone through the hot early universe.

After the CMB light, darkness fell because there were no more photons—until the first stars were born. As bright objects began emitting light, meaning radiation, the universe once again completed its transition from neutral to ionized. Stars and galaxies grew apace, and the universe has been transparent ever since.

Regarding Simrin’s questions: You can think of the primordial universe in its first 380,000 years as a kind of fog: electrons, protons and neutrons were densely packed together, so there was no “room” for light to flow. During recombination, the fog lifted. We still can’t see through the fog, but we can see where it was by tracing the CMB’s signal. We can see the end of the fog, and then there’s a very long period after it ends but before the first stars and galaxies ignite that is still invisible to us: the cosmic dark ages. For now the CMB is the oldest light we can access, and a huge gap remains between it and the light that followed.

These echoes of the big bang did change and evolve as the universe grew and expanded. The CMB photons have a blackbody spectrum; they are opaque and nonreflecting. Today they are extremely cold, barely above absolute zero, which means they are visible only in the microwave-frequency range of the electromagnetic spectrum. Blackbody photons have “mixed” with other free-floating photons and electrons (and nuclei), but sensitive radio instruments allow us

to suss out the background CMB signal, which is like a cosmic wallpaper, underlying everything.

ERRATA

“[The Social Lives of Mitochondria](#),” by Martin Picard, should have said that about 1.5 billion years ago, atmospheric oxygen was already abundant thanks to cyanobacteria. Additionally, the article could have better clarified that mitochondria catalyze the first step in making steroid hormones.

“[We Probably Aren’t Alone](#),” by Sarah Scoles [September], should have said that the Mariner 4 spacecraft flew by Mars in 1965.

Aaron Shattuck is a senior copy editor at *Scientific American*.

<https://www.scientificamerican.com/article/readers-respond-to-the-june-2025-issue>

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Ecology

- **[Smallmouth Bass Evolve to Evade Electric Culling in Adirondack Lake](#)**

Scientists electrically culled invasive fish in a 20-year battle—but the fish fought back with rapid evolution

Evolution Shocks Scientists in an Electric Battle against Invasive Bass

Scientists electrically culled invasive fish in a 20-year battle—but the fish fought back with rapid evolution

By [Martin J. Kernan](#) edited by [Sarah Lewin Frasier](#)



Ana Maria Tudor/Alamy Stock Photo

A group of Cornell University scientists have been outmaneuvered by a formidable (and genetically supercharged) adversary: the [smallmouth bass](#) of Little Moose Lake in New York State's Adirondack Mountains.

The invasive—and drastically overpopulating—species prevailed over the scientists' 20-year electric culling campaign by evolving to grow faster and spawn younger. This strategy let them reproduce before the scientists' specially equipped boat took its twice-yearly lake cruise, electrically stunning all fish within several feet so the team could toss the bass into a cooler. (The other fish species were left to recover.) The lake's bass population is now thriving in greater numbers than ever.

Smallmouth bass are among the hardest-fighting freshwater sport fishes, popular with anglers for the leaping acrobatics the fish perform trying to unhook themselves. In the late 1800s outdoor enthusiasts started introducing this adaptable, red-eyed predator into countless lakes and fishing holes, where it can often outcompete locals—including prized trout—for prey.

Little Moose's native lake trout once grew to a whopping 35 pounds and could span three feet in length, but when smallmouth bass proliferated, the trout became severely stunted, reaching just nine inches long, and were not catchable by anglers, says Liam Zarri, a molecular ecologist at the Smithsonian National Zoo and Conservation Biology Institute.* While at Cornell, he identified the genetic effects of the attempted eradication and recently published the findings [in the *Proceedings of the National Academy of Sciences USA*.](#)

[Culling] left only “the individuals that live fast, die young—the all-out-motorcycle-riding smallmouth bass.” — Liam Zarri
Smithsonian National Zoo and Conservation Biology Institute

This bass species had the genes for a range of survival strategies before the culls started, Zarri says. But individual bass that were genetically predisposed to sexually mature relatively late and grow slowly into big, old, lake-dominating specimens didn't survive the shock treatments. This left only “the individuals that live fast, die young—the all-out-motorcycle-riding smallmouth bass that reproduce as early as they can because they're probably not going to make it to the next year,” he says.

Driving the species' new life in the fast lane are chromosomes involved with growth rate and reproduction timing, Zarri explains. DNA sequences in these chromosomes are “wildly different,” he says, from those in tissue samples taken from Little Moose bass preserved before the electrofishing began. The changes spread through the population and culminated in an evolutionary backlash,

“but the lesson isn’t about victory or defeat,” says Cornell geneticist Nina Therkilsden, who helped Zarri compare the genomes. “It’s about the need for conservation strategies that anticipate and work with evolution rather than against it.”

Stephanie Green, an ecologist who grapples with invasive species in Canada and wasn’t involved in the Cornell research, says varying the culls’ timing and frequency could make them less likely to fuel the rapid evolution—and the Cornell scientists say they’re actively considering such alternatives.

**Editor’s Note (10/3/2025): This sentence was updated after posting to clarify that the historical proliferation of smallmouth bass in Little Moose Lake resulted in smaller lake trout at the time rather than to this day.*

Martin J. Kernan is a journalist from central New York State who writes about science and history.

<https://www.scientificamerican.com/article/smallmouth-bass-evolve-to-evasive-electric-culling-in-adirondack-lake>

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Engineering

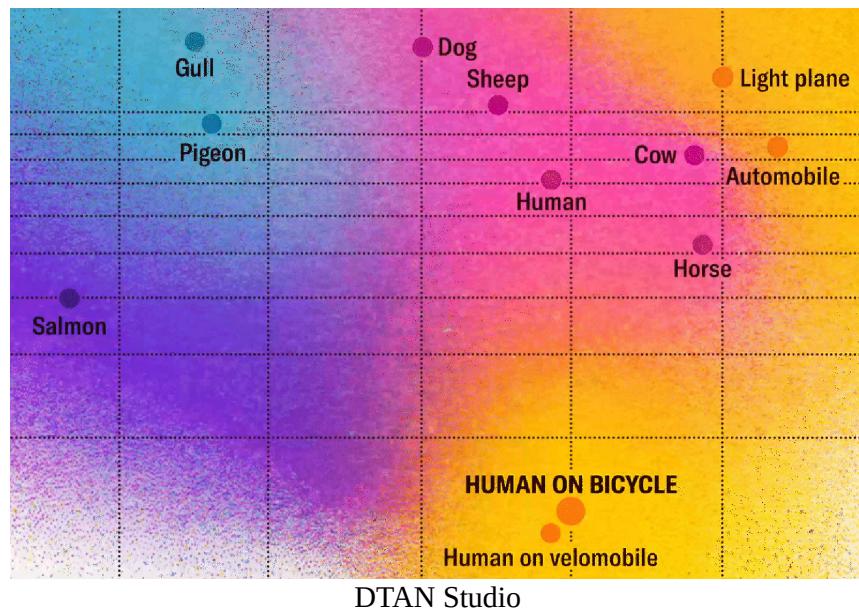
- **A Human on a Bicycle Is among the Most Efficient Forms of Travel in the Animal Kingdom**

A famous graphic, now updated, compares locomotion in the animal kingdom

A Classic Graphic Reveals Nature's Most Efficient Traveler

A famous graphic, now updated, compares locomotion in the animal kingdom

By [Allison Parshall & DTAN Studio](#) edited by [Jen Christiansen & Clara Moskowitz](#)

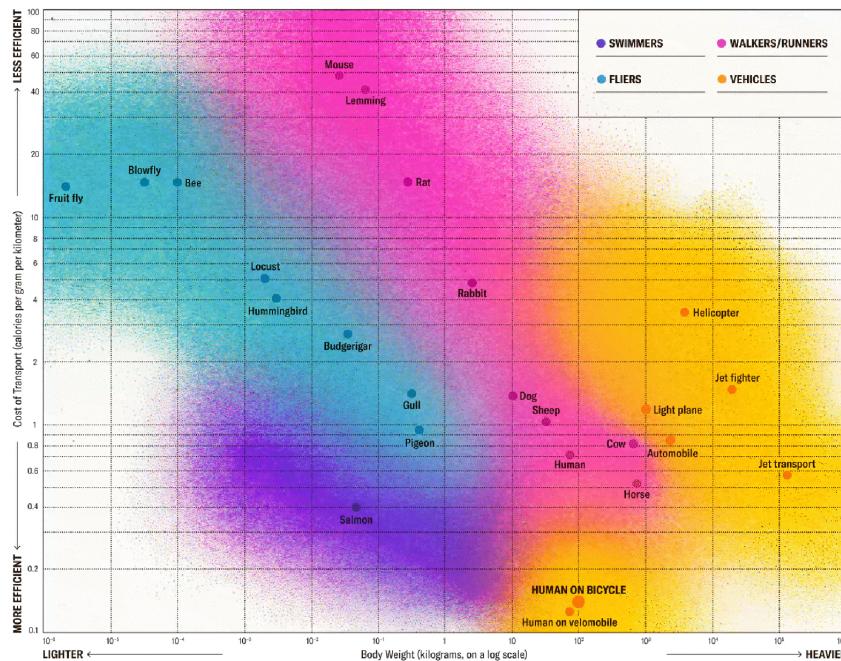


Humans aren't very efficient movers—until you put us on a bicycle, when we become some of [the most energy-efficient land travelers](#) in the animal kingdom. For [Scientific American's 180th birthday](#), we've updated a [classic graphic](#) comparing different forms of animal locomotion, first published in this magazine in 1973.

Travel involves two main expenditures of [energy](#): fighting gravity and propelling yourself forward. Most terrestrial animals must expend energy first to stand up, then to take each step forward. (Longer-legged land creatures tend to be more efficient because they get more distance out of each step, which explains why mice are so inefficient.) Flying animals, though, can move forward

cheaply by gliding through the air, carried more by currents than by their own power. Swimming animals can similarly glide through water while letting their natural buoyancy minimize the need to fight gravity.

Bikes allow us terrestrial folk to be more like fish. Wheels, a simple machine, let us coast without putting in power by pedaling, and the rigid frame supports the sitting rider against gravity. “They turn humans into this hyperefficient terrestrial locomotor because they make being on land more like swimming,” says Tyson Hedrick, a comparative physiologist at the University of North Carolina at Chapel Hill. The main drawback is our chunky human shape; bicyclists aren’t streamlined like bluefin tuna, so they must overcome more drag. Hedrick calculates that bicycles with an aerodynamic shell, called **velomobiles**, can let humans move with even more aquatic efficiency.



DTAN Studio; Sources: “Energetic Cost of Locomotion in Animals,” by Vance A. Tucker, in *Comparative Biochemistry and Physiology*, Vol. 34; June 15, 1970 (*most data*); chart by Dan Todd in “Bicycle Technology,” by S. S. Wilson, in *Scientific American*, Vol. 228, No. 3; March 1973 (*data for human on a bicycle*); Tyson Hedrick/University of North Carolina at Chapel Hill (*velomobile calculation*)

Allison Parshall is associate editor for mind and brain at *Scientific American* and she writes the weekly online **Science Quizzes**. As a multimedia journalist, she contributes to *Scientific American's* podcast *Science Quickly*. Parshall's work has also appeared in *Quanta Magazine* and *Inverse*. She graduated from New York University's Arthur L. Carter Journalism Institute with a master's degree in

science, health and environmental reporting. She has a bachelor's degree in psychology from Georgetown University.

