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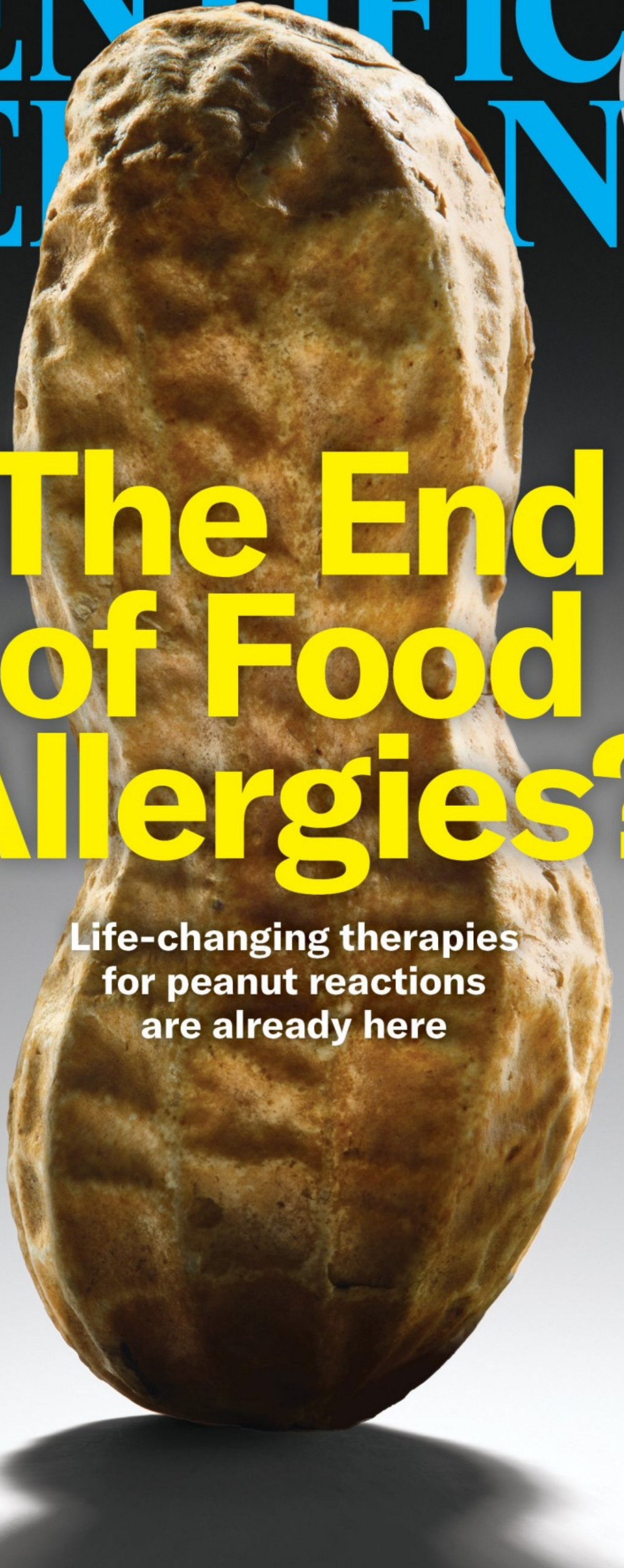

The End of Food Allergies?

Life-changing therapies for peanut reactions are already here

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Can Peanut Allergies Be Cured?

Remarkable new treatments can free millions of kids and adults from the deadly threat of peanut allergy, tackling one of our fastest-growing medical problems

By Maryn McKenna edited by Josh Fischman

Andrew B. Myers

Anabelle Terry, a slender, self-possessed 13-year-old, has heard the peanut butter story her entire life. At two and a half she ate nuts for the first time. Her mother, Victoria, had made a little treat: popcorn drizzled with melted caramel, chocolate and peanut butter. Anabelle gobbled it down. “And afterward, I felt really sick,” she says. A few minutes later she vomited on the kitchen floor.

There was more trouble ahead. A visit to an allergist confirmed that Anabelle was severely allergic to the peanut butter in the dessert, as well as to most other nuts. It began a life upheaval familiar to families of kids with allergies: learning to decode labels, to carry an EpiPen, and to interrogate friends and their parents about the ingredients in a birthday cake.

Every once in a while, there would be a slip-up. It might be a snack that someone hadn't scrutinized or a food package that didn't list all potential allergens. And every time, Anabelle's reactions got worse. Although she was just a schoolkid, she had to stay alert. "Eating lunch, all my friends would have PB&Js. And I'd be like, I'm going to sit a little bit farther away," she recalls. "And going over to friends' houses after school, we always had to make sure: 'Hey, would you mind making a nut-free meal?'"

Most of that caution is in Anabelle's past now. For the vast majority of patients, peanut allergy is an unpredictable, lifelong affliction. But thanks to a clinical trial that Anabelle entered when she was nine, she can now tolerate peanuts and tree nuts well enough to feel safe every day. The drug she received in that trial was approved for treating food allergies by the U.S. Food and Drug Administration last year, making it the second food allergy remedy to earn the agency's blessing since 2020. And an array of other clinical trials are tackling peanut allergy in a variety of ways, from new modalities for desensitizing patients to bold new applications of existing drugs. Some have reported striking successes. "It's an amazing time right now," says R. Sharon Chinthrajah, an associate professor at Stanford Medicine, who led the national trial Anabelle joined.

In fact, medicine's entire understanding of how to keep children safe from ever developing allergies is being rethought. With peanut reactions, for instance, there are real hopes that children can be protected—definitely from the worst effects and maybe from any at all. "The future looks very bright for our patients to have more choices in different periods in their lifetime," Chinthrajah says. "We're not yet at the cure, but we're definitely moving along on the therapeutic front to be able to deal with this chronic disease."

Peanut allergies are perplexing, in part because they appeared so recently. Food reactions have occurred throughout recorded history, but widespread peanut problems didn't begin to surface until the

1990s. The effects on everyday life were dramatic: airlines began to deprive passengers of peanuts and announce that certain snacks might threaten someone else onboard. Elementary schools set aside peanut-free tables at lunch, and food manufacturers began to label their baked goods “school-safe” to signal they were free of common allergens. Epinephrine auto-injectors, which can shut down severe allergic shock (and are usually called EpiPens, for the dominant trademarked version), were rare and carried mostly for the occasional beesting. Now they are a ubiquitous, nearly $3-billion product.

Scott Sicherer, a clinician and director of the Jaffe Food Allergy Institute at the Icahn School of Medicine at Mount Sinai in New York City, watched reports of peanut threats rise in real time. In 1997 he and his colleagues conducted the first survey of peanut and tree-nut allergy in the U.S., finding that 1.6 percent of adults and 0.6 percent of children described themselves as allergic based on reactions they had experienced. The group repeated the survey with a similar-size representative sample five years later and learned that the rate of nut allergies reported in children had doubled to 1.2 percent. In a third sampling, conducted 11 years after the first one, the overall rate tripled from that initial measurement to 2.1 percent of children, and peanut allergies were reported in 1.4 percent of kids.

Since then, the prevalence has risen even more. A large national survey of parents conducted between 2015 and 2016 by researchers in Illinois and California found that food allergies affect 7.6 percent of U.S. children, and peanut allergy affects 2.2 percent. An analysis of health-care payment data in 2018 asked how many new diagnoses of peanut allergy there are among children born each year—what statisticians call incidence, as opposed to prevalence—and reported a rate of 5 percent. And what’s more common is now also more dire: researchers at the Mayo Clinic have estimated that emergency-department visits for anaphylactic shock caused by foodborne allergies—the kind of reaction that can squeeze shut airways and trigger heart attacks—increased more than threefold between 2005 and 2014. The highest rate was for peanut allergies.

"One out of 10 individuals in the U.S., more than 33 million, has a food allergy," says Sung Poblete, CEO of Food Allergy Research and Education, an advocacy organization. "One out of 13 kids has food allergies. That's two kids out of every classroom."

> Medicine's entire understanding of how to keep children safe from allergies is being rethought.

This increase—which is happening around the world, though not at the same rate in every nation—is a mystery. Food allergy is fundamentally a disease of inflammation. The immune system recognizes certain proteins in a food as unwelcome and launches a cascading reaction that often involves an antibody called IgE. The antibody triggers a whole-body inflammatory response: hives, swelling, vomiting, and, in the worst cases, crashing blood pressure and an inability to breathe. "Inflammatory diseases of many kinds are more common than they used to be," says Brian Vickery, a professor of pediatrics at the Emory University School of Medicine and director of the Food Allergy Program at Children's Healthcare of Atlanta, who is a principal investigator on multiple clinical trials. "Eczema, type 2 diabetes, atherosclerotic cardiovascular disease, cancer, depression—all these things have inflammatory origins and are more common now."

The reasons seem to be varied. Researchers have proposed that cleaner modern life, early antibiotic exposure, and microbiome damage from detergents and surfactants—all components of what's called the hygiene hypothesis—might influence how often allergies develop. Genetics may predispose people to react to certain foods. There may be a clue as well in which foods provoke reactions. Up to 90 percent of food allergies are caused by just eight things: peanuts, milk, eggs, fish, crustaceans, tree nuts, wheat and soybeans. (These are the foods that, according to a 2004 U.S. law, have to be declared on labels; a separate 2021 law added sesame to the list.) Why these foods are especially allergenic also puzzles researchers. They contain complex proteins, which remain intact during digestion and may trigger the immune system in ways other foods do not; these proteins also may have similarities to common environmental allergens.

Regardless of the underlying causes, research is zeroing in on ways to mitigate food allergies. Peanut allergy is the priority because the disruptions it imposes have become so visible in society. But the hope is that some of the new approaches can be applied to other allergies—and to help children such as Anabelle who experience more than one.

The first priority in tackling peanut allergy has been children who are at extraordinary risk, the ones whose lives are at stake if they consume something with the smallest cross-contamination from a manufacturing error.

Andrew B. Myers

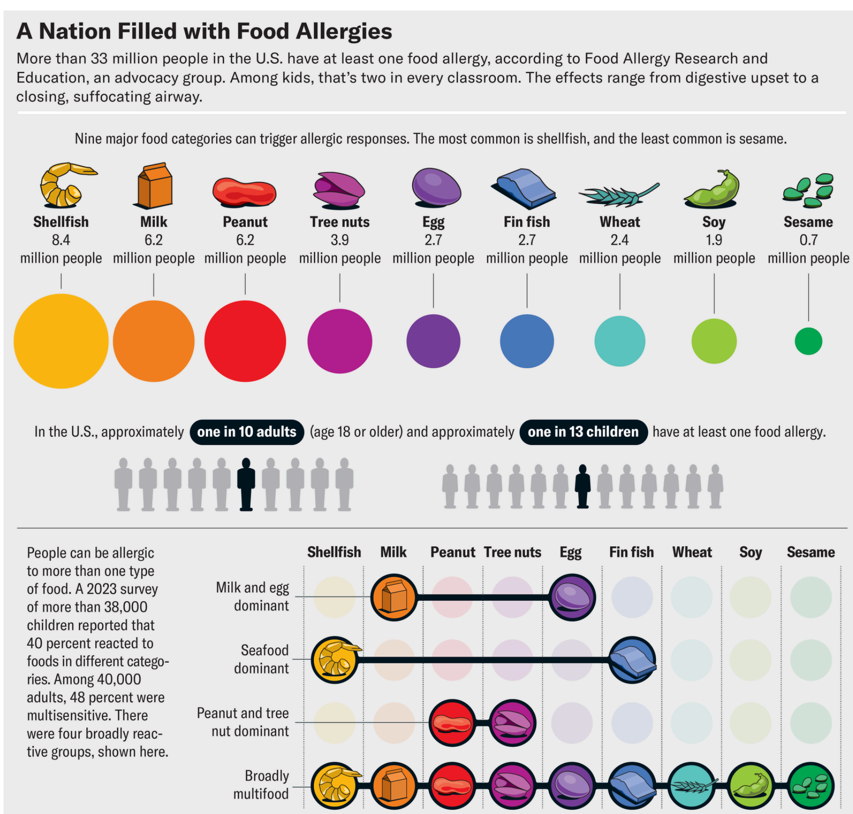
People who suffer from seasonal allergies often receive allergy shots, a program of injections that gradually decreases their sensitivity and keeps their reactions at a level they can tolerate. Allergy shots were briefly tried for peanuts as well, but they were abandoned because of safety concerns, including the 1991 death of a trial participant who received a miscalculated dose. After that, patients’ only remaining option was to change their diet, but mistakes and cross-contamination kept putting them at risk. It took more than a decade for immunologists to try a different method of desensitization for peanut allergies that had a century-old history: giving minuscule, escalating doses by mouth, a process called oral immunotherapy. A large international study in 2018 definitively proved that the approach worked, and it became the standard for treating kids whose families weren't willing to trust avoidance. In 2020 it led to the first-ever FDA approval of a therapy for peanut allergy, a powdered form of peanut protein with the trade name Palforzia that is dispensed over months in precisely metered doses.