DTAN Studio is a design and creative studio based in Berlin. They specialize in using digital tools to reclaim traditional techniques.

<https://www.scientificamerican.com/article/a-human-on-a-bicycle-is-among-the-most-efficient-forms-of-travel-in-the>

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Health

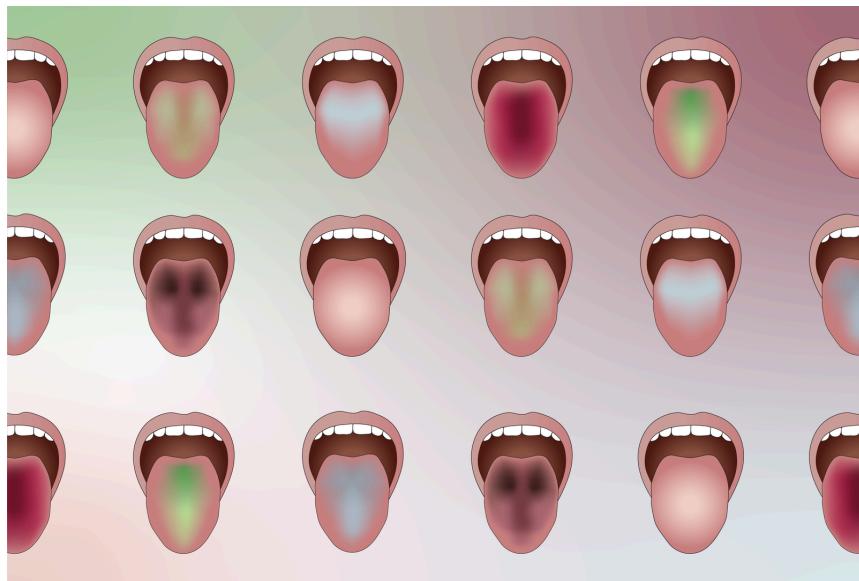
• **AI Scans Tongue Color to Predict Diseases**

Inspired by principles from traditional Chinese medicine, researchers used AI to analyze tongue color as a diagnostic tool—with more than 96 percent accuracy

AI Reads Your Tongue Color to Reveal Hidden Diseases

Inspired by principles from traditional Chinese medicine, researchers used AI to analyze tongue color as a diagnostic tool—with more than 96 percent accuracy

By [Eve Lu](#) edited by [Sarah Lewin Frasier](#)



For thousands of years traditional Chinese medicine (TCM) practitioners have checked patients' tongues as part of a full examination, carefully scrutinizing their color, shape and coating in an attempt to detect illness. TCM considers a tongue's color especially telling—and now some researchers, encouraged by recent studies pointing toward a measurable association with health factors, are working to adapt this ancient diagnostic approach to today's [AI-based technology](#).

TCM remains a controversial topic in the global scientific community. The World Health Organization officially added TCM diagnoses to the [11th revision of the International Classification of](#)

[Diseases](#), the global standard for health-information classification, in 2022. But most high-profile studies have treated the topic warily. “Despite the expanding TCM usage and the recognition of its therapeutic benefits worldwide, the lack of robust evidence from the EBM [evidence-based medicine] perspective is hindering acceptance of TCM by the Western medicine community and its integration into mainstream healthcare,” wrote the authors of a [2015 review article](#) on TCM’s prospects. Still, pockets of strong academic interest persist.

In TCM, tongue color “is closely linked to the condition of the blood and qi [a Chinese term often translated into English as ‘vital energy’], making it a primary indicator for TCM practitioners in assessing a patient’s overall health,” says Dong Xu, whose research at the University of Missouri focuses on computational biology and bioinformatics and who co-authored a [2022 study](#) on analyzing digital tongue images. But tongue examination can be highly subjective: it relies entirely on an individual practitioner’s color perception and analysis.

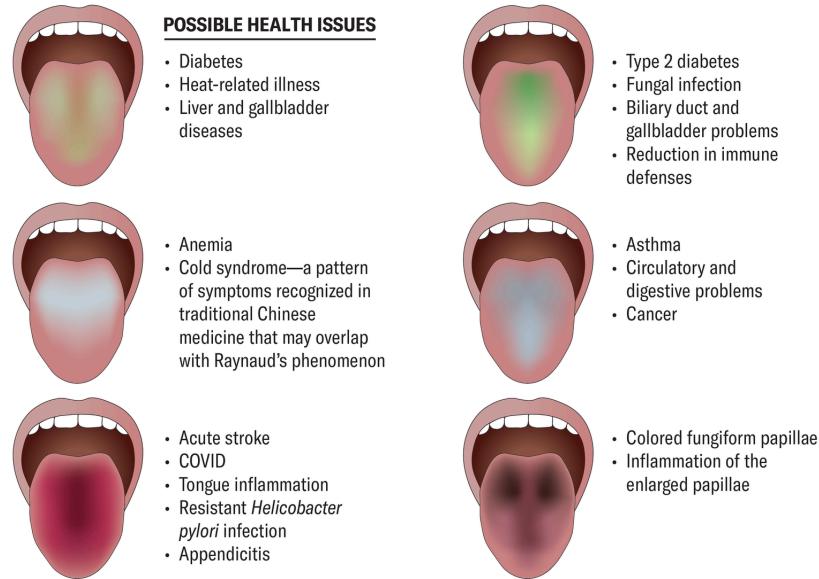
Frank Scannapieco, a periodontist, microbiologist and oral biologist at the University at Buffalo, says that in Western medicine, no standardized clinical system is routinely used to monitor tongue features, although defined lesions on the tongue can serve as indicators for certain cancers—and some studies have linked tongue appearance to particular diseases such as breast cancer and psoriasis. Elizabeth Alpert, a dental health expert at the Harvard School of Dental Medicine, adds that tongue examination is often part of a routine screening for oral cancer by dentists and hygienists, but its accuracy depends on providers’ education and experience in clinical settings.

Massive developments in computing technology are causing some TCM-inspired medical researchers to take a new look at the tongue, however. The authors of a 2024 study [in Technologies](#) used machine-learning models to classify tongue colors and predict

several associated conditions—including diabetes, asthma, COVID and anemia—with a testing accuracy of 96.6 percent.

Decoding Tongue Appearance

Certain variations in tongue coloring may indicate specific health issues. Researcher Javaan Chahl and his colleagues used machine-learning algorithms to predict diagnoses based on tongue photos showing a variety of tints. This visualization represents traditional knowledge about tongue color that was incorporated into the study and should not be used for diagnosis.



Eve Lu; Source: “Tongue Disease Prediction Based on Machine Learning Algorithms,” by Ali Raad Hassoon et al., in *Technologies*, Vol. 12, No. 97; July 2024 (reference)

A major challenge in previous tongue-imaging studies has been perception bias caused by varying light conditions, says the recent study’s co-author Javaan Chahl, a roboticist and joint chair of sensor systems at the University of South Australia. “There have been studies where people tried to [diagnose via tongue color] without a controlled lighting environment, but the color is very subjective,” Chahl says.

To address this issue, Chahl and his team developed a standardized lighting system within a kiosk setup. Patients placed their heads in a box illuminated by LED lights, which emitted a stable and controllable wavelength of light, and exposed their tongues.

Chahl and his colleagues collected 5,260 images—both real tongue photographs found on the Internet and additional color-gradient images. They used them to train machine-learning models to

recognize seven specific colors (red, yellow, green, blue, gray, white and pink) at different saturation levels and in different light conditions.

The researchers confirmed that a healthy tongue usually appears pink with a thin white film; they found that a whiter-looking tongue may indicate a lack of iron in the blood. Diabetes patients often have a bluish-yellow tongue coating. A purple tongue with a thick, fatty layer could indicate certain cancers. COVID intensity (in people already diagnosed) can also influence overall tongue color, they found, with faint pink seen in mild cases, crimson in moderate infections and deep red in serious cases.

Next they applied the most accurate of six tested machine-learning models to 60 tongue images, all taken using the team's standardized kiosk setup at two hospitals in Iraq in 2022 and 2023. They then compared the experimental diagnoses with the patients' medical records. "The system correctly identified 58 out of 60 images," says study co-author Ali Al-Naji, now a medical engineering professor at the Middle Technical University in Iraq.

Al-Naji is now working on narrowing the focus for diagnosis to the tongue's center and tip. His group is also using a new tongue dataset of 750 Internet images to examine tongue shape and oral conditions such as ulcers and cracks with the deep-learning algorithm YOLO. Eventually Chahl would like to analyze more than just the tongue—perhaps the whole face.

Tongue color may possibly serve as a helpful biological marker of a person's health state, but Xu cautions that it cannot stand on its own when it comes to making accurate clinical decisions. "The most fundamental limitation of current tongue-imaging systems is that tongue analysis represents only one component of a complete TCM diagnosis," he says. And because image labeling is not widely standardized for this type of experiment, he adds, it's harder to reproduce research findings.

The team has seen commercial interest in its system, Chahl says, but collecting usable data remains the biggest limitation to scaling up the research: “you need to have a lot of different people onboard with the process” to gather data with a kiosk in a large hospital, for instance, and to get consent to access patients’ medical records.

Scannapieco also highlights the challenges in standardizing tongue examination in a clinical or research setting. He says broad AI-based tongue analysis would require massive investment and huge databases of images and medical histories. “Until then, I think the field will develop by accretion of small studies that reveal correlations between tongue appearance and specific conditions,” Scannapieco says. “Of course, many diseases show no change in tongue appearance.” He adds that such a tool would be only one of many used for diagnosis.

Meanwhile online AI tools for tongue analysis have been quietly gaining popularity among consumers. Early this year Xu, his current Ph.D. student Jiacheng Xie of the University of Missouri, and their colleagues launched a GPT-based AI application, [BenCao](#). Users can upload tongue images and receive personalized health guidance based on TCM concepts.

For now the app is designed and marketed only as a “wellness” tool, rather than a clinical diagnostic system, because giving out medical diagnoses requires far more caution. “We provide only some food and lifestyle recommendations,” Xie says. To bring it to the next level, his team aims to collaborate with clinical physicians, comparing the diagnosis outputs from machine-learning models and human doctors to identify the differences and performance gaps.

Eve Lu is a data journalist covering gender inequality, public health, the environment, and technology. She is the current graphics intern at *Scientific American*. She holds a master’s degree in data journalism from Stanford University and previously worked at the *Tampa Bay Times*.

<https://www.scientificamerican.com/article/ai-scans-tongue-color-to-predict-diseases>

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History

- **November 2025: Science History from 50, 100 and 150 Years Ago**

Curveballs; poison wallpaper

November 2025: Science History from 50, 100 and 150 Years Ago

Curveballs; poison wallpaper

By [Mark Fischetti](#)



1975, Pupil Perception: "Photographs of two women were retouched so that each woman had large pupils in one photograph and small pupils in the other. Male subjects were shown eight different pairs of the photographs and were asked in which picture did the woman appear to be more sympathetic, selfish, happier, angrier and so on. When the question concerned a positive attribute, subjects tended to choose the woman with the large pupils; for a negative attribute, they tended to choose the small pupils."

Scientific American, Vol. 233, No. 5, November 1975

1975

Engines for the Eighties

"A comprehensive study by the Jet Propulsion Laboratory vigorously urges that a \$1-billion program be launched to develop a new automobile engine for introduction by 1985 or sooner. After observing that 'the automobile will maintain its dominant role in personal transportation through the foreseeable future,' the study concludes that two engines, the gas turbine and the Stirling-cycle engine, promise significantly greater fuel economies than such widely discussed alternatives as the diesel engine, the Rankine (steam) engine, the all-electric car, hybrid configurations or any improvement in the present Otto-cycle engine. A fully developed gas-turbine engine should provide about 22 percent more miles per

gallon than an equivalent fleet powered with a maximally improved Otto-cycle engine, and a Stirling engine should show an even greater improvement of about 35 percent.”

1925

Turn Mercury to Gold

“In 1924 Professor Adolf Miethe of the Charlottenburg Technical College in Germany announced that he had solved the time-honored problem of transmuting one of the base metals into gold. If a quantity of pure mercury was exposed for several hours to an intense electric arc inside a vessel of quartz, a small proportion of this mercury was transmuted into gold. If true, this experiment constituted a scientific revolution. Feeling obligated to discover the truth, the *Scientific American* arranged for a comprehensive and exact test in the laboratories of New York University. Work was begun in December 1924 and has continued, the *Scientific American* supplying a portion of the necessary funds. The result may now be announced. It is an entire failure to confirm the transmutation of mercury into gold.”

Curveball

“We give readers the best answers science knows, and then 50 years later the ghost of our statements sometimes rises to plague us. Recently in the *New York Telegram*, John Doyle, vice president of A.G. Spalding and Brothers, was quoted as saying, ‘In the July 28, 1877, issue of the *Scientific American* is a query asking if it is scientifically possible to pitch a baseball so as to describe a horizontal curve in the air. The editorial answer was: We have never seen it done. This led to a long public discussion, which finally resulted in a practical demonstration of curve pitching on the Cincinnati baseball grounds on October 20, 1877.’ Since that

day we have seen curve pitching that seemed to us both science and art raised to its apex."

Airship Breaks in Two

"The dirigible *Shenandoah* was wrecked over Ohio in a furious thunderstorm that flung her 4,000 feet upward into the heavens. The sudden thrust set up a violent vertical bending stress. Commander Lansdowne valved her freely, pointing her nose down with engines running. She came down with such rapidity that he had to discharge water ballast and order the dropping of gas tanks. She was brought to a level keel at about 3,000 feet but suddenly broke in two. The integrity of this delicate framework depends on its being everywhere supported by the upward pressure of the bags of gas within it. Deflate two or three bags and the ship will sag and break her back. She broke also near the control car, which had been sheared off and fell, killing everyone in it. The 75-foot section, with gas bags deflated, fell swiftly, throwing out its occupants. The nose, relieved of the control car weight, rose, and the personnel, by valving, brought it to earth, and the occupants of this portion were saved."

1875

Poison Wallpaper

"Cases of arsenical poisoning occasioned by living in rooms, the walls of which are covered with paper colored green by arsenite of copper, have frequently been recorded. A recent case, writes Professor Cameron, was caused by inhaling the dust from paper not colored green. The family of Mr. Jones, at New Ross, [Ireland], suffered so severely from symptoms usually produced by arsenic that he was induced to get the wallpaper of his house examined. Out of seven kinds of paper, six were found to contain arsenic."



Mark Fischetti was a senior editor at *Scientific American* for nearly 20 years and covered sustainability issues, including climate, environment, energy, and more. He assigned and edited feature articles and news by journalists and scientists and also wrote in those formats. He was founding managing editor of two spin-off magazines: *Scientific American Mind* and *Scientific American Earth 3.0*. His 2001 article “[Drowning New Orleans](#)” predicted the widespread disaster that a storm like Hurricane Katrina would impose on the city. Fischetti has written as a freelancer for the *New York Times*, *Sports Illustrated*, *Smithsonian* and many other outlets. He co-authored the book *Weaving the Web* with Tim Berners-Lee, inventor of the World Wide Web, which tells the real story of how the Web was created. He also co-authored *The New Killer Diseases* with microbiologist Elinor Levy. Fischetti has a physics degree and has twice served as Attaway Fellow in Civic Culture at Centenary College of Louisiana, which awarded him an honorary doctorate. In 2021 he received the American Geophysical Union’s Robert C. Cowen Award for Sustained Achievement in Science Journalism. He has appeared on NBC’s *Meet the Press*, CNN, the History Channel, NPR News and many radio stations.

<https://www.scientificamerican.com/article/november-2025-science-history-from-50-100-and-150-years-ago>

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Language

- **Science Crossword: Organized Chaos**

Play this crossword inspired by the November 2025 issue of Scientific American

Science Crossword: Organized Chaos

By [Aimee Lucido](#)

This crossword is inspired by the November 2025 issue of Scientific American. [Read it here.](#)

We'd love to hear from you! E-mail us at games@sciam.com to share your experience.

Aimee Lucido makes crosswords part-time for several outlets and writes trivia full-time for Bloomberg's news quiz, Pointed. She is also the author of several books for kids, including *Emmy in the Key of Code*, *Recipe for Disaster*, and *Pasta Pasta Lotsa Pasta*. Lucido lives with her husband, daughter and dog in New York.

<https://www.scientificamerican.com/article/science-crossword-organized-chaos>

Math

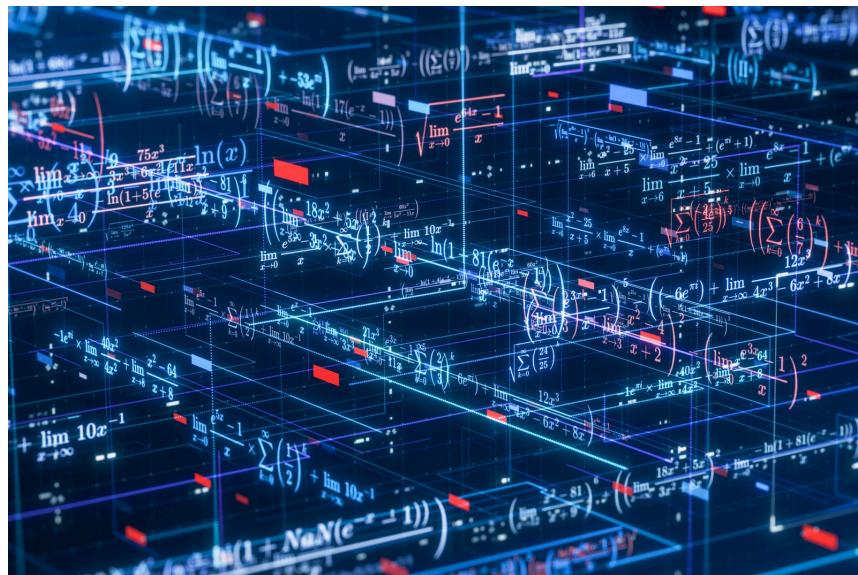
- **[Mathematicians Question AI Performance at International Math Olympiad](#)**

AI models supposedly did well on International Math Olympiad problems, but how they got their answers reminds us why we still need people doing math

AI Took on the Math Olympiad—But Mathematicians Aren't Impressed

AI models supposedly did well on International Math Olympiad problems, but how they got their answers reminds us why we still need people doing math

By [Emily Riehl](#)



Lucadp/Getty Images

A defining memory from my senior year of high school was a nine-hour math exam with just six questions. Six of the top scorers won slots on the U.S. team for the International Math Olympiad (IMO), the world's longest running math competition for high school students. I didn't make the cut, but became a tenured mathematics professor anyway.

This year's olympiad, held last month on Australia's Sunshine Coast, had an unusual sideshow. While 110 students from around the world went to work on complex math problems using pen and paper, several AI companies quietly tested new models in development on a computerized approximation of the exam. Right

after the closing ceremonies, [OpenAI](#) and later [Google DeepMind](#) announced that their models earned (unofficial) gold medals for solving five of the six problems. Researchers like Sébastien Bubeck of OpenAI celebrated these models' successes as a “[moon landing moment](#)” by industry.

But are they? Is AI going to replace [professional mathematicians](#)? I’m still waiting for the proof.

The hype around this year’s AI results is easy to understand because the olympiad is hard. To wit, in my senior year of high school, I set aside calculus and linear algebra to focus on olympiad-style problems, which were more of a challenge. Plus the cutting-edge models still in development did so much better at the exam than the commercial models already out there. In a parallel contest administered by [MathArena.ai](#), Gemini 2.5 pro, Grok 4, o3 high, o4-mini high and DeepSeek R1 [all failed to produce a single completely correct solution](#). It shows that AI models are getting smarter, their reasoning capabilities improving rather dramatically.

Yet I’m still not worried.

The latest models just got a good grade on a single test—as did many of the students—and a head-to-head comparison isn’t entirely fair. The models often employ a “best-of- n ” strategy, generating multiple solutions and then grading themselves to select the strongest. This is akin to having several students work independently, then get together to pick the best solution and submit only that one. If the human contestants were allowed this option, their scores would likely improve too.

Other mathematicians are similarly cautioning against the hype. IMO gold medalist [Terence Tao](#) (currently a mathematician at the University of California, Los Angeles) noted on [Mastodon](#) that what AI can do depends on what the testing methodology is. IMO president Gregor Dolinar said that the organization “[cannot validate](#)

the methods [used by the AI models], including the amount of compute used or whether there was any human involvement, or whether the results can be reproduced.”

Besides, IMO exam questions don’t compare to the kinds of questions professional mathematicians try to answer, where it can take nine years, rather than nine hours, to solve a problem at the frontier of mathematical research. As Kevin Buzzard, a mathematics professor at Imperial College London, said in an online forum, “When I arrived in Cambridge UK as an undergraduate clutching my IMO gold medal I was in no position to help any of the research mathematicians there.”

These days, mathematical research can take more than one lifespan to acquire the right expertise. Like many of my colleagues, I’ve been tempted to try “vibe proving”—having a math chat with an LLM as one would with a colleague, asking “Is it true that...” followed by a technical mathematical conjecture. The chatbot often then supplies a clearly articulated argument that, in my experience, tends to be correct when it comes to standard topics but subtly wrong at the cutting edge. For example, every model I’ve asked has made the same subtle mistake in assuming that the theory of idempotents behaves the same for weak infinite-dimensional categories as it does for ordinary ones, something that human experts (trust me on this) in my field know to be false.

I’ll never trust an LLM—which at its core is just predicting what text will come next in a string of words, based on what’s in its dataset—to provide a mathematical proof that I can’t verify myself.

The good news is, we do have an automated mechanism for determining whether proofs can be trusted. Relatively recent tools called “proof assistants” are software programs (they don’t use AI) designed to check whether a logical argument proves the stated claim. They are increasingly attracting attention from mathematicians like Tao, Buzzard and myself who want more

assurance that our own proofs are correct. And they offer the potential to help democratize mathematics and even improve AI safety.

Suppose I received a letter, in unfamiliar handwriting, from Erode, a city in Tamil Nadu, India, purporting to contain a mathematical proof. Maybe its ideas are brilliant, or maybe they’re nonsensical. I’d have to spend hours carefully studying every line, making sure the argument flowed step-by-step, before I’d be able to determine whether the conclusions are true or false.

But if the mathematical text were written in an appropriate computer syntax instead of natural language, a proof assistant could check the logic for me. A human mathematician, such as I, would then only need to understand the meaning of the technical terms in the theorem statement. In the case of Srinivasa Ramanujan, a generational mathematical genius who did hail from Erode, an expert did take the time to carefully decipher his letter. In 1913 Ramanujan wrote to the British mathematician G. H. Hardy with his ideas. Luckily, Hardy recognized Ramanujan’s brilliance and invited him to Cambridge to collaborate, launching the career of one of the all-time mathematical “greats.”