That was a huge advance—but, for some families, still not a solution. Initially Palforzia was not approved for children younger than four years of age. Dosing needed to be extremely precise and, according to some practitioners, was tricky to manage. Plus, the drug debuted at the start of the COVID pandemic, when repeat office visits for dose changes became especially challenging. And as the drug's own labeling acknowledges, taking it poses the possibility of reactions. That has left some allergy families searching for alternatives to oral immunotherapy. "Ten to 20 percent of patients can't finish the treatment because of the side effects," says Edwin H. Kim, an associate professor at the University of North Carolina at Chapel Hill School of Medicine and director of the UNC Food Allergy Initiative. "And up to 10 percent of patients experience anaphylaxis at some point while they're on the treatment itself."

Kim is participating in research into two other methods for presenting smaller doses of peanut allergens to the immune system safely: sublingually (under the tongue) and on the skin. The skin method involves a patch containing peanut protein that is applied daily at home for as many as three years; the patch always contains the same dose but is gradually applied for escalating amounts of time. In a phase 3 clinical trial, the results of which were published in 2023, 67 percent of toddlers who were too young to receive Palforzia and who wore the patch were able to raise the amount of peanut protein they could safely consume to the equivalent of three or four peanuts. That was twice as many children as in the placebo group.

The hope for the patch, which has not yet been approved by the FDA, is that it will be easier for kids to tolerate because of its lower dose and easier for parents to manage logistically. Lora Milburn's son, Vance, wore it for a year as a trial participant. He was eight months old when he started showing allergy symptoms—too young to have words for what he was experiencing—and four years old when he entered the trial through Kim's clinic. He was expected to finish the trial in August of this year, and his mother already thinks his sensitivity is diminished. She does not know whether he received the real treatment or a placebo, but she has noticed the way he reacts to the patch. "Some days he doesn't really complain about it; some days he's scratching his back against the wall trying to get the itchies out," she says. "But he knows why we're doing it. If it's nighttime, he's like, 'Mommy, take my patch off, put my new patch on.'"

All these exposure therapies—the patch, the oral doses, the version that goes under the tongue—target reactions to specific peanut allergens. But a separate cadre of researchers has envisioned the struggle to control peanut responses as an entryway to remodeling the way that the immune system reacts to food more broadly.



Jen Christiansen: Sources: “Food Allergy Facts and Statistics for the U.S.,” published by FARE (Food Allergy Research & Education), July 2024, foodallergy.org (*prevalence data*); “The Epidemiology of Multifood Allergy in the United States: A Population-Based Study,” by Christopher M. Warren et al., in *Annals of Allergy, Asthma & Immunology,* Vol. 130; May 2023 (*multiallergy reference*)

In 2013 they began testing the efficacy of an existing drug, a monoclonal antibody named omalizumab (marketed as Xolair) that is already approved for severe asthma caused by allergies. “It’s an anti-IgE biologic, and IgE antibodies are at the center of the whole allergic inflammatory cascade,” Chinthrajah explains. “And the beauty of something like that, where you’re targeting allergic inflammation, is that it has the potential to help all allergies.”

Investigators launched a trial that admitted children and adults who showed allergies to peanuts and at least two other foods; Anabelle Terry was one of the participants. Using a complex study design with several stages, the scientists tested whether regular doses of the injectable drug worked better to reduce allergic sensitivity than did placebos; whether shorter or longer courses of the drug made a difference; whether it worked best alone or combined with oral immunotherapy; and how often and in what amounts people could consume allergenic food once they stopped the treatment.

In 2024 the researchers (a very large team working in multiple medical centers) published the first results. In children aged one to 17 years, 67 percent of those who received the drug were able to eat the equivalent of four peanuts, enough to keep them safe from any accidental exposure. Based on those results, and anticipating more data, the FDA immediately approved Xolair as a protection against peanut allergy.

Participating in the trial was a significant commitment for families. Jennifer Jennison’s son, Jack, was two years old and allergic to eggs, peanuts and cashews—among other foods—when the trial accepted him at its Atlanta site. Every two weeks she or her husband, David, would take time off work to bring their son for an injection. After around seven months, the protocol added tests of small doses of food allergens in applesauce to the office visits; after several hours of observation to make sure the dose was safe, the family carried home boxes of premeasured allergen powder for Jack to eat every day. And in a third phase, Jack progressed to a daily maintenance regimen with actual food: powdered egg white, a cashew and seven Reese's Pieces.

Jack's experience is similar to Anabelle's. She was in the same arm of the trial and now eats a daily dose of peanuts, walnuts and cashews to keep her protection up. But what happened to the Jennisons afterward shows that no peanut-allergy protection is perfect yet. Convincing a child to eat the same foods every day is no small task. First Jack refused his maintenance dose of cashew. After a while he started to resist the Reese's Pieces, too.

The Jennisons live in Atlanta, the corporate home of Chick-fil-A, and seemingly every kid's birthday party features the restaurant's nuggets as well as a cake—which both contain eggs. "For us, eggs are the most important," Jennifer says. "I still feel more comfortable with the cross-contamination risk of peanut knowing that he had built up a tolerance. But for now we're back to avoidance."

Because new approaches to desensitization have worked so well for severely affected kids, researchers have begun to address the needs of those who are somewhat less allergic. For instance, some kids can eat half a peanut before suffering a reaction. That's a tiny amount from the perspective of a nonallergic person, but it's a huge, life-threatening dose to a highly allergic one. Such people, whom some immunologists call "high threshold," include possibly 800,000 kids with peanut allergies just in the U.S. But their triggers are so different from those of highly allergic people that they had been excluded from some trials of desensitization strategies. Indeed, immunology didn't have a clear understanding of whether desensitization that started from their baseline would even achieve the same results as in highly allergic kids.

All of that is now changing because after years of diagnosing patients in this class, medical practitioners could perceive that the group was being left behind. “We would have children who maybe would eat half of a serving before they would start to have symptoms,” Sicherer says. “And what we would tell those individuals is: ‘Your symptoms weren’t so bad, so you’re not really that much in danger. You still need to avoid it, but if there were a small accident, maybe you would be okay.’”

Jackson Esteves was 10 months old when his parents discovered his allergies. His mother, Holly, who was so thoughtful about her children’s diets that she made her own baby food, was starting to introduce him to solids. She made a spinach pancake for her older daughter, slid a few morsels onto Jackson’s high-chair tray, and then watched in horror as raised red hives rippled down his body. The pancake contained eggs, and tests showed that Jackson was allergic to them—and to dairy, sesame, tree nuts and peanuts.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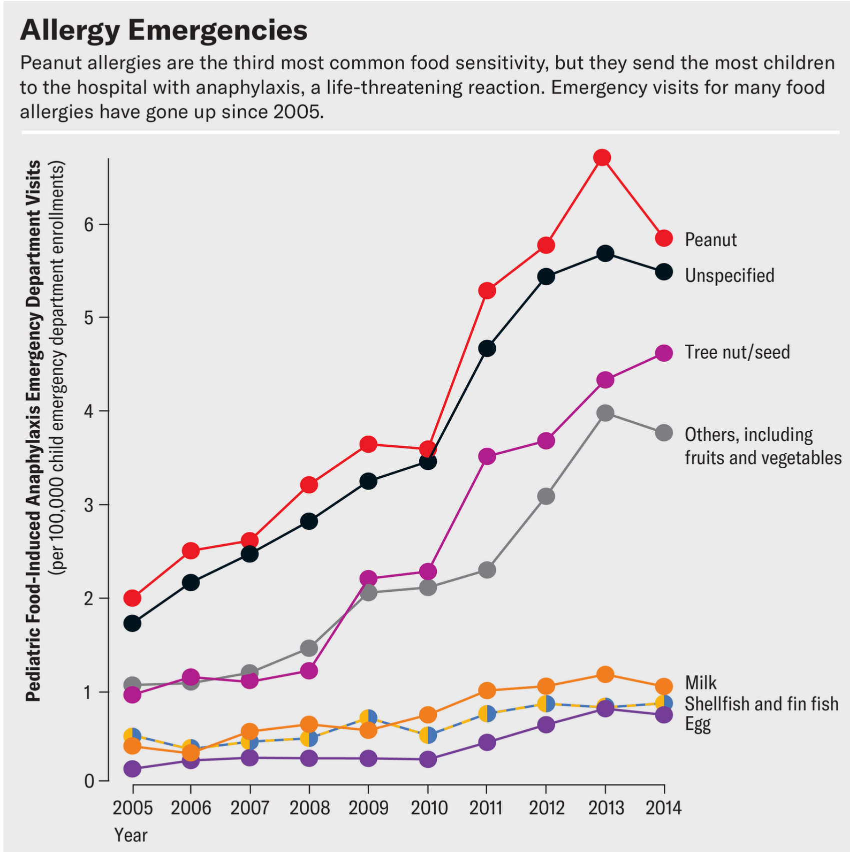
The diagnosis sent the Esteveses, who live on Long Island in New York State, hurtling into a landscape familiar to other allergy families. “I was suddenly attuned to every food label,” Holly says now, 10 years later. “I was learning how to modify recipes. I became very insecure in social settings, family parties, birthday parties. I had to bring everything for him.” What made it even more complex was that no one else in the family—Jackson’s parents, his older sister, or a younger sister who was born soon after the pancake incident—shared Jackson’s allergies.

The Esteves family didn’t know it at the time, but Jackson’s allergies concealed a kernel of promise. Although he was extremely reactive to some foods, medicine considered him just minimally allergic to peanuts—and that made him eligible for a new trial launched by Sicherer and a team of researchers from several institutions, called CAFETERIA. (Allergy researchers seem to be exceptionally fond of complex acronyms. “CAFETERIA” comes from “Challenging to Foods with Escalating Thresholds for Reducing Food Allergy.” The Xolair study was known as OUtMATCH, which stood for “Omalizumab as Monotherapy and as Adjunct Therapy to Multi-Allergen Oral Immunotherapy in Food Allergic Children and Adults.”)

Starting in 2019, children between four and 14 years old consumed escalating doses of peanut butter, first under medical supervision and then at home, first with a carefully measured eighth of a teaspoon and increasing every eight weeks until they were consuming one tablespoon daily. Then they were asked to eat two tablespoons of peanut butter—the amount that would go in a sandwich, which an allergic child would never try to consume—every week for 16 weeks but not necessarily in daily doses. Finally, they had to refrain for eight weeks before being tested a final time.

It worked. Among the 32 kids in the peanut-eating arm of the study (as opposed to a control group that avoided peanuts), every child achieved the study’s final goal of consuming the equivalent of about three tablespoons of peanut butter without a reaction.

That result was “amazing,” says Patricia Fulkerson, chief of the food-allergy section of the National Institute of Allergy and Infectious Diseases, which funded the escalation study. “A 100 percent response rate is hard not to be happy with.”



Jen Christiansen; Source: "National Trends in Emergency Department Visits and Hospitalizations for Food-Induced Anaphylaxis in US Children," by Megan S. Motosue et al., in *Pediatric Allergy and Immunology*, Vol. 29, August 2018 (*emergency department data*)

The study's authors say it needs to be repeated in more kids and at different medical centers. Jackson, who turned 11 this year, has been able to eat peanuts safely since he completed his participation in CAFETERIA; he'll even eat a PB&J once in a while, although it is not a favorite food. Most of his allergies to other foods remain unaffected, but "he was a success story" all the same, his mother says. "He's over peanut allergy."

Even though the CAFETERIA study worked at its small scale and the different approach in the OUtMATCH trial resulted in an FDA drug approval, those tests and others share a limiting feature: they are hard for both the children going through them and the parents guiding them. The kids have to push themselves to swallow something that has made them ill in the past and that still, even in tiny doses, might produce an unpleasant reaction. Parents have to endure the stress of watching their children undergo food challenges to test their progress, knowing that life-threatening anaphylaxis might result.

Plus, for highly allergic people, the most that desensitization can offer is to keep them safer, not completely safe. “Ultimately we’re not curing the allergy,” Vickery says. “We’re kind of providing a protective shell around the patient, a way to defend them against an accidental exposure. They’re still reading labels, they’re still avoiding the food, they are still carrying their epinephrine.” What families long for is something that could make peanut allergy just go away.

A newly proposed treatment might manage that by rewriting the immune system’s memory of antigens—although research into it is in such early stages that results have been reported for only one patient, and the first small clinical trial is just beginning.

The treatment involves successive administration of two drugs, both made by biotechnology company Regeneron. The first drug, dupilumab (marketed as Dupixent), is a monoclonal antibody that is already FDA-approved for treating moderate to severe eczema and asthma and a few other conditions; it works by blocking the action of specific cytokines, signaling proteins that encourage the production of IgE. The second, linvoseltamab, is also an antibody and recently received FDA approval for treating relapsed or refractory multiple myeloma. This cancer affects plasma cells, a category of white blood cell that produces antibodies, including IgE.

Investigators initially thought that dupilumab could be a solo treatment for peanut allergy. But several trials showed that although peanut-specific IgE levels went down under its influence, there was no long-term practical benefit. Even immediately after a course of the drug in one trial, participants were unable to tolerate peanuts. In another test, drug recipients showed improved peanut tolerance right away, but it dropped three months later. IgE availability bounced back.

That led researchers to look at the second drug, which deals a mortal blow to the cells where IgE is manufactured. In mice and monkeys, administering a dose of linvoseltamab during an ongoing course of dupilumab destroyed the cells producing IgE. Continuing the dupilumab while the plasma cells grew back suppressed allergic inflammation and kept the animals' immune systems from restarting the overreactions.

"This is very different than other approaches of trying to build tolerance in patients or trying to just decrease IgE," says Jennifer Maloney, who leads Regeneron's therapeutic work on immune, inflammatory and infectious diseases. "This is something that potentially could remove that allergic antibody from the person."

The company has shared results from just one patient so far, a 20-year-old man with multiple severe allergies. Regeneron described his treatment at the J. P. Morgan Healthcare Conference in January 2025, documenting a dramatic drop in his IgE production during the dupilumab course and after the linvoseltamab was given. His case has not yet been published in a peer-reviewed journal, but the company is now recruiting a small group of patients for an early-phase trial that will primarily test safety. Vickery plans to enroll one patient at Emory, where linvoseltamab is already being used to treat cancer patients.

"We're going to learn something really important," he says. "If we wanted to cure the disease and make it go away, would this be a viable approach to doing so? If it doesn't work, we're going to learn things about why it didn't work and what we might need to do in the next trial."

There is another goal of peanut-allergy science. It's the ultimate goal: prevention, not desensitization or cure. And that may be possible for children being born now, thanks to a British study that has been running for more than a decade—and to a snack.

In the early 2000s Gideon Lack, an immunologist then at Imperial College London, went to Tel Aviv to give a talk on how food allergies were rising around the world. He asked the audience, all Israeli pediatricians and allergists, how many of them had treated children with peanut allergy. From his own experience in the U.K., he expected every hand to shoot up. Only a few did.

This low show of hands was extraordinary, and it immediately presented an opportunity to ask why the U.K. and Israel were so different. After returning home, Lack set up a survey to compare national rates of peanut allergy. To rule out some undetected genetic difference in the Israeli kids, he chose to limit the survey to Jewish children, recruiting roughly 5,000 in each country. The results revealed that the occurrence of peanut allergy in Israeli kids was one-tenth the rate among U.K. ones. A second set of questions posed to a subset of the children, 77 in the U.K. and 99 in Israel, hinted at why the rates were so different. Before their first birthday, Israeli children frequently ate peanuts, often in a ubiquitous snack called Bamba—something like Cheetos but coated in peanut butter instead of cheese. By the time they were 14 months old, almost 80 percent of the Israeli children were eating at least a few grams of peanut protein every month. In contrast, 80 percent of the British children had never tasted peanuts.

> Early introduction clearly will prevent peanut allergy. “It does work. It’s the right thing to do.” —Gideon Lack, *King's College London*

It made sense that kids in the U.K. weren’t eating peanuts because at the time, medical authorities there and in the U.S. recommended that allergy-causing foods be kept out of the diets of children from allergy-prone families until they were three years old. Lack and his team wondered whether the Israeli experience showed that this well-meaning advice might be wrong. They set up a fresh study, recruiting families with infants who were between four and 10 months old and had severe eczema or showed evidence of egg allergy, signs that their IgE production was already disrupted. The babies were tested for preexisting peanut allergy, and if they were negative, they went into one of two groups. The families of one group were told to keep their children from eating peanuts until they were five years old. The rest of the families were encouraged to introduce their kids to peanut products, preferably Bamba or peanut butter.

When the investigators tested the children five years later, the differences were stark. Among the children told to avoid peanuts, 13.7 percent developed peanut allergies. Among the children who began eating peanuts early, only 1.9 percent did—an 86 percent difference.

Lack published the results in 2015, working with a team primarily from King's College London, where he had moved to research pediatric allergies. This study, called LEAP (for "Learning Early About Peanut Allergy," in a departure from long acronyms), caused an earthquake in allergy science. Anthony Fauci, at the time the director of NIAID, which helped to fund it, said it had "the potential to transform how we approach food-allergy prevention."

Two more studies cemented the findings. In one, published the following year, children from both arms of the LEAP study were asked to not eat peanut products in their sixth year. Allergy rates rose further among the children who had refrained from peanuts all along, but children who started eating peanut products early maintained their low rates of allergy.

In a third, published in 2024, the team went back to children who had been in the LEAP study and were at least 12 years old to check whether the preventive effect lasted. It had. In the group that refrained from peanuts up to age five, 15.4 percent were allergic to peanuts. In the group that ate peanut products early, only 4.4 percent had bad reactions. Early introduction “overwhelmingly will prevent peanut allergy,” Lack says. “It clearly has been shown that it does work. It’s the right thing to do.”

But there have been persistent challenges to implementing that idea. Health authorities no longer recommend that parents avoid feeding allergy-related foods for three years—but most national and international guidelines still recommend exclusive breastfeeding for six months, and the cultural pressure to maintain that time frame is immense. In 2019 the American Academy of Pediatrics did revise its guidance to allow the introduction of potential allergens at four to six months for children who seem likely to be at high risk, indicated by symptoms of eczema.

Lack worries this approach doesn’t provide exposure as early in life as the immune system needs; the children in the LEAP study and in an unrelated 2016 study of early introduction began peanut exposure at four and three months, respectively. “To introduce peanuts effectively in a four-month-old baby, they need to be trained to eat solids already,” he says. “If you start the weaning process at four months, then the baby may not get peanut butter in significant quantities until five to six months of age. And if it’s a baby with eczema, it’s too late.”

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The challenge of prevention at this point may be not the science of immunology but rather the science of implementation. Scientists have to persuade parents and health-care providers that it's safe to implement new knowledge. Immunologists and allergists are aware that early feeding prevents allergy. Pediatricians, who have to handle many additional issues in young children's lives, might not have caught up. But "an allergist isn't going to see somebody who doesn't have peanut allergy already," NIH's Fulkerson says. "You have to get the pediatricians involved because they're the ones who see the babies first."

As many advances as there have been in the past decade, scientists worry that the fundamentals of peanut allergy still elude them. Why it exists, what triggers it, what keeps the immune system from outgrowing it—these basic questions remain unanswered. But the ability to tackle them is growing. “This field is still relatively early in its development compared with oncology or respiratory medicine, which are targeting very specific biological pathways with very specific precision treatments,” Emory’s Vickery says. “We’re not close to that yet. But can I see that on the horizon? Yes.”

The very latest approaches may involve new technologies. At the University of California, Los Angeles, a team led by Andre Nel has developed a lipid nanoparticle that uses mRNA—the same technology used in the COVID vaccines that were developed rapidly in 2020—to create fragments of peanut allergens. Those fragments are presented to specific cells. In mice, the treatment damped down the IgE cascade that triggers anaphylaxis.

But this is a difficult time for biomedical research, given political decisions in the White House and its newly created Department of Government Efficiency (DOGE) to cancel much of the science emanating from the NIH and the National Science Foundation. Peanut allergy may be due for particular attention from the Trump administration. The president’s Secretary of Health and Human Services, Robert F. Kennedy, Jr., has several times endorsed an unsupported contention that peanut allergy is caused by childhood vaccinations. Earlier this year “peanut allergies” appeared on a list of topics that would cause grants to get extra scrutiny within the NIH.

Despite the potential political interference, for now the future seems bright for patients such as Anabelle Terry. As she grows up, the science that has reduced the risks of her allergy is growing along with her. It already has improved her life. It might one day change it for good. “If I go off to summer camp, I have to go away from the other kids for a while and take my nuts to make sure nobody else who has a nut allergy gets sick,” she says. “Going on vacations, I always have to bring a giant bag of nuts with me in my backpack. It would feel pretty nice just being able to go in for a little visit and just get a shot. That would let off a big burden."

Maryn McKenna is a journalist specializing in public health, global health and food policy and is a contributing editor at *Scientific American*. She is author of *Big Chicken: The Incredible Story of How Antibiotics Created Modern Agriculture and Changed the Way the World Eats* (National Geographic Books, 2017).

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https://www.scientificamerican.com/article/new-treatments-can-free-kids-from-the-deadly-threat-of-peanut-allergy

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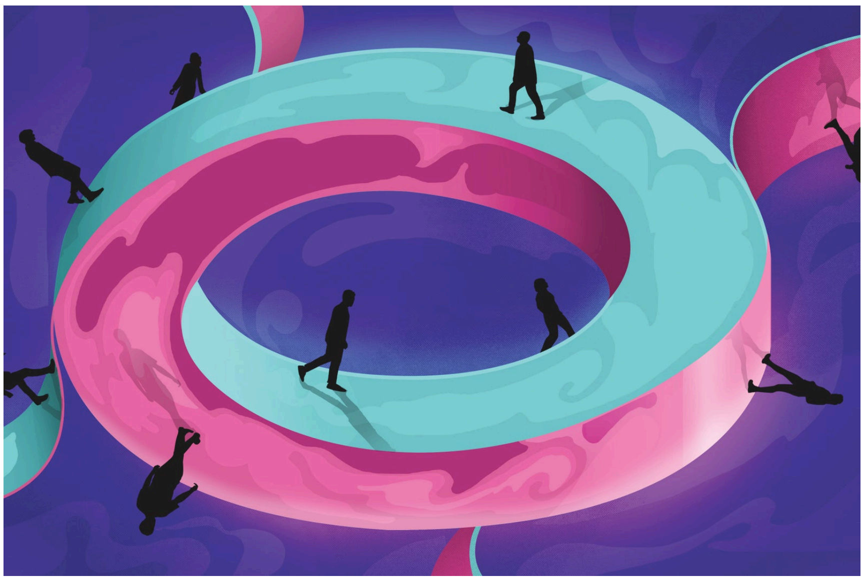
What Happens When an Entire Generation of Scientists Changes Its Mind

Total reversals in scientific thinking are rare—but earth-shattering

By Charles C. Mann edited by Josh Fischman


Sam Falconer

If there is a beginning time point for the Age of Scientific Reversal, it may be 1887—the year when Albert A. Michelson and Edward W. Morley conducted what is often called the world's most famous failed physics experiment.