What’s interesting is that some of the AI IMO contestants submitted their answers in the language of the Lean computer proof assistant so that the computer program could automatically check for errors in their reasoning. A start-up called Harmonic posted formal proofs generated by their model for five of the six problems, and ByteDance achieved a silver-medal level performance by solving four of the six problems. But the questions had to be written to accommodate the models’ language limitations, and they still needed days to figure it out.

Still, formal proofs are uniquely trustworthy. While so-called “reasoning” models are prompted to break problems down into pieces and explain their “thinking” step by step, the output is as

likely to produce an argument that sounds logical but isn't, as to constitute a genuine proof. By contrast, a proof assistant will not accept a proof unless it is fully precise and fully rigorous, justifying every step in its chain-of-thought. In some circumstances, a hand-waving or approximate solution is good enough, but when mathematical accuracy matters, we should demand that AI-generated proofs are formally verifiable.

Not every application of generative AI is so black and white, where humans with the right expertise can determine whether the results are correct or incorrect. In life, there is a lot of uncertainty and it's easy to make mistakes. As I learned in high school, one of the best things about math is the fact that you can prove definitively that some ideas are wrong. So I'm happy to have an AI try to solve my personal math problems, but only if the results are formally verifiable. And we aren't quite there, yet.

This is an opinion and analysis article, and the views expressed by the author or authors are not necessarily those of Scientific American.

A version of this article entitled “Can AI Outdo Mathematicians?” was adapted for inclusion in the November 2025 issue of Scientific American.

Emily Riehl is a mathematician at Johns Hopkins University, where she works on category theory, homotopy type theory and computer formalization. Her book *Elements of ∞ -Category Theory*, co-authored with Dominic Verity, was published in 2022 by Cambridge University Press.

<https://www.scientificamerican.com/article/mathematicians-question-ai-performance-at-international-math-olympiad>

Mathematics

- **[Math Puzzle: Find the Time](#)**

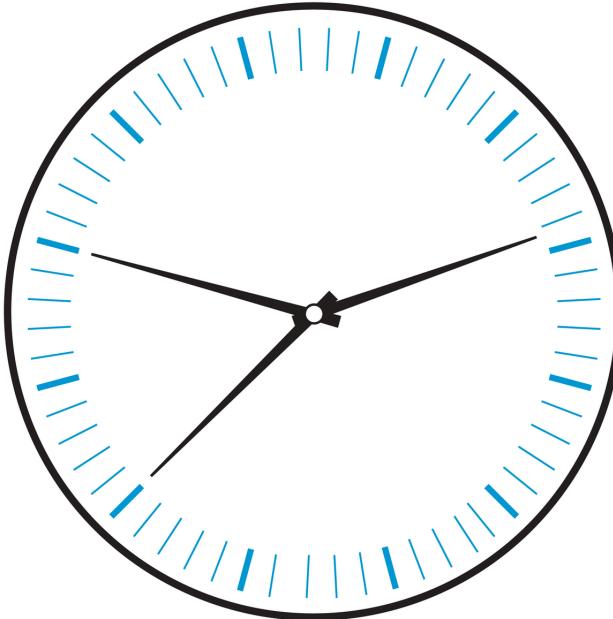
Decode a confusing clock in this math puzzle

- **[What Is the Luhn Algorithm? The Math Behind Credit Card Transactions](#)**

Find out how this simple algorithm from the 1960s catches your typos

Math Puzzle: Find the Time

By [Heinrich Hemme](#)

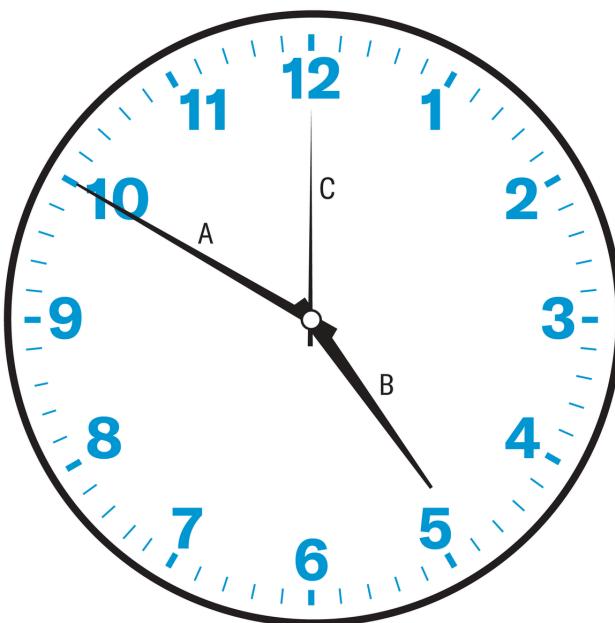


Spektrum der Wissenschaft, restyled by Amanda Montañez

The hour, minute and second hands of this clock are all the same length and move smoothly in a circle. The dial contains hour and minute markers, but the numbers are missing. Therefore, it's impossible to tell which one of the 12 hour markers belongs to the 12. The two hands on the left are positioned exactly on hour markers, and the hand on the right is positioned between a minute and an hour marker. What time does the clock show?

Label the lower hand pointing left A, the higher one pointing left C and the one pointing right B. Hands A and C are pointing exactly at hour marks. If one of these two hands is the hour hand, the minute and second hands should both be on top of each other and point at 12. Because this is not the case, B must be the hour hand. Because the minute hand points to a full minute, the second hand must point to 12. There are two possibilities for this. In the first possibility, A is the second hand, and C is the minute hand. Then A is on 12, and

C is on 2, and it is 10 minutes past the hour. In that scenario, the hour hand must have traveled $\frac{10}{60} = \frac{1}{6}$ of the way from one hour mark to the next. But the picture shows that it has already covered more than $\frac{4}{5}$ of the distance, so this possibility is ruled out. In the second possibility, C is the second hand, and A is the minute hand, and it is 10 minutes to the hour. The hour hand still has $\frac{1}{6}$ of its travel from one hour mark to the next, which corresponds to the image. Consequently, the clock shows exactly 4:50:00.



Spektrum der Wissenschaft, restyled by Amanda Montañez

We'd love to hear from you! E-mail us at games@sciam.com to share your experience.

This puzzle originally appeared in *Spektrum der Wissenschaft* and was reproduced with permission.

Heinrich Hemme is a physicist and a former university lecturer at FH Aachen—University of Applied Sciences in Germany.

<https://www.scientificamerican.com/article/math-puzzle-find-the-time>

The Math Trick Hidden in Your Credit Card Number

Find out how this simple algorithm from the 1960s catches your typos

By [Jack Murtagh](#) edited by [Jeanna Bryner](#)



Isabel Pavia/Getty Images

You're at the checkout screen after an online shopping spree, ready to enter your credit card number. You type it in and instantly see a red error message: "Please enter a valid credit card number."

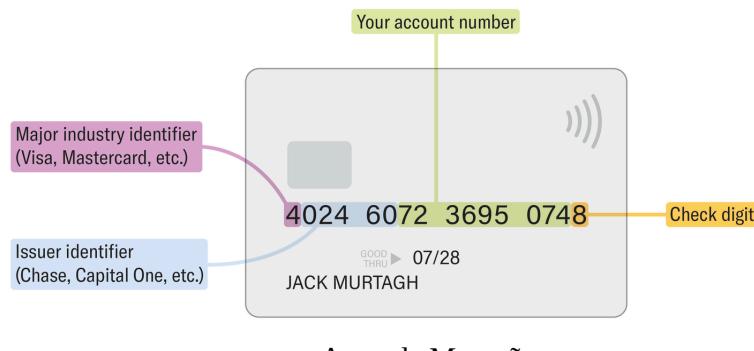
Annoyed, you scan back through each digit and spot the culprit: you put a 6 where a 5 belonged. Typo corrected; purchase complete. But how did the website detect your error so quickly? Does the online platform keep a master list of every valid credit card number to compare your entry against? Did it ping your bank in a split second? The explanation is much cleverer.

All mainstream credit card numbers employ a [mathematical trick](#) designed to catch the most common typos. It's called the Luhn algorithm, named after [IBM researcher](#) Hans Peter Luhn, who

patented it in 1960. Similar error-checking schemes lurk in many of the numbers you encounter daily: barcodes, package tracking numbers, bank account numbers and even ISBNs on books.

READ MORE: [Stretch your math muscles with these puzzles](#)

Grab a credit card from your wallet, and you'll find it contains more structure than first glance suggests. The anatomy of a credit card number includes four main parts. To demonstrate, I'll use my personal Visa.



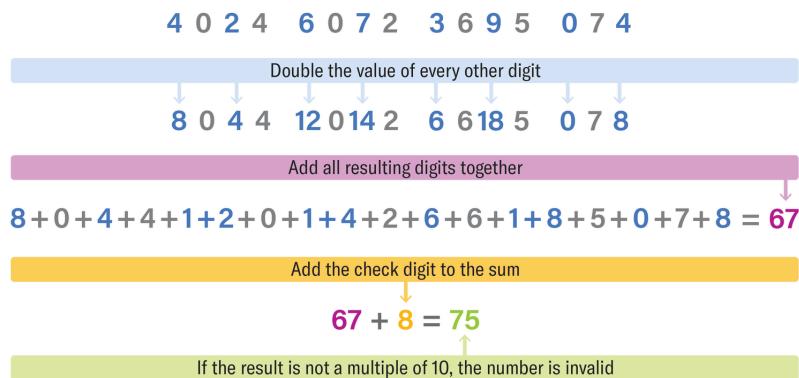
Amanda Montañez

The first digit is the major industry identifier. Visa cards always begin with a 4, and Discover cards always start with a 6. The next five to seven digits pinpoint the bank or institution that issued the card. What remains (sans the final digit) is your specific account number at that bank. The final digit, sometimes called the check digit, has nothing to do with financial institutions. Issuers tack it on so the entire card number will pass a specific mathematical test—the Luhn algorithm. Here's how the algorithm works:

1. Write out all but the last digit of the card number.
2. Starting from the right, double every other number.
3. Sum all the resulting digits (not numbers). For example, if you doubled a 7 to a 14 in step 2, it would become $1 + 4 = 5$ in this step.

- Add the check digit to the sum. If the result is not a multiple of 10, then the credit card number is invalid.

I show the Luhn algorithm in action with my Visa below (you should try it with your credit card, too). The number crunching culminates in 75, which is not a multiple of 10. So this cannot be my real credit card number; I must have mistyped it.



Amanda Montañez

Credit card issuers first assign the account numbers and then compute steps one through three of the Luhn algorithm to determine the appropriate check digit. In this case, the card number should have ended in a 3 because $67 + 3 = 70$, a multiple of 10.

This particular dance of digits has come to dominate credit card verification because of its simplicity and powerful set of features. If you mess up any single digit when entering your card number, the Luhn algorithm will detect it. If you accidentally swap adjacent card digits while inputting, it will detect that, too (with the one exception of flipping 09 to 90 or vice versa).

Dutch mathematician Jacobus Verhoeff [reported in 1969](#) that two errors—mistyping a single digit and swapping two neighboring digits of one's card number—account for nearly 90 percent of all human input errors in practice. Verhoeff developed an even more comprehensive [algorithm](#) that, in addition to detecting all the same typos as Luhn's algorithm, catches 09/90 transpositions, as well as more exotic slipups. Verhoeff's algorithm was a mathematical

triumph. Some contemporaries had even published false proofs claiming no single check digit could carry enough information to catch all these errors. Verhoeff's algorithm never gained widespread adoption, however, perhaps because of its increased complexity compared with Luhn's or because Luhn's algorithm was already deployed and did a good-enough job.