For more than two centuries researchers had proposed that light was a wave of some kind traveling through an ineffable material that pervaded everything, even the space between atoms. No evidence of this all-permeating substance—the aether, as it was called—had ever been detected. Still, most scientists firmly believed it must exist. How could a wave be seen to travel unless there were something it was traveling through? Working in Cleveland, Ohio, Michelson and Morley sought to measure the aether's effects with some of the most sensitive equipment ever built. To their shock, they found absolutely no trace of it.

Baffled and discouraged, the two men gave up plans for follow-up experiments. Other physicists were even more dismayed. The great theoretical physicist Hendrik Lorentz said the results put him "utterly at a loss."

Yet they were not a loss for science. The Michelson-Morley tests actually led to a remarkable intellectual 180-degree turn and a forward leap in physics. The aether, scientists had believed, would provide a fixed background—a universal reference for all celestial objects. The discovery that outer space was a featureless, nearly empty vacuum—which stemmed from Michelson and Morley's work—meant that objects could be located only in reference to one another. And that realization fed into an even bigger 180-degree turn: Albert Einstein's theories of special and general relativity, which upended previous notions of gravity and turned space and time into a single curvature created by mass and energy.

Or ... or ... maybe the Age of Scientific Reversal began after 1860, when chemist and microbiologist Louis Pasteur presented a long, bluntly written memoir that proved fermentation was caused by microorganisms, not some self-starting chemical reaction, which was the reigning theory. Pasteur's work led to a pitched intellectual battle—and the eventual triumph of germ theory, which overturned earlier ideas about infectious disease.

Coming one after another, such volte-faces gave rise to a popular notion of scientific progress as a series of upheavals in which mavericks throw out the entrenched views of the past. In countless stories in movies, television and novels, revolutionary thinkers (or, rather, wannabe revolutionaries) have their ideas dismissed by hidebound colleagues, yet they triumph in the end.

But that’s not how science works. Or, more precisely, it’s not how science works except in two specific, relatively unusual circumstances.

The first is when research disciplines are young, thinly populated and just developing instruments of sufficient power to test their initial beliefs, as was the case with the Michelson-Morley experiment and Pasteur’s fermentation. The second, possibly more consequential situation is when scientific findings lead to so much public interest that they become of concern to political authorities. Contemporary examples, such as the fraught debate over whether women under the age of 50 should be routinely screened with mammograms, have filled recent headlines. But these political issues have influenced science in the U.S. since at least the 19th century, when the country began trying to move immigrants across the Mississippi River into what might or might not have been hostile, uninhabitable land.

The image of scientific rebels forcing other researchers to reverse themselves was codified in philosopher Thomas Kuhn’s 1962 book, *The Structure of Scientific Revolutions*. In Kuhn’s view, there are periods of “normal science” in which researchers have a shared consensus—a paradigm, in his terms—about how nature works. Then a new theory or experiment shatters the paradigm. Believers in the old paradigm resist furiously, but eventually the old ideas are ejected. From the reversal emerges a new paradigm, which will be thrown over in turn.

Structure was a bombshell. It is one of the few academic tracts to leap outside the classroom and influence the larger culture. Since its publication, stories about “revolutionary” new scientific studies that “overthrow everything we believed” have become staples in journalism, Hollywood and YouTube health-influencer videos.

The lighter side of this trope is embodied in characters such as Doc Brown, the DeLorean-driving inventor in the 1985 movie *Back to* *the Future*, whose unconventional ideas about time travel cause his colleagues to dismiss him as a crackpot. The darker side leads to figures such as discredited anti-vaccine researcher Andrew Wakefield and germ-theory denialist and writer Mike Stone, whose followers claim that their findings have been suppressed by the scientific establishment in the name of profit and political ideology.

The reality is closer to what happened with Michelson and Morley. Physics as a field of knowledge has existed at least since the times of Greek savant Thales (circa 625–545 B.C.E.). But the professional discipline—practiced by credentialed professors who work in specialized laboratories and belong to learned societies—was in its infancy when the two scientists looked for the aether. The U.K.'s first specialized physics group, the Physical Society of London, had been founded just 13 years earlier.

Physicists in those early years were reexamining ideas that often dated back to the Greeks (Aristotle, in the case of the aether) and had yet to be probed with modern tools. Michelson and Morley bounced light among 16 specially prepared mirrors with positions that had to be adjusted so precisely that the two men had to machine custom-calibration screws with 100 threads per inch—implements that couldn't have been made in Thales's time or even Isaac Newton's. Given that many of the foundational assumptions in physics had never been carefully tested, it seems almost inevitable in retrospect that a considerable number would fall to the first scrutiny.

Consider the long-standing belief that the universe preserves parity—that the mirror reflection of any physical process is identical to its unmirrored counterpart except for being flipped from left to right. This is obviously true in the world we live in: shooting one billiard ball at another will have the same effect no matter what direction the cue ball comes from. But matters are less obvious in the quantum realm.

In 1956 physicists Chen Ning Yang of Princeton University and Tsung-Dao Lee of Columbia University wondered whether anyone had proved that parity was preserved in quantum interactions—and found that nobody had checked the "weak nuclear force," which is responsible for radioactive decay. The first research team that looked at the weak interactions, led by Columbia's Chien-Shiung Wu, found that the weak force did not conserve parity. Stunned, Yang sent a telegram to physicist J. Robert Oppenheimer about Wu's experiment. "Walked through door," the gobsmacked Oppenheimer cabled back.

Lee and Yang won the 1957 Nobel Prize in Physics for beginning the parity U-turn. But that was arguably the last time particle physics went through such a sweeping reversal. Yes, the field has seen extraordinary discoveries since then—quarks and gluons, neutrino oscillations, gravity waves, you name it. But they were new phenomena, not refutations of prior beliefs.

The lack of 180s partly results from the way that scientific disciplines ground themselves over time. In retrospect, one can't be surprised that the first experiment to carefully examine the aether failed to find it. But it would be extremely surprising if, after decades of experimental verification, quarks were shown not to exist. In addition, as disciplines grow older and bigger, they end up naturally absorbing people with minority points of view. So instead of entire disciplines executing a U-turn, these minority beliefs shift and twist while becoming acceptable to the majority.

In particle physics, an idea known as *S*-matrix theory dominated in the 1950s and 1960s, but it always had skeptics. When experiments pointed toward an alternative—a quantum field theory and quark model—the field shifted. But it wasn't exactly a U-turn, because quantum field theorists had been working on their ideas all along. And *S*-matrix theory never vanished. It morphed into string theory, a current attempt to unify relativity and quantum mechanics.

Similarly, one of the first vogues in the field of artificial intelligence was the perceptron, a computational system that 1960s-era AI researchers argued would rival human intelligence and ultimately lead to machines with true consciousness. Researchers published thousands of papers extolling and developing perceptrons—an outburst that stopped abruptly after 1969, when computer scientists Marvin Minsky and Seymour Papert took a careful, Michelson-Morley-style look at the idea. They detailed basic tasks that perceptrons could never do, including distinguishing between odd and even numbers. With this embarrassment the perceptron bubble popped. But it didn't disappear. As AI research slowly grew, perceptrons changed into more sophisticated neural networks, which in turn played a role in the development of today's "large language model" artificial intelligence.

Youthful fields can take even more dramatic turns. Pasteur's work on the role of microorganisms in infectious disease inaugurated the modern discipline of microbiology—and led to a host of about-faces in previous medical beliefs. German researcher Robert Koch, often considered microbiology's co-founder, then discovered the microbes that caused anthrax, cholera and tuberculosis. All cast aside earlier ideas. For instance, many in Koch's Germany believed tuberculosis was a hereditary disease passed down through families until 1882, when the scientist unveiled *Mycobacterium tuberculosis*, the bacterium responsible for the disease.

> In the researchers' map of the region drained by the Mississippi was a label: GREAT AMERICAN DESERT.

These reversals did not always have the revolutionaries and traditionalists one would expect on opposing sides. French physician Alphonse Laveran spotted microscopic living creatures in malaria patients' blood in 1890. Since the Greeks, doctors had believed that malaria was brought about by "miasma"—misty air polluted with particles from decomposed matter. (The disease's name comes from *mal aria*, or “bad air” in early Italian.) Based on his observations, Laveran proclaimed that malaria was caused by protozoans. These microbes are now known to be several species in the genus *Plasmodium*.

Laveran’s fiercest critics were not miasma theorists, however, but Pasteur’s disciples, who insisted a la Kuhn on yet another paradigm: infectious diseases were caused by bacteria—bacteria floating in mist, in this case. France’s leading malaria authority sneered at Laveran, as did Koch. In reaction, Laveran doubled down, proposing that *Plasmodium* was carried by mosquitoes, not mist. This, too, was dismissed. But Laveran was subsequently proved right, and within a decade scientists had to reverse themselves again.

Paralleling the rise of institutional particle physics, the microbiology of Pasteur and Koch expanded into an enormous discipline with thousands of researchers, multiple subfields—and ever fewer reversals. Today the International Union of Microbiological Societies has 57 groups from 45 nations; Italy, long a center for this kind of research, has six professional societies of its own. Last year’s annual meeting of the American Society for Virology attracted more than 2,000 attendees from 50 countries.

Then there is the political influence on scientific 180s. In the U.S., politics and science collided right after 1803, the year of the Louisiana Purchase. The U.S. government knew so little about its new possession that it dispatched no fewer than four teams to survey the territory. One, led by U.S. Army officers Meriwether Lewis and William Clark, crossed the continent by a northern route and became a celebrated part of American history. The three other expeditions went into the southern and central plains and were repelled by Spanish troops and Indigenous nations. Not until 1819 did the U.S. try again, sending a team led by engineer Stephen H. Long.

Although Long didn't know it, the southern plains were beset by a multiyear drought. While surveying the Platte and Canadian Rivers, his team almost starved. Unsurprisingly, the expedition's report portrayed the southern plains as "presenting the aspect of hopeless and irreclaimable sterility." The land was "almost wholly unfit for cultivation, and of course uninhabitable by a people depending upon agriculture for their subsistence." In the center of the team's map of the "country drained by the Mississippi" was a capitalized label: GREAT AMERICAN DESERT.

Today we know that in the central and southern plains, long-term atmospheric fluxes from the Pacific (the El Niño–Southern Oscillation, for example) and the Atlantic (the Atlantic Multidecadal Oscillation) mix with warm, moist air currents from the Gulf of Mexico and cold, dry air from the Arctic jet stream. These phenomena collide unpredictably, causing tornadoes, blizzards, severe hailstorms, epic heat waves and, notably, lengthy droughts—the 1930s Dust Bowl being the most well known.

In what one pictures as the usual course of events, Long's report would have been followed by other surveys, some challenging his views, some backing them. Presumably the back-and-forth would slowly have revealed that Long's belief that aridity was the region's permanent state was incorrect because arid and wet periods came in irregular cycles. Yet that realization was not what happened, because politicians and wealthy interests, especially new railroad tycoons, wanted people to move to the plains and create communities that both produced and bought goods and crops. These would, of course, be transported by trains.

So not only did critics dispute the existence of the Great American Desert, but they said that rainfall in the area was increasing—because of farming. In his 1880 book *Sketches of the Physical Geography and Geology of Nebraska*, University of Nebraska scientist Samuel Aughey explained that prior to the arrival of Europeans, the prairie had been "pelted by the elements and trodden by millions of buffalo," which packed the soil too hard to absorb water. But with settlers' plows breaking up the hardpan, "the rain as it falls is absorbed by the soil like a huge sponge." More water retained in the land means more evaporation over it, which "must give increasing moisture and rainfall." A slogan emerged: "Rain follows the plow."

Researchers led by geologist John Wesley Powell, director of the U.S. Geological Survey, countered that the region was too drought-prone to sustain agriculture. Early rangeland scientists mocked the idea that the precolonial grasslands couldn't retain moisture. But their assertions were buried underneath floods of flyers, leaflets and advertisements from railroads that extolled Long's Great American Desert as a Great American Garden. When a multiyear drought overtook the region in the 1890s, it was a shock—a complete reversal of the expectations of migrants who had relied on the railroads' descriptions. They fled the area in droves. After the rains returned, new migrants poured in. The 1930s Dust Bowl, when it came, was just as much of a shocking 180 to them as the previous drought had been to their predecessors.