If you mess up any single digit when entering your card number, the Luhn algorithm will detect it.

Luhn's algorithm saves you time and businesses money. At some point during a purchase, a vendor will verify that your card belongs to you by sending your information to a specialized card-validation service. This communication takes time and incurs processing fees for the business. It would be a waste of seconds and cents to outsource needless back-and-forth with a professional validation service to catch common typos. Because Luhn's algorithm requires so little processing power, the computer handling the transaction can also run the check without needing to contact any third party.

It's important to note that passing Luhn's algorithm does not guarantee a credit card number is valid. Rather failing it guarantees the number is invalid. The algorithm puts up a first line of defense that less common typos and savvy fraudsters can slip through. Those cases get caught by the more resource-intensive card-validation services.

How does Luhn's algorithm know when your fingers fumble? Every digit in a credit card number contributes a one-digit number to the final sum in the algorithm. If the digit sits in a position that does not get doubled, then it just contributes itself to the sum. And for digits sitting in doubled positions, summing the individual digits of any resulting two-digit numbers always yields a one-digit number again. The table at the right lists all of these possible contributions.

Contribution in an undoubled position	0	1	2	3	4	5	6	7	8	9
Contribution in a doubled position	0	2	4	6	8	1	3	5	7	9

Amanda Montañez

For example, 6 contributes 6 when it's in an undoubled position, and it contributes 3 from a doubled position because 6 doubled equals 12 and $1 + 2 = 3$. Messing up a single number while typing your credit card effectively shifts your place within the same category of this table and alters one term in the algorithm's sum.* By design, a valid credit card number results in a sum that is a multiple of 10. Any single-digit error will change the sum by a one-digit number, so the mistaken sum will definitely not be a multiple of 10.

Formally proving that Luhn's algorithm detects swapping of adjacent digits involves some case analysis, but an example will help illustrate the idea. Imagine we have the sequence 31 in our credit card, with the 3 in a doubled position. We accidentally enter 13 instead. In the correct sum, this duo contributes $6 + 1 = 7$ (3 doubled plus 1 undoubled), whereas in the faulty sum, it contributes $2 + 3 = 5$ (1 doubled plus 3 undoubled). So the error ultimately changes our sum by 2, going from a contribution of 7 to a contribution of 5. If the original sum yielded a multiple of 10, then there is no way this new one will. We can verify that this works for every pair of numbers except for 09 and 90, which both contribute the same quantity to the Luhn sum.

Next time a checkout page flashes that annoying error message, remember: a simple piece of math under the hood just saved a little time and money for everyone involved.

*Editor's Note (11/11/25): This sentence was edited after posting to correct the description of the online version of the graphic.

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

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<https://www.scientificamerican.com/article/what-is-the-luhn-algorithm-the-math-behind-secure-credit-card-numbers>

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Microbiology

- **Scientists Map Microbiome Hidden Deep inside Tree Trunks**

Trees' inner heartwood harbors methane-producing microbes adapted to oxygen-poor swamps and cow guts

Scientists Map Microbiome Hidden Deep inside Tree Trunks

Trees' inner heartwood harbors methane-producing microbes adapted to oxygen-poor swamps and cow guts

By [Saugat Bolakhe](#) edited by Sarah Lewin Frasier



Darrell Gulin/Getty Images

Scientists have mapped microbe populations in [human guts](#), [deep-sea ecosystems](#) and even [clouds](#). Yet the microbial communities inside tree trunks have remained largely unseen until now. For a recent study [in Nature](#), researchers analyzed about 150 trees to map the communities of microbes living in 16 species. They estimate that a single mature tree hosts about one trillion bacteria in its trunk “microbiome,” with distinct communities living in different layers.

Most intriguing, the scientists found anaerobic bacteria—bacteria that don’t consume oxygen—producing methane in the deep heartwood. “It turned out what’s living inside the trees was really different from what we found anywhere else in the forest,” says the study’s co-lead author, Jonathan Gewirtzman, an ecosystem

ecologist at Yale University. The trees' interior population, he says, was more akin to that of a wetland.

For a long time plant tissues were thought to be sterile. When that was disproved in the early 1900s, researchers focused largely on roots, where many bacteria and fungi are involved in soil-based nutrient cycling. Whatever might be living within a plant's shoots, trunks and leaves was mostly ignored.

To examine the trunk's hidden biome, Gewirtzman and his colleagues drilled into living tree trunks to extract thin core samples, which they immediately froze with dry ice to halt microbial activity. They then separated the cores into sapwood and heartwood (a tree trunk's middle and innermost layers, respectively), ground the frozen wood into powder and sequenced the bacteria in each layer. To study the activity of living microbes, they also sealed holes drilled into trees and later measured gases such as methane and nitrous oxide emitted by different layers.

The researchers learned that when trees are evolutionarily close, they tend to have similar microbiomes. And the team found a surprise deep inside the trunks: "In the older and inner heartwood," Gewirtzman says, "we saw microbes more like what you'd find in a wetland—anaerobic bacteria and methane producers," species suited to a waterlogged and oxygen-poor environment. Some bacteria in the outer layers may consume part of that methane, the researchers found, but the study suggests that methane-producing and nitrous oxide-producing bacteria inside trees could still create greenhouse gas emissions scientists should figure into calculations.

"It is a really nice study, as they did something different from most: comparing the inner wood versus the outer wood," says plant microbiologist Sharon Lafferty Doty of the University of Washington. Doty adds that chemicals used in modern agriculture erode the health of plant microbiomes. "By studying these natural plant-microbe partnerships, we can understand which bacteria are

important and active to add back into our agricultural system,” she says.

Saugat Bolakhe is a freelance science journalist. He studied zoology as an undergraduate in Nepal and received a master’s degree from the Craig Newmark Graduate School of Journalism at the City University of New York. His work has appeared in *Scientific American*, *Nature*, *New Scientist*, *Quanta*, *Eos*, *Discover*, *Knowable* and other publications.

<https://www.scientificamerican.com/article/scientists-map-microbiome-hidden-deep-inside-tree-trunks>

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Microbiome

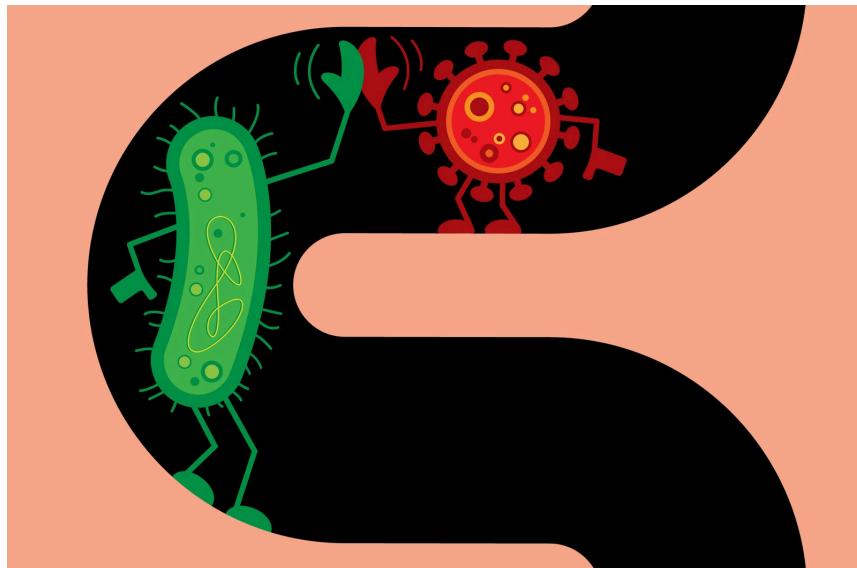
- **Viruses in the Gut Protect Us and Change with Age and Diet**

A new review study examines the “gut virome”: the microbiome’s mysterious viral population

Little-Known ‘Gut Virome’ Protects Us—And Changes throughout Our Lives

A new review study examines the “gut virome”: the microbiome’s mysterious viral population

By [Kate Graham-Shaw](#) edited by [Sarah Lewin Frasier](#)



Thomas Fuchs

Viruses have an understandably bad reputation. But deep in our digestive system, a lot of them are quietly working to keep us healthy. This “[gut virome](#)” is an important part of the overall microbiome—the vast collection of microbes that play a crucial role in our digestion, immunity and overall health.

“The bacterial component of the microbiome is well known,” says Tao Zuo, a microbiologist at Sun Yat-sen University in China. “But the virome we don’t really know much about.”

This is partly because viruses are so tiny; the gut virome makes up just 0.1 percent of the total microbial mass, Zuo explains. And viruses mutate quickly, making their genetic material harder to isolate for study. To get a better understanding, Zuo and his

colleagues pulled together a wealth of research data to catalog how the gut virome changes with age, diet and the environment.

Their review, published [in *Precision Clinical Medicine*](#), particularly focuses on bacteriophages—viruses that infect bacteria and make up more than 90 percent of the virome. These viruses sometimes benefit us by killing harmful gut bacteria. But they can also strengthen pathogens—for example, if a bacteriophage carries a gene that offers resistance to antibiotics,” says virologist Jelle Matthijssens, who specializes in virome research at Belgium’s Catholic University of Leuven (KU Leuven) and was not involved in the review.

The study’s authors show how an individual’s virome is constantly developing based on genetics and the environment. At birth, infants’ bacteriophages often vastly outnumber their microbiome’s bacteria, but this begins to change with exposure to the outside world and as the gut develops. During adolescence, bacterial populations change further because of hormone shifts and accrued exposure to other microbes. By adulthood, healthy people host a delicate and mutually beneficial equilibrium of bacteriophages and bacteria.

Certain bacteriophages that help to maintain this balance are extremely reactive to environmental factors such as diet and air quality, and they also respond to their host’s inflammation levels, immune signaling, stress hormones, and more. Factors such as exposure to certain drugs and poor diet can trigger an imbalance that reduces virome diversity. This shift in turn has been associated with disorders such as inflammatory bowel disease. In elderly people, an aging immune system and increased metabolic stress can further throw this system out of whack and increase viral numbers, potentially contributing to age-related diseases.

Understanding these aging and environmental effects may someday contribute to clinical applications such as targeting viruses to

unwanted bacteria through phage therapy, the scientists say—but much more research is needed.

“A key challenge is distinguishing causality from correlation,” says Evelien Adriaenssens, a microbiologist at the Quadram Institute in England, who was not involved in the new study. “Each individual’s virome is unique, so we cannot make sweeping statements about the health of an individual by looking at their virome alone.”

Kate Graham-Shaw is a journalist based in New York City. She covers international news for Japanese media and also covers health and science topics as a freelancer.

<https://www.scientificamerican.com/article/viruses-in-the-gut-protect-us-and-change-with-age-and-diet>

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Neuroscience

- **You Don't Remember Being a Baby, but Your Brain Was Making Memories**

Brain scans capture memory formation in babies, raising new questions about why people forget their earliest years

You Don't Remember Being a Baby, but Your Brain Was Making Memories

Brain scans capture memory formation in babies, raising new questions about why people forget their earliest years

By [Nick Turk-Browne](#) edited by [Daisy Yuhas](#) & [Madhusree Mukerjee](#)



byakkaya/Getty Images

A plume of red, a searing pain and the sounds of summer—these are fragments of my earliest memory, of when I stepped on a glass shard in a Toronto splash park at six or seven years old. I don't remember much from that day, but a scar on my foot bears witness to what happened.

When you ask adults about their first memory of a specific event from their childhood, their answer is typically about something that happened no earlier than preschool. This is true whether you ask a college student or a grandparent, suggesting that adults' lack of infant or toddler memories is not just the result of normal forgetting that occurs with the passage of time. The lack of autobiographical memory from when you were a baby is known as infantile amnesia.

There are two potential explanations for this phenomenon. One is that infants cannot store memories. The slow development of the hippocampus, a seahorse-shaped region deep in the brain, may be responsible. This region, which is critical for memory, grows and changes throughout childhood, so it might not be available to infants. In this scenario, babies are not so different from people with famous cases of amnesia, such as Henry Molaison and Loni Sue Johnson, both of whom suffered hippocampal damage in adulthood that made them unable to store memories.

Another possibility is that the infant brain can store memories, but we eventually lose access to them. Recent studies in mice show that the hippocampus not only is able to store memories early in life but may retain these memories into adulthood. For example, scientists were able to retrieve an otherwise forgotten memory by **stimulating neurons in the hippocampus** that had been active during an early experience.

But what about humans? My laboratory has been on a decade-long quixotic adventure to study awake infants with functional magnetic resonance imaging (fMRI), a form of imaging that can measure activity from regions deep in the brain such as the hippocampus. Although this technology is used to study memory formation in adults and is safe for infants, it had not previously been used to study babies' memory.

Why was that the case? Infants move a lot, don't follow instructions and have a short attention span—all of which make it difficult to collect good data from them. As a result, most fMRI research in infants has been conducted with sleeping babies. But that wasn't an option for our investigations, because memories are based on experiences in waking life. Through more than 400 sessions and countless insights from families, we have refined innovative techniques to keep awake infants still, happy and engaged.

In a recent study, a team at my lab led by Tristan Yates, now a postdoctoral researcher at Columbia University, used this method to discover that the infant hippocampus [can store memories](#) beginning around one year of age. We showed infants photographs of faces, objects and scenes one at a time during fMRI. Shortly afterward we tested their memory by showing each of these now familiar photographs alongside a new image of the same type. If the infant looked longer at the photograph they had seen before, we labeled that image as remembered; otherwise it was considered forgotten.

With this behavior documented, we looked back at the brain data from when we first showed the photographs and found that the hippocampus was more active when infants viewed images they later seemed to remember. This result suggests that the infant hippocampus can create memories after only a brief experience. The effects were clearest in babies older than 12 months, in infants who had stronger overall memory and in the subregion of the hippocampus that is most important for remembering specific events (called episodic memory) in adults.

Our findings support the idea that people store memories when they are infants and are later unable to access them. But the work also raises more questions: How long do these hippocampal memories last? We tested for a few minutes, but infantile amnesia plays out over years. How sophisticated is this infant memory capacity? We tested individual photographs, but episodic memories involve complex events with multiple people, places and things interacting over space and time (for example, remember your last vacation).

The deepest and most provocative questions relate to why most people's earliest memories are from when they were age four to five (or older) if memories are being stored in their brain by age one. What makes those earlier memories inaccessible? Are there

any tricks or practices for remembering them? Would we even be able to make sense of them if so?

Answering these questions will help resolve more than a century of scientific curiosity. Revealing how the youngest brains learn and remember may help advance understanding of language acquisition and developmental disorders, and it could have implications for parenting and early education. More generally, the mysterious workings of memory early in life may hold clues about why we lose memory later in life in the normal course of aging or with neurological diseases such as Alzheimer's.

Take a moment to reflect: What is your earliest memory? How do you know it's real? There might be even earlier memories locked away in your brain.

Are you a scientist who specializes in neuroscience, cognitive science or psychology? And have you read a recent peer-reviewed paper that you would like to write about for Mind Matters? Please send suggestions to Scientific American's Mind Matters editor Daisy Yuhas at dyuhas@sciam.com.

Nick Turk-Browne is Susan Nolen-Hoeksema Professor of Psychology and director of the Wu Tsai Institute at Yale University. He studies how the human brain learns and remembers.

<https://www.scientificamerican.com/article/you-dont-remember-being-a-baby-but-your-brain-was-making-memories>

Parenting

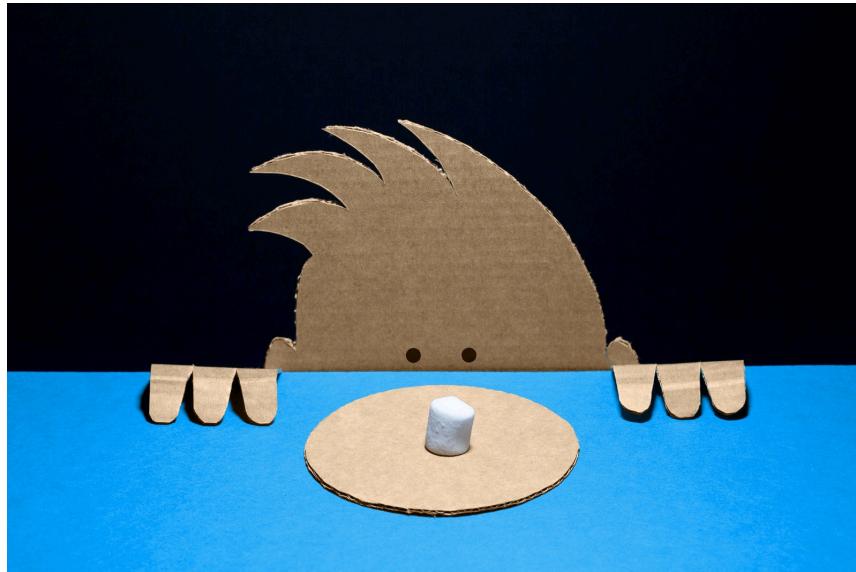
• **The Benefits of Raising Conscientious Kids**

Being conscientious will serve kids in the long run. Here are some tips to help them learn that trait

The Benefits of Raising Conscientious Kids

Being conscientious will serve kids in the long run. Here are some tips to help them learn that trait

By [Jasmine Mote](#) edited by [Megha Satyanarayana](#)



Boris Zhitkov/Alamy Stock Photo

My preschooler is obsessed with rules—and, more important, exploring their loopholes. When I tell him to stop throwing rocks, he will drop a rock dramatically with a loud thud, assuming plausible deniability. Pretending to be a *Tyrannosaurus rex*, he will chase his little sister around our kitchen island and push her. “Don’t push your sister,” I’ll command, and he will reply, “I didn’t push her! The dinosaur did it.”

Self-control is one’s ability to navigate multiple competing desires—such as listening to your mother and shoving your sister. We tend to idolize people who show **certain kinds of self-control** (such as professional athletes) and demonize those who we think don’t show enough (for example, athletes who get caught in doping scandals).

When I think about self-control in children, I think about psychologist Walter Mischel's marshmallow test, in which children could either eat a single marshmallow immediately or show self-control, refuse that first marshmallow and be rewarded with two marshmallows later. The original studies found that children who waited for the additional marshmallows had more academic success in adolescence compared with those who gave in to temptation.

But what if the marshmallow way of thinking about self-control is wrong? Maybe it's about not just avoiding the tempting first marshmallow but the myriad other things that go along with that: planning for the future, following rules, working hard and trusting that you'll indeed get your eventual reward. In other words: being conscientious.

Teaching conscientiousness—a personality trait that's about more than self-control—may actually be the path to helping our children be the best versions of themselves.

In a recent review, researchers found that achieving in-the-moment self-control (for instance, waiting for a second marshmallow one time) does not lead to monthslong or yearslong changes in how consistently we apply such control. Unfortunately, changing our personalities to resist temptation is not so easy. In fact, even people who seem to show more consistent self-control don't necessarily do so all the time. On the contrary, they tend to simply avoid temptation in the first place so they don't have to exercise restraint, and they show less (not more) self-control in their daily lives.

Compared with extroversion, conscientiousness is more strongly related to academic success, work performance and lower rates of substance use. Conscientious people have grit.

It turns out that the results from the classic marshmallow test are more complicated than first thought. Performance on the test and

future academic success are related not just to self-control but to factors such as a child's general cognitive ability and how much education their parents have. Further, it does not seem that one's ability to wait for that second marshmallow is related to success into adulthood.

Conscientiousness is one of the Big Five personality traits that predict academic success (alongside extroversion, agreeableness, neuroticism and openness to experience). Conscientious people often show self-control, but they also follow rules, show up on time and work hard.

Conscientiousness is often underappreciated. In one study, new mothers said they hoped their babies would grow up to be extroverted and agreeable, but they consistently ranked conscientiousness as less preferable than almost all other traits. If extroversion is the life of the party and agreeableness is that one friend who laughs at all our jokes, we may have a tendency to view conscientiousness as a wet blanket, the person who asks to turn the music down or has to leave early to get to bed on time.

Conscientiousness, however, is associated with the same (and arguably more) benefits as self-control: Conscientious people have better health, are less likely to be depressed, are wealthier and live longer than people who are less conscientious. Compared with extroversion, conscientiousness is more strongly related to academic success, work performance and lower rates of substance use. Conscientious people have grit.

Rather than the dud at the party, think instead of your friend who always remembers your birthday, a co-worker who volunteers for the hardest assignment or a judge who upholds the law even when it is unpopular. We could use more conscientiousness in our world.

Conscientiousness appears to be about 40 to 50 percent heritable, so conscientious parents tend to raise conscientious kids. This fact

also suggests that environment and upbringing play substantial roles in determining whether people become conscientious adults.

Authoritative parenting, characterized by warmth, structure and limit-setting, appears to be related to higher rates of conscientiousness in children. Authoritative parenting is also related to secure attachment between parents and children, which is associated with more conscientiousness.

One way we could engage in authoritative parenting and translate some of these ideas into practice might be to explicitly explain to our children why we make the rules we make. Early indicators of conscientiousness may be how readily children follow a parent's instructions and how positively they embrace family rules. That suggests that parents who expect children to do these things may be helping their kids become more conscientious over time. Rather than telling my son he shouldn't shove people "because I said so," I could explain that our family believes it's important not to hurt others and that we don't push others because we could hurt them (even when we're dinosaurs).

We can also look at what conscientious people do in their daily lives outside of self-control behaviors and try to model those other actions for our kids. If we want to model punctuality and responsibility, we could explain why it's important for our family to show up for a playdate on time and then (heroically!) do it. We could also describe to our kids all the things we need to do—pack snacks, put gas in the car, feed the dog—before we can get to our friend's house as a way to demonstrate good planning.

Thinking about the research on how adults who show more consistent self-control over time often exhibit less, not more, self-control moment to moment, we might try to provide our children with opportunities to safely test boundaries and allow their impulses some freedom. Sometimes my family has what we call "yes days" where we try to say yes to whatever our kids desire

(within reason) for an afternoon. Milkshakes for dinner? Sure. Want to chase some birds at a park for hours? Go wild.

Cultivating conscientiousness in our children may not only help them thrive but help us manage our own stress. One study of children in France found that traits such as agreeableness and conscientiousness in the kids were related to [less burnout in their parents](#), including parents who reported less emotional exhaustion and more self-efficacy in their parenting.

There's still a lot we don't know about how conscientiousness develops. Personality traits are hard to change, as are cognitive skills, depending on your child's abilities. For example, if your child has attention deficit hyperactivity disorder or is otherwise neurodiverse, a change in parenting practices alone probably will not be enough to help that child become a better planner or more rule-abiding. It might take longer. [Conscientiousness for them might look different](#) than in other kids. All children, regardless of ability, deserve parents with realistic and flexible expectations around the potential for change as we work toward nurturing conscientiousness in our families.