The argument over the climate in the plains was an early example of an increasingly common phenomenon: the mismatch between the slow, unsteady movement of scientific understanding and the immediate, short-term imperatives of politics and economics, which can lead to what seem like vertiginous scientific reversals.

Examples are as near to hand as the COVID pandemic. Early in the epidemic, in March 2020, the World Health Organization (WHO) avowed that COVID could not be transmitted through the air—people picked up the SARS-CoV-2 virus from surfaces. ("FACT"—the agency tweeted—"#COVID19 is NOT airborne.") Other public health outfits followed suit. Air-pollution specialists, including those within these outfits, were astounded by the claims. In their discipline, it was well known that large particles of soot could travel through the air for miles.

Lidia Morawska, an aerosol specialist at the Queensland University of Technology in Brisbane, Australia, led a group of aerosol researchers and ventilation engineers that contacted WHO about the lengthy travel distances days after the "FACT" tweet. Dismissing this evidence as weak, a WHO advisory group insisted in August 2020 that "SARS-CoV-2 is not spread by the airborne route to any significant extent."

In part, WHO's reluctance was a legacy of the previous battle over miasma theory. The fight to eliminate the fear of vapors led infectious disease experts to take as given that nearly all infectious pathogens were spread by "droplets," generally defined as more than five microns in diameter. Droplets fly out of sick people's mouths and noses when they cough, shout, sing or sneeze. The particles then land directly on other people or on nearby surfaces that people later touch. Implicit in the definition of droplets was that their relatively large size limited their ability to travel. Thus, WHO focused on getting people to wash surfaces and hands to stop the spread of the virus. Aerosol transmission, in which smaller organisms travel farther in vapor clouds, was thought to occur only for a few well-known diseases, mainly tuberculosis and measles.

WHO tenaciously stuck to its paradigm despite a tsunami of reports of aerosol transmission. Only gradually did the agency admit that such transmission was possible in specific "crowded and inadequately ventilated [indoor] spaces" (July 2020), that the virus could travel in the air "farther than 1 metre" in specific settings (April 2021), and, finally, that "airborne" transmission could occur in some places (December 2021)—a move that was greeted as a long-overdue 180.

The reversal was Kuhnian in the sense that WHO's scientific paradigm was overturned after resistance. But the scientists who rejected Michelson-Morley were motivated mainly by adherence to scientific orthodoxy, whereas WHO researchers were also responding to an intensely political environment. Agencies such as

WHO are supposed to provide guidance for others to act on. Under public pressure to be definitive, they often end up digging in their heels on research questions that are poorly understood. What would in other circumstances be ordinary back-and-forth as researchers resolved questions is transformed into a series of stark, headline-grabbing reversals.

Perhaps nothing better illustrates this type of politically driven reversal than the five-decade controversy over mammograms for women between 40 and 50 years of age. In the early 1970s the National Cancer Institute (NCI) and the American Cancer Society launched the Breast Cancer Detection Demonstration Project (BCDDP) to test the potential of large-scale mammography. Some cancer researchers protested that repeatedly exposing women under 50 to x-rays would do more harm than good, so the BCDDP restricted enrollment of younger women to those at "high risk."

The results were released in the 1980s. Although the BCDDP design had weaknesses, the study authors said the results showed that mammograms detected breast tumors that would not otherwise have been spotted. And screening did not produce excessive false positives, which can lead to needless biopsies and surgeries.

The NCI and almost 20 other medical organizations met to establish guidelines for mammography. A reanalysis of another, earlier, smaller trial, the Health Insurance Program of Greater New York study, also showed that mammography for younger women had positive effects. The combined result was nationwide recommendations, issued in 1989, that women should begin screening for cancer at age 40—and spurred a big advertising campaign by advocacy groups to convince women to do it.

But then, in 1992, the Canadian National Breast Screening Study of cancer—the first randomized clinical trial designed specifically to examine the effectiveness of under-50 mammography—released a contradictory result: testing younger women did not reduce death rates.* Big randomized clinical trials are generally considered the best way to understand the efficacy of medical treatments. Nevertheless, this one was furiously attacked by cancer advocacy groups, clinicians and radiologists, who asserted something must be wrong with the way the trial was done. Oddly, after the NCI convened a workshop on the issue that concluded "there is no reduction in mortality from breast cancer that can be attributed to screening," the institute also insisted there was no need to change the recommendation for earlier screening. It cited vague "inferential" benefits.

Troubled by the idea of basing nationwide recommendations on what experts judged as low-quality evidence, Samuel Broder, then director of the NCI, announced the institute would not promote screening for women in their 40s. In his view, the potential good effects (possibly catching a few relatively rare cancers early) were far outweighed by the potential bad effects (those false alarms that scare women and can lead to many painful and unnecessary surgeries).

The National Cancer Advisory Board—an NCI advisory group of federal-agency officials, representatives from cancer associations, and cancer researchers—asked Broder not to pull back right away. He and the NCI stuck to their guns. Then U.S. Congress members erupted, calling the institute callous and sexist.

The American Cancer Society, the American College of Radiology, and other medical groups conceded that there weren't good data to support under-50 mammography. But they felt obligated to do something to address younger women's fear of breast cancer—a fear that was inflamed, in part, by the organizations' own public-relations campaigns promoting mammograms and breast self-exams.

Both sides continued their standoff until 1997, when the NIH convened a consensus conference to try to resolve the issue. It concluded that the current data didn't support under-50 screening. But the hoped-for consensus collapsed when critics, such as a mammography director at a private practice in New Mexico, charged that the agency statement was "tantamount to a death sentence for thousands of women in their forties." Congress voted 98–0 to order the NCI to back screening for younger women. The institute caved. The American Cancer Society joined it to state that screening for women in their 40s was "beneficial and supportable with current evidence."

Little of this controversy was visible in doctors' offices, where women were being told that screening that begins at 40 saves lives. Outside of those offices, advocacy groups were saying the same thing. So many patients were shocked by headlines in 2009, when the U.S. Preventive Services Task Force (USPSTF), an independent and influential expert board advising the federal Department of Health and Human Services (HHS), went in the opposite direction. It said that almost 2,000 younger women would have to be screened to save one life. The other 1,900-plus women would be exposed to the risks of radiation and surgery.

The White House denounced the USPSTF's stance. The task force backed down, saying instead that women should consult their doctors—an embarrassing break with its mission, which was to assess the state of evidence for entire fields rather than telling patients to rely on the opinions of individual practitioners. Congress passed a law explicitly telling HHS to disregard "the current recommendations of the United States Preventive Services Task Force regarding breast cancer screening."

Then, in 2024, there was an actual reversal. The USPSTF issued *another* set of recommendations—but this time it came out in favor of routine mammograms for women in their 40s.

Throughout this time, the data had changed little. When put together, the eight big randomized controlled trials of mammography for women under 50 have shown that the tests produce very high, specific benefits for a small number of women and impose other costs on a much larger number of women. Yet the glare of publicity transformed a slow but fairly typical research debate into a huge controversy culminating in a big 180.

This kind of politically charged reversal shows little sign of declining. Likely future reversals may include causes and treatment of obesity or of Alzheimer's disease. All are the subjects of intense lobbying by commercial and public-interest groups.

As for reversals in fields where scientific ideas compete in disciplines that lack adequate investigatory tools, who knows? But hints may come from cosmology, where grand ideas about the nature of the universe jostle for prominence. These notions are constrained by the difficulty of gathering data but still driven forward by scientists seeking the thrill of causing yet one more scientific twist.

**Editor's Note (9/10/25): This sentence was edited after posting to correct the name of the Canadian National Breast Screening Study.*

Charles C. Mann is author of, most recently, *The Wizard and the Prophet* (Picador, 2018). His book *1491* won the National Academy of Sciences' Keck Award for best book of 2006.

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https://www.scientificamerican.com/article/what-happens-when-an-entire-scientific-field-changes-its-mind

How Scientists Finally Learned That Nerves Regrow

Long dismissed as impossible, nerve regrowth is offering new hope for healing injuries and treating neurodegeneration

By Diana Kwon edited by Madhusree Mukerjee

Sam Falconer

Billions of nerve cells send signals coursing through our bodies, serving as conduits through which the brain performs its essential functions. For millennia physicians thought damage to nerves was irreversible. In ancient Greece, founders of modern medicine such as Hippocrates and Galen refused to operate on damaged nerves for fear of causing pain, convulsions or even death.

The dogma stood relatively still until the past two centuries, during which surgeons and scientists found evidence that neurons in the body and brain can repair themselves and regenerate after injury and that new nerve cells can grow throughout the lifespan. In recent decades this knowledge has inspired promising treatments for nerve injuries and has led researchers to investigate interventions for neurodegenerative disease.

In humans and other vertebrates, the nervous system is split into two parts: the central nervous system, composed of the spinal cord and brain, and the peripheral nervous system, which connects the brain to the rest of the body.

Attempts to suture together the ends of damaged neurons in the peripheral nervous system date back to the seventh century. It was only in the late 1800s, however, that scientists began to understand how, exactly, nerves regenerate. Through his experiments on frogs, British physiologist Augustus Waller described in detail what happens to a peripheral nerve after injury. Then, in the 1900s, the influential Spanish neuroanatomist Santiago Ramón y Cajal provided insight into how nerve regeneration occurs at the cellular level. Still, there remained fierce debate about whether stitching nerves together would harm more than help.

It was against the backdrop of bloody world wars of the 20th century that physicians finally made significant advances in techniques to restore damaged neurons. To treat soldiers with devastating wounds that typically involved nerve damage, doctors developed methods such as nerve grafts, in which pieces of nerves are transplanted into the gap in a broken nerve.

Over time physicians learned that some peripheral nerve injuries are more conducive to repair than others. Factors such as the timing, location and size of the injury, as well as the age of the patient, can significantly impact the success of any given intervention. Crushed nerves are likelier than cut ones to be repaired, and injuries that occur closer to a nerve’s target tissue have a greater chance of regaining function than those that occur farther away. Take the ulnar nerve, which stretches the entire length of the arm and controls key muscles in the lower arm and hand. A person with nerve damage near the wrist is much more likely to regain function in the arm and hand after undergoing treatment than someone who injures the same nerve near the shoulder, in which case it must regrow from the shoulder all the way to the wrist.

Even today many peripheral nerve injuries remain difficult to treat, and scientists are striving to better understand the mechanisms of regeneration to facilitate healing. One notable development in recent years, according to neurologist Ahmet Höke of the Johns Hopkins University School of Medicine, is a “nerve transfer,” in which a branch of a nearby nerve is rerouted to a damaged nerve. In cases where, for example, a nerve is damaged far from its target muscle, existing techniques may not be sufficient to enable regrowth across the long distances involved within a time frame allowing for recovery. This detour provides an alternative pathway to regain function. Susan Mackinnon, a plastic and reconstructive surgeon at Washington University in St. Louis, has largely driven the advances in nerve transfer, enabling patients to use their limbs after peripheral nerve injuries that previously would have led to a permanent loss of movement in them.

For instance, Oskar Hanson, a high school baseball player, lost sensation and movement in most of his left arm after a surgery to mend a ligament injury ended up damaging the ulnar nerve in that arm. “There was zero hope that he would be able to have use of his arm again,” says his mother, Patricia Hanson. But after Mackinnon performed a nerve transfer procedure, most of the function returned. “She saved his life with that surgery,” Hanson says.

Despite the leaps that were made in treating peripheral nerve injuries, the notion that neurons within the central nervous system—the brain and spinal cord—were incapable of regrowth persisted until the late 20th century.

A pivotal moment came in the early 1980s, when Canadian neuroscientist Albert Aguayo and his colleagues demonstrated that in rats, neurons of the spinal cord and brain stem could regrow when segments of peripheral nerves were grafted into the site of injury. These findings revealed that neurons of the central nervous system can also regenerate, Höke says: “They just needed the appropriate environment.”

In succeeding years, neuroscientists worked to uncover what, exactly, that environment looked like. To do so, they searched for differences in the peripheral and central nervous systems that could explain why the former was better able to repair damaged neurons. Several key differences emerged. For example, only injuries within the central nervous system led to the formation of glial scars—masses of nonneuronal cells known as glial cells. The purpose of these scars is still debated, however.