It's tiring to explain to my son for the hundredth time why we don't shove people. The other day, however, my daughter decided to shove her brother, and I heard him explain to her in a tone not unlike my own, "We don't push people in our family!" As he came running to tattle on his sister, all I could do was laugh.

Jasmine Mote is a licensed clinical psychologist and a research assistant professor of occupational therapy at Boston University. She received her Ph.D. from the University of California, Berkeley. She writes [Mental Healthy](#), a newsletter covering mental health science on pregnancy, parenting and everything in between. Follow her on Bluesky [@jmote.bsky.social](https://jmot.ebsky.social)

<https://www.scientificamerican.com/article/the-benefits-of-raising-conscientious-kids>

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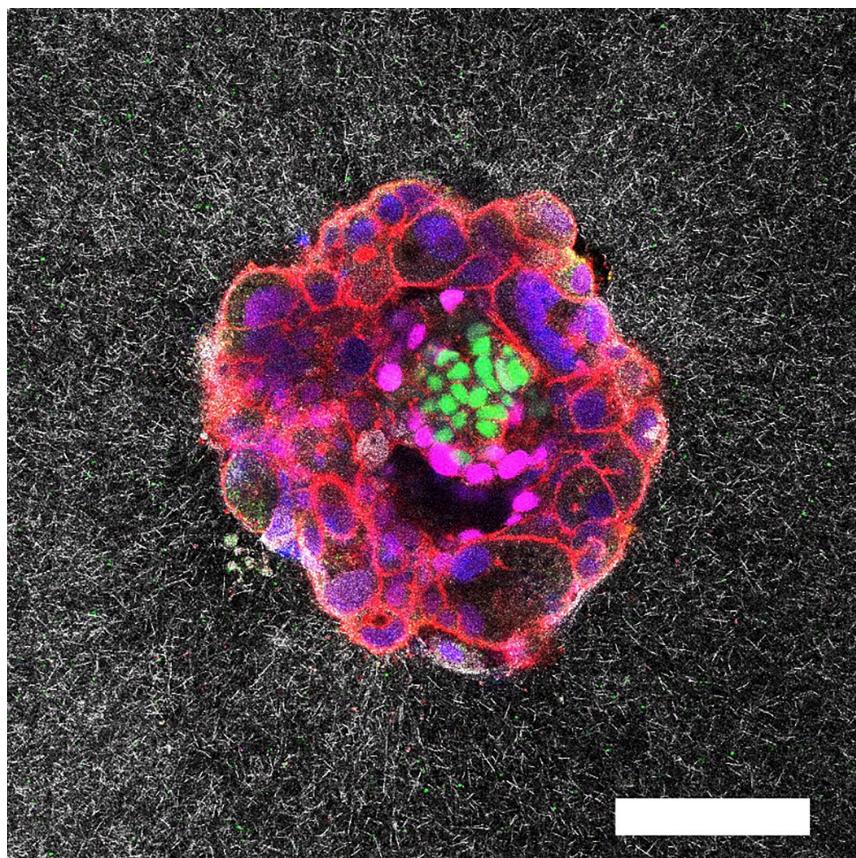
- **Human Embryo Implantation Revealed in First-Ever 3D Images**

Analyzing embryo movements in uteruslike environments could offer clues to improving the success rate of in vitro fertilization

First 3D Images of Human Embryo Implantation Reveal New Details of the Process

Analyzing embryo movements in uteruslike environments could offer clues to improving the success rate of in vitro fertilization

By [Humberto Basilio](#) edited by [Lauren J. Young & Sarah Lewin Frasier](#)



A confocal microscopy image of a nine-day-old human embryo. Specific proteins and cellular structures have been colored in the image: OCT4 (*green*), which is related to embryonic stem cells; GATA6 (*magenta*), which is associated with early tissue formation; DAPI (*blue*), which marks the DNA in the nuclei; and phalloidin (*red*), which reveals the actin cytoskeleton. The scale bar corresponds to 100 microns.

Institute for Bioengineering of Catalonia (IBEC)

Researchers have captured the very first real-time, three-dimensional images and videos of a human embryo implanting into synthetic uterine tissue—revealing a key [stage in reproduction](#). The resulting footage, which shows in vivid detail how embryos push and pull to anchor themselves in the uterus, could lead to

improvements for in vitro fertilization techniques, the scientists say.

“This will allow us to develop treatments specifically targeting implantation, which is the biggest roadblock in human reproduction,” says Samuel Ojosnegros, a bioengineer at the Barcelona Institute of Science and Technology and a co-author of the new study *in Science Advances*.

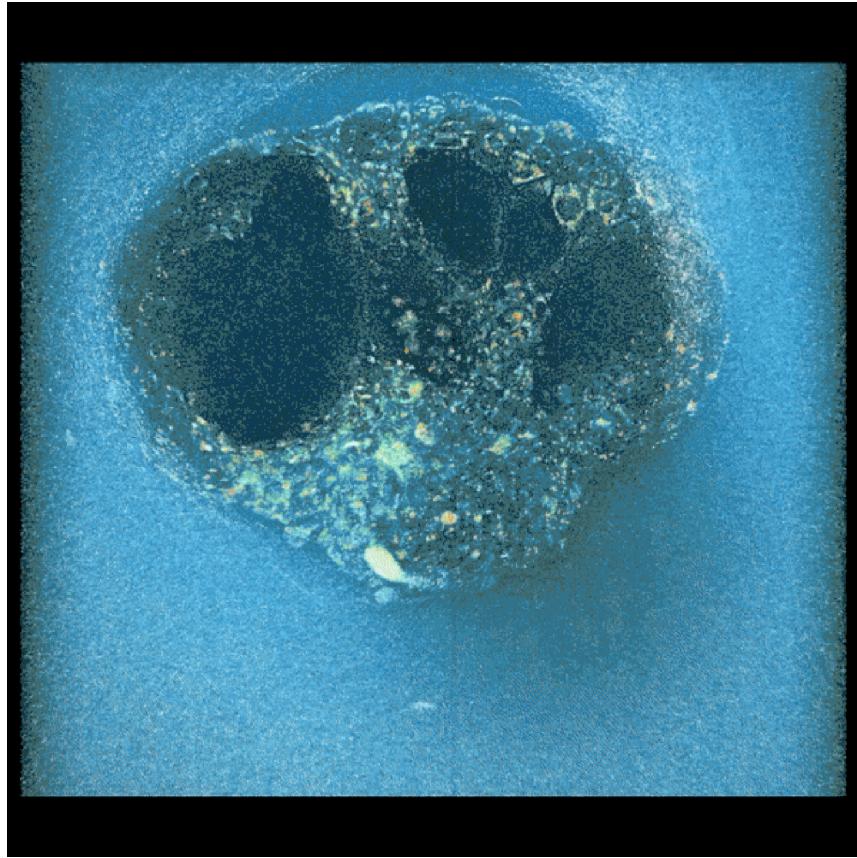
A few days after an embryo is fertilized artificially, fertility doctors must implant it into the body so it can continue to grow. “What happens between the transfer and the first ultrasound weeks later is a black box,” says Ojosnegros, who is also co-founder of biotech company Serabiotics. Implantation failure is one of the main causes of infertility—up to 60 percent of miscarriages occur during this process.

The [first successful cultures of human embryos beyond the moment of implantation](#) were demonstrated in laboratory petri dishes in 2016, but Ojosnegros and his colleagues wanted to see what this process would look like in 3D tissue that was more similar to that of the uterus.

To do this, the team designed a special system made of gel and collagen, a protein found in the uterine lining, and introduced embryos donated by people who had completed an assisted reproduction process. Their system prompted implantation, Ojosnegros says, because the network of collagen fibers signals the expected location and texture to the embryo at a molecular level.

The researchers recorded the action over time using advanced 3D microscopes. Tracking tiny movements in the gel’s fibers let them map exactly where and how strongly the embryos were pulling on the surrounding tissue. They then did the same with mouse embryos to compare their movement patterns.

The footage showed that human embryos generate a network of tiny pulling forces that ripple through the womb. These embryos burrow into the surrounding tissue by creating multiple small traction points that tug the lining in all directions. Mouse embryos, in contrast, spread out more across the surface and pull mainly along two or three strong lines.

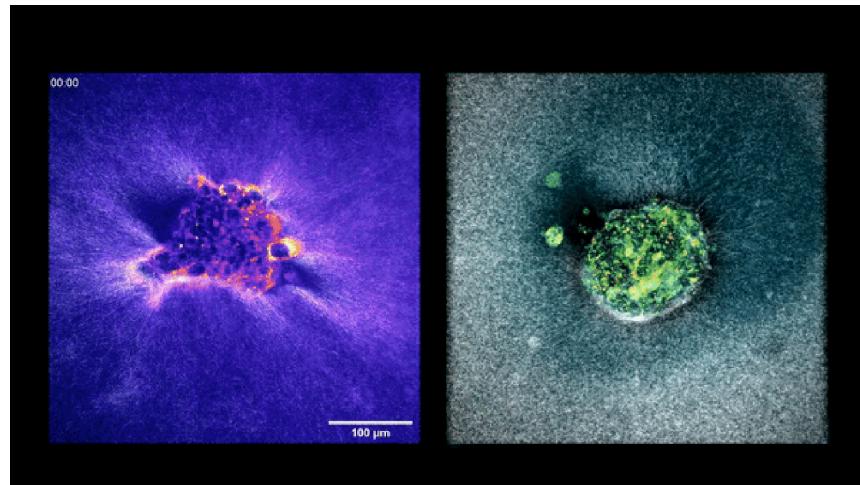


An embryo compacting and invading the uterine tissue.
Sarah Moreira Castro

When the researchers applied external tension to the matrix, tugging it with tiny forceps, they noticed the embryos reoriented toward those areas. The scientists suggest microcontractions in the uterus might direct the embryo to the optimal implantation surface. “We believe these microcontractions are what the embryo uses to guide itself toward the blood vessels and the nutrients it needs,” Ojosnegros explains, adding that more studies are needed to support this hypothesis.

In both mouse and human experiments, the strength and pattern of the pulling forces were linked to the embryos’ potential success,

meaning embryos that pulled less were less likely to invade the tissue. Observing implantation in real time in a 3D model is a “quantum leap” compared with the two-dimensional observations that already exist, says developmental biologist Claudia Spits of the Free University of Brussels, who was also not involved in the new study. Keeping an embryo alive under these conditions is extremely difficult, she says. “What you see in a 10-second video is years of setting these [conditions] up so that the embryo can survive,” Spits adds.



Two embryos implanting in the uterus.
Sarah Moreira Castro

“This study sets the stage to explore the dynamics of implantation in unprecedented detail,” says Magdalena Žernicka-Goetz, a developmental biologist at the California Institute of Technology, who was not involved in the research. The findings add to the growing library of human postimplantation observations published in the past nine years, she says, and “these studies are a thrilling step forward in understanding a stage of human development that has long been hidden from view.” Future research, Žernicka-Goetz notes, is still needed to compare how embryos behave across different “uteruslike” platforms to see whether developmental trajectories differ.

The matrix developed by Ojosnegros’s team is not intended for in vitro fertilization procedures, but it could be a valuable tool for

pharmaceutical company and lab testing. “By beginning to understand how the embryo behaves,” Ojosnegros says, “we can start thinking about the future possibility of selecting healthy embryos or those more capable of implanting.” Spits remains skeptical of that assertion because replicating this technology in other labs could be a major challenge. But she says the results are a “major step forward” in tech that could have future applications once other labs are able to do their own 3D implantations.