Today the search for the specific mechanisms that prevent or enable neuron regrowth—in both the body and the brain—remains an active area of investigation. In addition to uncovering the processes at play in humans, scientists have pinpointed molecules that enable nerve cell repair in other organisms, such as “fusogens,” gluelike molecules found in nematodes. Researchers are attempting to harness fusogens to help with difficult-to-treat human nerve injuries.

Modern neuroscientists have also challenged another long-standing doctrine in the field: the belief that the adult brain does not engage in neurogenesis, the creation of brand-new nerve cells.

Early clues for neurogenesis in the brain emerged in the 1960s, when researchers at the Massachusetts Institute of Technology observed signs of neurons dividing in the brains of adult rats. At the time, these findings were met with skepticism, says Rusty Gage, a professor of genetics at the Salk Institute for Biological Studies in La Jolla, Calif. “It was just too hard to believe.”

Then, in the early 1980s, neuroscientist Fernando Nottebohm of the Rockefeller University discovered that in male songbirds, the size of the brain region associated with song-making changed with the seasons. Nottebohm and his colleagues went on to show that cells in the animals' brains died and regenerated with the seasons. Inspired by these findings, researchers looked for signs of adult neurogenesis in other animals. In 1998 Gage and his colleagues revealed evidence of this process occurring in the brains of adult humans—specifically within the hippocampus, a region linked with learning and memory.

Although support for adult neurogenesis in humans has amassed over the years, some experts still debate its existence. In 2018 a team co-led by Arturo Alvarez-Buylla, a neuroscientist at the University of California, San Francisco, who had worked with Nottebohm on songbirds, published a study stating that the formation of new neurons was extremely rare, and likely nonexistent, in adult human brains.

Still, there's a growing consensus that neurogenesis does happen later in life—and that this growth appears to be largely limited to certain parts of the brain, such as the hippocampus. This past July a team at the Karolinska Institute in Sweden reported that the molecular signatures of precursors of neurons, known as neural progenitor cells, were present in the human brain across the lifespan—from infancy into old age. Researchers are now trying to understand the purpose of these budding nerve cells and asking whether they might offer clues for treating neurodegenerative disorders such as Alzheimer's disease. Some scientists are even exploring whether, by targeting neurogenesis, they can improve the symptoms of psychiatric conditions such as post-traumatic stress disorder.

Understanding that a neuron can regrow and be repaired and identifying details of that process has been a great achievement, says Massimo Hilliard, a cellular and molecular neurobiologist at the University of Queensland in Australia. The next step, he adds, will be figuring out how to control these processes: “That’s going to be key.”

Diana Kwon is a freelance journalist who covers health and the life sciences. She is based in Berlin.

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https://www.scientificamerican.com/article/how-scientists-finally-learned-that-nerves-regrow-even-in-the-adult-brain

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Plastics Started as a Sustainability Solution. What Went Wrong?

Synthetic polymers were supposed to free us from the limitations of our natural resources. Instead they led to an environmental crisis

By Jen Schwartz edited by Seth Fletcher

Sam Falconer

In 1864 *Scientific American* published a competition launched by a billiard-table manufacturing company: “Ten Thousand Dollars for a Substitute for Ivory.” The owners of Phelan & Collender were pleased to see it; they wrote to the magazine to elaborate on what they were looking for in an “ivory alternative” that could be used to make billiard balls and hoped it would “have the effect of stimulating the genius of some of your numerous readers.” The real stuff from elephant tusks had become scarce, but its elasticity, hardness and density were hard to find in another material.

A printer from Albany, N.Y., named John Wesley Hyatt came up with an answer in celluloid, a moldable, compound material made up of cellulose nitrate, a polymer that held the ball together; camphor, an organic compound that provided flexibility and durability; and ground-up cow bone, to give the ball the right mechanics for play. Rather than accepting the $10,000 reward and signing away the rights to his invention, Hyatt patented his object in 1869 and started his own company, selling celluloid billiard balls that conservation scientist Artur Neves, writing in 2023, called "the founding object of the plastics industry."

The creation of the "first plastic" was essentially an answer to a sustainability problem. There were only so many elephants, tortoises and silkworms to go around, and their tusks, shells and fibers were increasingly in demand. Articles and advertisements from the early era of the plastics industry portray such materials as relieving pressure on natural resources. In a 2023 paper in *PNAS Nexus*, Neves and his colleagues called Hyatt's celluloid billiard balls one of "the first successful efforts to substitute materials to assist the survival of endangered animals."

The billiard ball and other reinforced polymer composites were predecessors to commercial plastics. But the term "plastic" was nebulous, more marketing language than scientific category. Philip H. Smith, writing in *Scientific American* in 1935, defined it as "the name given to a more or less arbitrarily chosen group of substances which, when properly compounded and treated, become plastic and can be molded or cast to shape."

In *American Plastic: A Cultural History*, published in 1995, Jeffrey L. Meikle writes that the fear of an ivory shortage that stimulated plastics development shifted in the 20th century to the idea of democratizing luxury items. Mass production of plastics for a wide range of uses began in the 1940s, when production in the U.S. nearly tripled over the war years. This expansion coincided with the replacement of bio-based materials (such as cotton, soybeans and sugar) in polymer bases with fossil fuels, which were promoted as an abundant resource. To give products specific properties, additives such as colorants, plasticizers (such as phthalates and bisphenol A) and flame retardants were included in the polymers during manufacturing.

You know where this story goes. By the 1970s, Meikle writes in his book, “plastic’s ability to transcend nature often no longer seemed utopian but instead simply disastrous.” Plastics had ushered in an era of excessive stuff that was cheap to make. Materials originally celebrated for their durability and longevity became popular in single-use items. Ninety percent of plastics aren’t technically recyclable anyway, and some now argue that recycling campaigns only encouraged people to feel better about buying more plastic things. Because plastic is not biodegradable, it simply accumulates, fragmenting into ever smaller pieces over hundreds or thousands of years. In 2009 the first comprehensive review of the impact of plastics on the environment and human health was published—a collection of consequences and warnings that have gotten only more dire.

Now researchers are investigating the wide-ranging presence and effects of microplastics—tiny specks that leach toxic chemicals into the environment. Single-use items such as water bottles are an obvious part of the problem, but there are many other culprits. Until the mid-1990s, natural fibers dominated the fashion industry; in 2023 synthetic polymers made up 67 percent of global fiber production, with polyester alone making up 57 percent of all new clothing, home textiles and shoes. These products shed microplastic fibers with every wash, contributing to pollution in groundwater. These contaminants, which are basically impossible to clean up, are not just present in soil and water: A new study found the leaves of plants absorb microplastics from the air. All animals studied, including us, are not just eating plastic in our food and drinking it in our water; we now have plastic in our organs.

The solution to one environmental sustainability problem has become one of the biggest and most intractable environmental crises of our time. As Rebecca Altman wrote in a 2021 article in *Science*, celluloid "purportedly spared the elephant, especially from the billiard ball industry. [But] market data show that celluloid did not decrease ivory demand, which grew in the years after celluloid's introduction." Celluloid, she adds, also accelerated the demand for camphor, a product distilled from an evergreen tree prevalent in Taiwan. Competition to control the camphor trade destroyed Taiwan's forests and displaced its Indigenous communities. The advent of synthetic polymers didn't free humanity from the limits of natural resources.

What started as a competition to invent an alternative to ivory has turned into competitions for inventing methods to clean up the Great Pacific Garbage Patch and other sprawling plastic icebergs in oceans the world over. In 1942 Williams Haynes, a historian and promoter of the chemical industry, declared that synthetic materials would have "more effect on the lives of our great-grandchildren than Hitler or Mussolini." He couldn't have imagined the biggest impact on future generations might be nanoplastic fragments in their brains.

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.

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https://www.scientificamerican.com/article/how-plastics-went-from-a-sustainability-solution-to-an-environmental-crisis

The Universe Is Static. No, Expanding! Wait, Slowing? Oh, Accelerating

The universe has a habit of disproving "unassailable" facts

By Richard Panek edited by Clara Moskowitz

Sam Falconer

To astronomers in the 1990s, these three facts were self-evident: The universe is expanding; all the matter in the universe is gravitationally attracting all the other matter in the universe; therefore, the expansion of the universe is slowing.

Two scientific collaborations assigned themselves the task of determining the rate of that deceleration. Find that rate, they figured, and they would know nothing less than the fate of the universe. Is the expansion slowing just enough that it will eventually come to a halt? Or is it slowing so much that it will eventually stop, reverse itself and result in a kind of big bang boomerang?

The answer, which the two teams reached independently in 1998, was precisely the opposite of what they expected.

The expansion of the universe isn't slowing down. It's speeding up.

Cosmology has often lent itself to unthinking assumptions that turned out to be exactly wrong. The ur-example is geocentrism. Over the couple of millennia before the invention of the telescope in the early 1600s, the occasional philosopher suggested Earth orbits the sun and not the other way around. But the vast majority of astronomers could simply look up and see for themselves. The sun orbits Earth. The evidence was, well, self-evident.

But then, most of the history of astronomy had relied on an unthinking assumption: The heavens would always be out of reach. Like the prisoners in Plato's parable, we would forever be at the mercy of our perceptual limitations, trying to make sense of the motions in a two-dimensional celestial realm that was the cosmic equivalent of a cave wall. The invention of the telescope in the first decade of the 17th century overturned both those assumptions: Earth orbits the sun; the heavens are at our fingertips.

More telescopic discoveries followed that, to varying extents, contradicted one self-evident "fact" after another: mountains on the moon, moons around Jupiter, new stars, new planets. Some assumptions turned out to have been not just unthinking but unthinkable. How could anyone in the history of civilization ever have looked at Saturn and thought, "I'm assuming it doesn't have rings"?

That the universe is expanding—the major premise leading to the 1990s search for the deceleration rate—was a revelation that nobody saw coming, including the two theorists who made the discovery not only conceivable but inevitable.

The first, Isaac Newton, would have had to make two counterintuitive leaps of logic to reach such a shocking conclusion. He would have needed to imagine that the universe was capable of doing what it self-evidently was not doing: collapsing. Then he would have needed to conceive of it as doing the opposite: getting bigger.

Albert Einstein, the second theorist who paved the way for the expansion discovery, did conceive of it. In November 1915 he presented the equations underlying his general theory of relativity; 15 months later he applied those equations to, as he phrased the topic in the paper's title, "cosmological considerations." According to his math, the universe should be volatile over time, either expanding or contracting. To avoid that unsettling implication, he introduced a variable, L, the Greek symbol for lambda, to balance his equation. The value of lambda would be whatever it needed to be to satisfy Einstein's preference for a universe in perfect balance.

Each theorist's "blunder," as Einstein characterized his own refusal to trust his math, was understandable. Newton and Einstein, however intellectually exceptional, were still only human. The universe was static. If evidence to the contrary existed, it certainly wasn't obvious.

And then it was. In the early 1920s American astronomer Edwin Hubble deployed the new 100-inch telescope atop Mount Wilson in California to observe some of the nebulous smudges at the farthest reaches of previous telescopes. Using Cepheid variables (stars that brighten and dim with clockwork regularity) as a measure of distance, he inferred that at least some of those nebulae were actually "island universes"—galaxies—beyond our own Milky Way. Next he used the redshifts of those galaxies to infer not only that the galaxies are moving away from us and from one another—itself a science-redefining discovery—but also their rate.

When Hubble plotted those distances against those velocities on an *x/y* graph, he found a direct correlation: the more distant the galaxies, the faster they were moving away from us. Thus, the universe must be expanding. Belgian astronomer Georges Lemaître independently reached the same conclusion, working not from his own data but from Einstein’s equations. Trace the expansion backward, he argued, and you would arrive at a “primeval atom.”

Evidence supporting the existence of such a “big bang” didn’t come until 1964, in the form of a background of microwave radiation that seems to pervade all of space. Theorists had predicted the existence of such a background as the relic of an explosive origin, although the two Bell Labs astronomers who first detected the radiation initially dismissed it as noise, possibly the result of pigeon droppings lining the giant horn of their radio antenna. Four physicists at nearby Princeton University, however, recognized that the observation matched the key prediction of the big bang theory.