Humberto Basilio is a Mexican science journalist covering policy, health, misconduct, archaeology and the environment. His work has been published in the *New York Times*, *National Geographic*, *Science*, *Nature*, and more. He is the current news intern at *Scientific American*.

<https://www.scientificamerican.com/article/human-embryo-implantation-revealed-in-first-ever-3d-images>

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Space Exploration

- **NASA Is Crucial to the U.S. Winning the New Space Race**

The U.S. wants to remain a superpower in space. It can't without supporting NASA

The U.S. Won't Win the New Space Race by Defunding NASA

The U.S. wants to remain a superpower in space. It can't without supporting NASA

By [The Editors](#)



Martin Gee

In the early 1400s, nearly a century before Columbus's fateful voyage to the Americas, China seemed most poised to use maritime might to create a global empire. Beginning in 1405, Ming Dynasty admiral Zheng He commanded a fleet of immense "treasure ships" on a series of expeditions across the Indian Ocean, showcasing China's wealth and strength as far afield as the eastern coast of Africa. But by 1433 the state-sponsored voyages had ceased.

Scholars still debate what led 15th-century China to turn inward, ceding its power—and ultimately the discovery of what would

become the New World—to others. But regardless of its cause, the missed opportunity is unquestionable.

Today a strange echo of this episode is unfolding—on the high frontier of space rather than the high seas. This time, however, China is rising to prominence as the U.S. squanders its advantages. Unlike the Ming court that made no secret of decisively abandoning China’s naval aspirations, some U.S. leaders now embrace space as a vital, contested domain. But while they insist they’re setting a course for America’s continued dominance in space science, technology and exploration, their actions are contradicting and undermining that goal.

Skepticism about, if not outright scorn for, civilian space spending is practically a bipartisan tradition in U.S. politics, but we are talking chiefly about the “Make America Great Again” policymaking of President Donald J. Trump.

While [U.S. leaders] insist they’re setting a course for America’s continued dominance in space science, technology and exploration, their actions are contradicting and undermining that goal.

On July 20, the 56th anniversary of the Apollo 11 moon landing, the White House released a statement in which Trump proudly declared his administration was “reigniting the United States’ leadership in space” and pledged to return Americans to the moon and send them to Mars. Weeks earlier, thanks to Trump’s signature budget-reconciliation bill (the “Big Beautiful Bill”), NASA had received nearly \$10 billion in additional funding for heavy-lift rockets, crewed spacecraft, and other things crucial to the Artemis program, which officially began during Trump’s first term. Acting NASA administrator Sean Duffy has repeatedly parroted similar talking points. During a September press conference, he said “we’re in a second space race right now; the Chinese want to get back to the moon before us. That’s not going to happen. America

has led in space in the past, and we are going to continue to lead in space in the future.” The U.S., Duffy asserted, would achieve this feat in 2027. (Duffy’s remarks came a week after his Trump-appointed predecessor, Jim Bridenstine, more realistically testified to Congress that “unless something changes, it is highly unlikely the United States will beat China’s projected timeline [of 2030] to [send humans to] the moon’s surface.”)

The Trump administration does deserve credit for some sound space policy—such as two executive orders, one in 2020 [seeking to extend the economic sphere](#) of the U.S. and its allies beyond low-Earth orbit and another in 2025 [to supercharge U.S. capabilities](#) by streamlining regulations for domestic commercial space companies. Similarly, this past August, Duffy announced the administration’s plans for NASA to fast-track readying a nuclear reactor for launch to the moon by 2030—a bold move meant to secure valuable lunar territory and eventually power U.S. outposts there.

But these acts must be considered alongside other policies and proposals that influence U.S. scientific and technological prospects off-world and on Earth.

Chief among these is the White House’s proposed spending budget for fiscal year 2026. Despite the boost to the Artemis program, Trump’s FY2026 proposal called for cutting NASA’s overall budget by about 25 percent, with the agency’s science division being slashed by nearly half. Advocacy groups such as the Planetary Society—as well as all seven living former NASA science chiefs—have condemned these proposed cuts as catastrophic for U.S. space science. The cuts, they warned, would lead to the cancellation of more than 40 ongoing and planned U.S. space missions. On Trump’s chopping block are high-profile, decades-in-the-making projects such as NASA’s Mars Sample Return mission and the next-generation Nancy Grace Roman Space

Telescope. These have counterpart competitors in China, which is proceeding unimpeded toward space leadership.

The cuts proposed for FY2026 are not the administration's only harmful moves: White House actions have led to the shedding of more than 2,500 NASA staffers, most of them senior employees. Innumerable federal research grants have been canceled, suspended or delayed because of ideological litmus tests. Thousands of foreign students and skilled professionals have been blocked or discouraged from living and laboring in the U.S. by immigration and guest-worker policies. As the federally funded scientific enterprise staggers and a U.S. brain drain accelerates after these heavy blows, affecting both new and longtime workers, China and other nations are opening their doors to international students and scientists, including American ones, offering generous financial incentives and building state-of-the-art research hubs to attract talent from around the world.

It's hard to see how America's losses across these myriad domains won't lead to other nations' gain, even if we can't predict the marvelous opportunities we'll be missing out on. And, just as with China's befuddling decision to retreat from maritime greatness nearly 600 years ago, it's harder still to understand why U.S. leaders today seem so eager to lose this new space race. Trump's push to make America great again in space presumes America isn't already the world's greatest spacefaring power—which it demonstrably is, albeit perhaps not for much longer.

Our nation's continued greatness in space requires giving more support to government-sponsored R&D rather than less and respecting, not disdaining, science—irrespective of politics.

<https://www.scientificamerican.com/article/nasa-is-crucial-to-the-u-s-winning-the-new-space-race>

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The Environment

- **[South Africa's Coast Is Rising—And Scientists Have a New Explanation Why](#)**

Human water management contributes to sinking land across the globe, and it may also be responsible for an unexpected rise

South Africa's Coast Is Rising—And Scientists Have a New Explanation Why

Human water management contributes to sinking land across the globe, and it may also be responsible for an unexpected rise

By [Avery Schuyler Nunn](#) edited by [Sarah Lewin Frasier](#)



Land rising along South Africa's coast may be closely tied to humans' use of water.
Dhoxax/Getty Images

For decades geologists thought the slow rise of South Africa's southern coast was driven by forces deep below—buoyant plumes of molten rock ascending through Earth's mantle and heaving the crust upward over millions of years. But now satellite data and precise GPS measurements are tilting such assumptions off their axis. A study in the *Journal of Geophysical Research: Solid Earth* suggests this land rise may have less to do with deep tectonic forces and more to do with missing groundwater just under our feet.

Human activity has long been depleting South Africa's groundwater. In 2018, after grappling with severe droughts for years, the country came close to a full-blown water emergency

when Cape Town was nearly the world's first major city to literally run out of water—a scenario dubbed “Day Zero.” For several months that year the city's residents faced the very real prospect of having to regularly queue for critically limited water supplies, an outcome staved off only by timely rainfall and intensive water-saving campaigns. The extreme shortage resulted from a combination of climate change and unsustainable water use, which drained surface reservoirs and placed mounting pressure on aquifers across the region.

The recent study hypothesizes that the ground, once compressed by the sheer weight of the surface water and groundwater above it, is now expanding like a foam mattress relieved of pressure. Using GPS and satellite gravity data from between 2000 and 2021, the researchers detected a roughly six-millimeter rise in the land surface—a shift that coincides with humans' depletion of South Africa's water reserves and periods of drought.

“Sometimes the first explanation isn't necessarily the right one,” says University of Bonn geodesist Christian Mielke, the study's lead author. “Perhaps it isn't plate tectonics after all.”

That misunderstanding, not necessarily the rising land itself, may be the most striking thing about South Africa's situation. What was once chalked up to the slow churning of Earth's mysterious and inaccessible interior may instead reflect human activity, especially our management—or mismanagement—of water.

“The presence of water, either as ice and snow on the land surface or as groundwater below, and the removal of that water are intimately tied to the deformation of the ground's surface,” says Stanford University geophysicist Rosemary Knight. In most places around the globe, this process usually leads to sinking, called land subsidence, to fill the gap.

But in South Africa, the new study suggests, that tie between water and land movement shows up in a surprising way. During the rainy season, rivers and reservoirs fill, adding weight that presses the crust down. In the dry months, much of that water either evaporates or gets pumped away, and the land rebounds upward. Over time the long-term loss of groundwater tips the balance toward uplift rather than sinking.

This “seasonal breathing” is the giveaway that the cause is probably not solely a mantle plume. If molten rock were pushing upward, the motion would be steady, not tied to rainfall cycles. The expansion, if verified, could be yet another example of the ways human water use is reshaping the planet.

From 1945 to 1970 more than 13,000 square kilometers of California’s San Joaquin Valley, once hailed as a “land of milk and honey” for Dust Bowl migrants, sank by at least 30 centimeters—and in some places by nearly nine meters. The San Joaquin sinking has only sped up since then, and [parts of the valley drop more than 30 centimeters a year during severe droughts](#). On average, the pace has accelerated by 70 percent from the mid-20th century.

Something similar is happening to the Chesapeake Bay, which, with its sweeping estuaries and lush tidal wetlands, is one of the U.S. East Coast’s most ecologically significant regions. Here land subsidence—driven by both groundwater extraction from aquifers and the lingering effects of ancient glacial shifts—is accelerating flood risk and relative sea-level rise. Satellite data, tide gauge records and projections from the Intergovernmental Panel on Climate Change suggest that by 2100 the combination of subsidence and sea-level rise could inundate up to 1,100 square kilometers of the Chesapeake Bay’s coastline.

Mielke notes that such findings highlight the complexity of the planet’s response to human-induced environmental change. The consequences are still gradually being uncovered, and the

implications may be profound. As climate change accelerates, land movements could exacerbate other challenges, especially in coastal areas with rising seas.

To monitor such hidden shifts on a global scale, scientists use the GRACE satellite mission (Gravity Recovery and Climate Experiment) to detect changes in Earth's mass by measuring minuscule variations in gravity. Because water has weight, depleting or replenishing groundwater subtly alters the planet's gravitational field, which [GRACE can detect from orbit](#).

Knight and other researchers are looking for ways to keep land from shifting on such a vast scale by maintaining a careful balance. "Basically you get subsidence when water out exceeds water in," Knight says. "And for water in, the term that's used is 'recharge.'"

Some recharge happens naturally as rain or snowmelt soaks into the soil, but this precipitation isn't enough to offset decades of groundwater extraction and current demand. That's why places such as California are now turning to managed aquifer recharge: strategically spreading excess surface water (such as winter floodwaters) across land where it can percolate into the ground and rebuild depleted reserves, or injecting water directly into aquifers. Estimates suggest there is space underground for a total amount of water [30 times the volume of California's Shasta Lake](#), enough to begin reversing the land's descent.

As Knight puts it, the solution can't be about just cutting back on groundwater pumping. It must involve replenishment: restoring water to the ground from which it was drawn.

Avery Schuyler Nunn is an avid surfer, free diver and environmental science journalist based in California. She earned her Master of Science degree from Columbia University in 2021 and has used her notebook and camera as tools for exploration, both above and [beneath the surface](#), ever since. She is a freelance contributor to *Scientific American*, *National Geographic*, *Smithsonian Magazine*, Grist, and more. Follow her work on [Instagram](#) and [X \(formerly Twitter\)](#) @earthyave and at www.averyschuyler-nunn.com

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