Six years later American astronomer Allan Sandage cast cosmology as “the search for two numbers.” One number was the “rate of expansion” now. The other, however, harbored the unthinking assumption that would motivate two teams of researchers a quarter of a century later: “the deceleration in the expansion” over time.

Both teams trying to measure cosmic deceleration followed Hubble’s methodology of plotting velocity versus distance on a graph (using the magnitudes of a type of exploding star, or supernova, rather than Cepheid variables). Both collaborations expected to find the same direct correlation that Hubble did—at least at first. At some distance, though, they assumed that the line would depart from its 45-degree trajectory and dip, indicating that the apparent magnitudes of the supernovae were brighter, and therefore nearer, than they would be in a universe expanding at a constant rate.

And depart from its 45-degree trajectory the line did. Only it didn’t dip. It rose. The supernovae were dimmer, and thus farther away, than they would be in a universe expanding at a constant rate. The expansion of the universe, the rival teams concluded, isn’t slowing down. It’s somehow speeding up.

Dark energy—as cosmologists came to call whatever was causing the acceleration—soon became part of the standard cosmological model, along with dark matter and "regular" matter, the stuff of us. Observations of the same cosmic microwave background that, back in the 1960s, helped to validate the big bang interpretation of cosmology have revealed the universe's ingredients. By studying the patterns in the radiation, scientists have refined the contributions to the mass-energy density of the universe to an exquisite level of precision: 4.9 percent of it must be ordinary matter, 26.8 percent dark matter, 68.3 percent dark energy. The model, cosmologists believe, is solid.

But not flawless. Not even complete. What is dark energy? What is dark matter? Indeed, even after all these years: What is the fate of the universe? Just this year the Dark Energy Spectroscopic Instrument in Arizona provided evidence that dark energy may have changed over the course of the evolution of the universe. Cosmologists have found the evidence compelling, though its meaning—let alone its implications for the standard model of cosmology—remains elusive.

So: Is cosmology on the precipice of another reversal? Another revolution? If history is any guide, the answer is: Maybe. For all today's cosmologists know, they might be laboring under a seemingly unassailable, self-evident, yet incorrect assumption. Perhaps even an unthinking one.

It's happened before.

Richard Panek is the prizewinning author of *Pillars of Creation* (Little, Brown, 2024).

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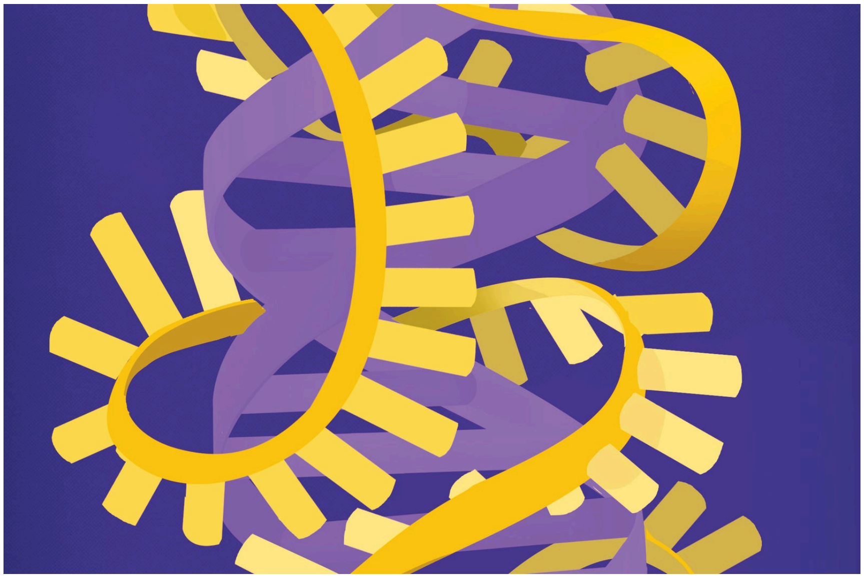
https://www.scientificamerican.com/article/the-universe-keeps-rewriting-cosmology

How RNA Unseated DNA as the Most Important Molecule in Your Body

DNA holds our genetic blueprints, but its cousin, RNA, conducts our daily lives

By Philip Ball edited by Mark Fischetti



Sam Falconer

In 1957, just four years after Francis Crick and other scientists solved the riddle of DNA's structure—the now famous double helix—Crick laid out what he called the "central dogma" of molecular biology, which his colleague James Watson later said implied that biological information flows inexorably from DNA to RNA to proteins. Although Watson was oversimplifying, the message was that the purpose of the double helix in our chromosomes is to hold, in encoded form, blueprints for the proteins that build and maintain our bodies. DNA's chemical cousin, RNA, was the messenger that carries DNA instructions from the double helix in the cell's nucleus to the protein-making machinery, called the ribosome, scattered around the cell.

Molecular biology's mission, it seemed, was to decipher those genetic instructions. But in recent years researchers have discovered a dizzying array of "noncoding" RNA (ncRNA) molecules that do something other than ferry DNA instructions for proteins. They perform a surprisingly wide range of biochemical functions. It now seems that our genome may be at least as much a repository of plans for vital, noncoding RNA as it is for proteins. This shift in thinking has been "revolutionary," says Thomas Cech, who shared the 1989 Nobel Prize in Chemistry with Sidney Altman for discovering RNA molecules, called ribozymes, that can catalyze biochemical reactions. "DNA is old stuff, 20th-century stuff," Cech says. "It's a one-trick pony. All it does is store biological information, which it does exquisitely well. But it's inert—it can't do anything without its children, RNA and proteins."

RNA is created when an enzyme called RNA polymerase reads a DNA sequence and builds a corresponding RNA molecule—a process known as transcription. The discovery, over the past three decades, of thousands of previously unknown noncoding RNAs "has been mind-blowing," says Maite Huarte, a molecular biologist at the University of Navarra in Pamplona, Spain. Noncoding RNA plays many roles, often involving the regulation of other genes—for example, determining whether protein-coding genes get transcribed to messenger RNA (mRNA) and how (or if) that molecule is edited and then translated into a protein. In this case, RNA seems to control how cells use their DNA. These functions turn the popular central dogma, which was a one-way street from DNA to mRNA to proteins, into an open system with information flowing in all directions among DNA, proteins, cells and organism.

Equally fascinating, Huarte says, is that ncRNAs don't belong to just one family of molecules. "RNA is highly versatile, and nature exploits this versatility," she says. Scientists have known since the 1950s that ribosomes contain ribosomal RNA and use transfer RNA to collect amino acids that are stitched together in proteins. But for a long time those seemed like anomalies. Then, in the 1980s, Cech and Altman discovered a new type of ncRNA: ribozymes that cleave and edit themselves and other RNAs. And in the 1990s researchers began to find human ncRNAs that had regulatory functions. A gene called XIST, involved in the "silencing" of one of the two X chromosomes in the cells of chromosomal females, encoded not a protein but a long noncoding RNA that appears to wrap around the chromosome and prevent its transcription.

> "Textbooks 25 years ago confidently stated that RNA consisted of [three types]. Now there are hundreds, likely many thousands, of other types." —Thomas Cech *University of Colorado Boulder*

Meanwhile molecular biologists Victor Ambros and Gary Ruvkun found short noncoding RNA molecules that interact with mRNA to silence a corresponding gene. This extra layer of gene regulation—controlling whether an mRNA is used to make a protein—seems to be an essential feature in the growth of complex organisms. Scientists have linked genetic mutations that hinder gene regulation by ncRNAs to a wide range of diseases, including cancers. "We're getting closer to some really exciting biomedical applications," Huarte says. "From new diagnostic tools to innovative, targeted therapies, the potential of ncRNAs is huge."

Cech says it was a "big surprise" RNA could perform such diverse roles. That surprise was apparent in 2012 when scientists working on an international project called ENCODE reported that as much as 80 percent of our DNA has biochemical function in some cells at some point, and much of that DNA is transcribed into RNA, challenging the long-held belief that most of our genome is "junk" accumulated over the course of evolution.

This is not a consensus view. Some researchers argue that, on the contrary, most of the RNA transcribed from DNA but not translated into protein is "noise," made because the transcription machinery is rather indiscriminate. Such noise will indeed be the end result for some transcription. It now appears that known noncoding genes outnumber genes encoding proteins by a factor of about three, according to some estimates.

It’s often hard, however, to figure out just what the RNA is doing. Some of these molecules might get transcribed only in particular types of cells or at a particular stage in embryonic development, so it would be easy to miss their moment of action. “They are incredibly cell-type specific,” says molecular biologist Susan Carpenter of the University of California, Santa Cruz. But because of that, she says, “the more we look, the more we find.”

Ambiguities notwithstanding, the rise of RNA has transformed molecular biology. “Textbooks 25 years ago confidently stated that RNA consisted of messenger RNA, transfer RNA and ribosomal RNA,” Cech says. “Now there are hundreds, likely many thousands, of other types.” The 21st century, he says, “is the age of noncoding RNA.”

We have much more to learn. We don’t know how much functional ncRNA there is, let alone what the many varieties do. And “when we answer one question, it raises 10 new ones,” Carpenter says. As scientists discover more about the many types and roles of RNA, medical researchers may discover potential therapeutic applications, yet the more profound implications are about how life works. For complex organisms to be viable, it’s simply not enough to have a “genetic blueprint” that gets read. They need to be able to change, on the fly, how their genes get used. RNA seems to offer incredibly responsive and versatile ways of doing that.

Philip Ball is a science writer and author based in London. His latest book is *How Life Works* (University of Chicago Press, 2023).

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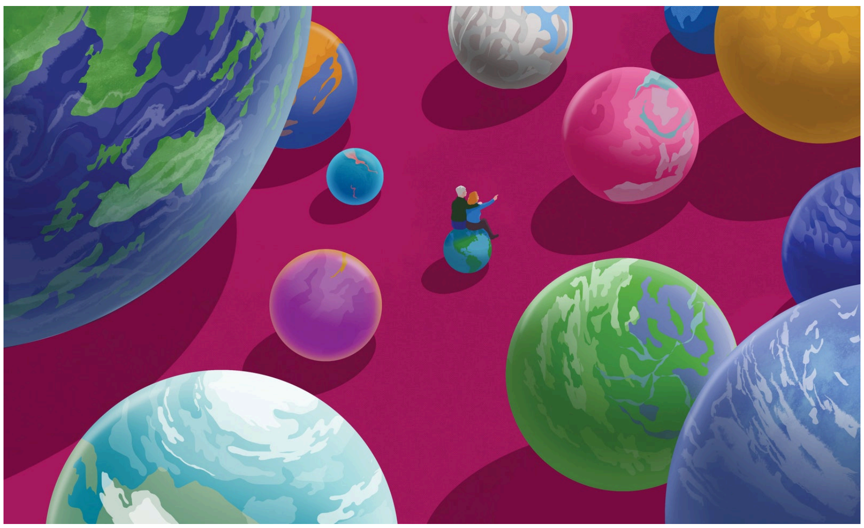
https://www.scientificamerican.com/article/we-thought-dna-ran-our-lives-until-we-discovered-rna-is-in-charge

The Search for Extraterrestrial Life Is a Roller Coaster of Hope and Disappointment

The search for extraterrestrial life has periodically been turned upside down

By Sarah Scoles edited by Clara Moskowitz



Sam Falconer

In the late 1800s Italian astronomer Giovanni Schiaparelli pointed a telescope at Mars and saw something curious: linear features that he called *canali*, meaning “channels” or “grooves.” A mistranslation of that word helped lead to a widespread belief that the planet closest to Earth hosted a civilization.

American astronomer Percival Lowell took Schiaparelli’s observations and ran with them. He became obsessed with the Martian markings, which he interpreted as evidence of a sophisticated network of water-transportation channels. “That Mars is inhabited by beings of some sort or other we may consider as certain as it is uncertain what those beings may be,” Lowell wrote in his 1906 book *Mars and Its Canals.*

It sounds ludicrous now, but it wasn't back then. At the time, ideas about life were evolving rapidly, says David Baron, author of the new book *The Martians: The True Story of an Alien Craze That Captured Turn-of-the-Century America*. In 1858 Charles Darwin published his theory of natural selection. One year later German scientists Robert Wilhelm Bunsen and Gustav Robert Kirchhoff invented the spectroscope, which they and others used to analyze the chemical signatures in light from the sun and the planets. These studies revealed that other worlds are made of the same elemental constituents as Earth. If life evolves by a natural process, and all planets form in similar ways, why wouldn't life take hold on the Red Planet, too?

More than 100 years later scientists searching for extraterrestrial life are guided by the same reasoning: The universe is vast, and it's all made of the same basic stuff we are, so why wouldn't there be life elsewhere? Yet the evidence for intelligent life beyond Earth has taken several turns. In fact, the only constant has been hope: the desire that many people have to prove we are not alone. The question of extraterrestrial life's existence isn't just a neutral scientific debate—it matters to humans, including the humans searching for that life. And our optimism that we'll find it has tended to flip on and off.

The idea that Mars is home to canal-digging civilizations began to lose its sparkle in 1909, when French astronomer Eugène Antoniadi observed the Red Planet during one of its biannual close approaches. The lines, he found with a better telescope and a more intimate view, were an optical illusion. Those data didn't convince Lowell, and it didn't put the theory to rest—in 1916 *Scientific American* managing editor Waldemar Kaempffert was still convinced the canals were real. Nevertheless, belief in advanced life on Mars faded in the following decades. When the Mariner 4 spacecraft flew by Mars in 1965, relaying images of a dry and desolate world, the Martian hypothesis died for good.

And the signs weren't promising for extraterrestrials elsewhere, either. In 1950 physicist Enrico Fermi had pointed out what he called the "Great Silence": If life is likely to be plentiful, then where is everybody? The fact that humanity hadn't heard from other intelligent beings became known as the Fermi paradox. Maybe life is common, but advanced life is rare, scientists suggested. Or perhaps other civilizations arise often and then destroy themselves, as humanity seemed newly capable of doing after the invention of the atomic bomb in 1945.

Astronomers began a more systematic study of the question. In 1960 Cornell University researcher Frank Drake started Project Ozma, which used a radio telescope to scan for broadcasts from two distant star systems. In 1977 astronomers caught a batch of radio waves that blasted out for 72 seconds, looking more like a hugely powerful cosmic radio station than something natural. They called it the WOW! Signal and got excited. But the same transmission was never heard again. So far the search for extraterrestrial intelligence (SETI) has not found convincing evidence of broadcasting aliens.

Yet lately there are new reasons to hope. In 1992 astronomers Aleksander Wolszczan and Dale Frail discovered two rocky worlds circling a dense, rotating star called a pulsar. Although those planets are bombarded with too much radiation to be habitable, more exoplanet discoveries trickled in through the 2000s. Then the Kepler space mission launched in 2009. It revealed thousands of worlds beyond this one, with more than 5,900 total confirmed as of publication time. "Planets became the rule, not the exception," says Nathalie Cabrol, director of the Carl Sagan Center for the Study of Life in the Universe at the SETI Institute.

This wealth of worlds once again changed the calculus on the likelihood of life beyond Earth. Back in 1961 Drake developed a formula to calculate the odds of communicating with extraterrestrial civilizations. It factored in the rate of star formation, the fraction of stars with planets, the fraction of those that are habitable, the proportion of habitable planets that actually develop life, the proportion of that life that becomes intelligent, the fraction of civilizations that develop communications technology, and the length of time they are likely to be transmitting. Most of those variables were unknown at the time—and still are—but the exoplanet boom helped to narrow down the second variable, and it's making headway on the third. We now have a much better idea of how many stars host planets, and it's at least most of them.

We still don't know how life started here on Earth, so we don't know how it might happen elsewhere. And we don't know how likely advanced civilizations are to destroy themselves—a pressing question for reasons beyond SETI. But we do now know that primitive life can thrive in profoundly inhospitable conditions, and that means that microbial aliens may be a lot easier to find than intelligent ones.

In 1966 ecologist Thomas Brock discovered the first extremophile, *Thermus aquaticus,* living in the hot pools of Yellowstone. Since then, scientists have found microscopic organisms in hydrothermal vents at the bottom of the ocean and in toxic mine waste, in the interiors of rocks and in radioactive water. Just because a planet looks barren doesn't necessarily mean that it is. There is good reason to think primitive life could survive in the buried oceans of Jupiter's moon Europa and the geysers of Enceladus, a moon around Saturn. There might even be microbes in the pools of meltwater under the ice caps of Mars. More than a century after Percival Lowell and his illusory Martian civilization, science has given us plenty of reason to think we're not alone, even if aliens turn out to be single-celled organisms rather than canal-building architects.

Editor's Note (8/25/25): This article was edited after posting to correct the dates of Mariner 4's flyby of Mars and Frank Drake's *development of a formula to calculate the odds of communicating with an extraterrestrial civilization.*

Sarah Scoles is a Colorado-based science journalist and a contributing editor at *Scientific American*. Her newest book is *Countdown: The Blinding Future of Nuclear Weapons* (Bold Type Books, 2024).

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https://www.scientificamerican.com/article/in-the-search-for-life-beyond-earth-the-only-constant-is-hope

Humans Aren’t as Special as We Once Thought

Other species exhibit capabilities that were once thought to be exclusive to Homo sapiens

By Kate Wong edited by Seth Fletcher

Sam Falconer

It was the telegram exchange that sparked an identity crisis for humankind. In 1960 a young Jane Goodall working in a remote forest in Tanzania observed a chimpanzee she named David Greybeard using blades of grass and twigs to fish nutritious termites out of their nest. The primatologist wrote to her mentor, Kenyan paleoanthropologist Louis Leakey, to tell him about her observation, which flew in the face of the conventional wisdom that held that only humans made tools. Leakey replied: “Now we must redefine tool, redefine man, or accept chimpanzees as human.”

For decades—centuries, even—scholars have attempted to draw a hard line between our kind and the other organisms with whom we share the planet. They have argued that only humans have culture—sets of learned behaviors, such as toolmaking, that are passed down from generation to generation. They have proposed that only humans think symbolically, using signs to represent objects or ideas. That our species alone is self-aware, capable of planning for the future and experiencing emotions such as joy and fear, love and grief. That only humans are *conscious*, possessed of an inner world of subjective experience.

For his part, Charles Darwin, writing in the late 1800s, opined that nonhuman animals have the same cognitive abilities and emotions that humans have and that any differences were a matter of degree and not kind. In the absence of any way to reliably read animal minds, however, scientists who studied animal behavior and cognition took the position that ascribing human thoughts, feelings and motivations to animals—anthropomorphism—was a cardinal sin. But in recent decades examples of other species demonstrating these capabilities have emerged from across the tree of life. The findings have spurred fresh thinking about what, exactly, distinguishes *Homo sapiens*, with our vaunted intellect, from every other species on Earth.

Let's look first at our evolutionary nearest and dearest. We *H. sapiens* possess much larger brains than our closest living relatives, the chimps and bonobos, do—around three times as large. The brain requires 20 percent of our energy budget despite making up only 2 percent of our body mass. Naturally, anthropologists have wondered why we evolved such energetically expensive brains. At the same time, we know that *H. sapiens* is the sole surviving member of what was once a diverse group of humanoids. Surely our big brains and all the clever things they allow us to do were a major reason for our success as a species, a vital factor in why we alone went on to spread across the globe and thrive in every ecosystem we set our sights on, outcompeting other branches of humanity until we were the last hominin standing.

Yet virtually every trait that anthropologists have identified as one that might have set our kind apart has subsequently been found in another member of the family. Our closest evolutionary cousins, the Neandertals, left behind decorations that suggest they used symbols, which may indicate a capacity for language. The same goes for our smaller-brained relative *Homo erectus*. And some 3.3 million years ago, long before brain size began to expand in our lineage, an unknown hominin—possibly *Australopithecus afarensis*—shaped basalt cobbles into cutting tools, demonstrating an understanding of the material properties of stone and a vision for how to transform a lump of rock into a useful implement.

It's not just our closest hominin and great ape relatives that share our powers of cognition. Humans were long thought to be the only moral animals, uniquely equipped with a sense of right and wrong. But we now know that is not the case. The late primatologist Frans de Waal and Sarah F. Brosnan found in laboratory experiments that brown capuchin monkeys would decline a reward of a slice of cucumber if they observed another monkey receiving a better treat (a grape) for the same task. The monkeys' rejection of unequal payment for equivalent work demonstrated that they have a sense of fairness and experience moral outrage when they get a raw deal.

Other animals exhibit other elements of morality—including empathy. Mice, for instance, can share the emotional state of another individual, exhibiting increased sensitivity to pain if they see a companion showing signs of pain. Dogs recognize distress in their owners and will offer consolation. Rats will sacrifice their own gains to alleviate the suffering of a conspecific, forgoing a food reward if taking the food means inflicting pain on another rat.

Empathy and other complex emotions were long considered beyond the experience of nonhuman creatures. But mounting evidence indicates that they are widespread among mammals. Some of the most striking examples involve emotional responses to death. In 2018 an orca known as Tahlequah made headlines around the world when she carried her dead calf with her for 17 days while she swam 1,000 miles across the Salish Sea. In 2024 Tahlequah lost another calf. This time she held on to its corpse for at least 11 days before releasing it. Researchers characterized the mother orca's reaction to these losses as grief.

Apes, monkeys and elephants have been observed to mourn the loss of bonded individuals, too. It's not just large-brained mammals that appear to express sorrow, however. Barbara King, who is known for her research and writing on animal cognition and emotion, has described compelling examples of grief in peccaries, donkeys and ferrets, among others.

Our fellow mammals are not the only animals to show signs of thinking and feeling as humans do. The Eurasian Magpie, a species of bird, can recognize itself in the mirror—a sign of consciousness. Fish feel pain, another conscious experience: when given an injection that causes discomfort, lab zebra fish will vacate their preferred habitat, which has been decorated with pleasing rocks and vegetation, to visit a barren habitat whose water is infused with a pain reliever. And studies of bees and other insects suggest that they may experience both pain and joy. That is, these creatures, too, may be sentient.

Although the evidence for consciousness in fish, reptiles, insects, and other invertebrates has yet to accumulate to the degree that it has in mammals and birds, researchers are taking the possibility far more seriously than they have in the past. In 2024 dozens of scientists signed a declaration acknowledging that species quite different from humans may have experiences of consciousness and that this possibility needs to be considered in decisions that affect these animals. The document could help shape policies governing animal research ethics and welfare.

One more group of organisms deserves mention. In recent years researchers have increasingly begun to explore the idea that plants—traditionally viewed as noncognitive beings—can learn, remember, make decisions, communicate and experience the world uniquely. In this way, some investigators propose, they are conscious. Consider the Venus flytrap, a carnivorous plant that catches flies, ants, and other insects when they brush against the sensory hairlike structures in the plant's trap. The plant remembers when it has been touched. After two touches, the trap closes and imprisons the insect prey; after five touches, it produces the enzymes needed to digest its catch. Other plants sense when they are being munched by hungry insects and emit chemical signals that summon predators of their attackers.

We might not be as unique as we thought we were. But we needn't feel demoted. There's something marvelous about finding a common thread between flytrap and ferret, bee and human. We're not separate from nature, we're connected to it, part of the weave of life, in all its dazzling diversity.

Kate Wong is an award-winning science writer and senior editor at *Scientific American* focused on evolution, ecology, anthropology, archaeology, paleontology and animal behavior. She is fascinated by human origins, which she has covered for more than 25 years. Recently she has become obsessed with birds. Her reporting has taken her to caves in France and Croatia that Neandertals once called home, to the shores of Kenya's Lake Turkana in search of the oldest stone tools in the world, to Madagascar on an expedition to unearth ancient mammals and dinosaurs, to the icy waters of Antarctica, where humpback whales feast on krill, and on a "Big Day" race around the state of Connecticut to find as many bird species as possible in 24 hours. Kate is co-author, with Donald Johanson, of *Lucy's Legacy: The Quest for Human Origins*. She holds a bachelor of science degree in biological anthropology and zoology from the University of Michigan. Follow Kate on Bluesky.

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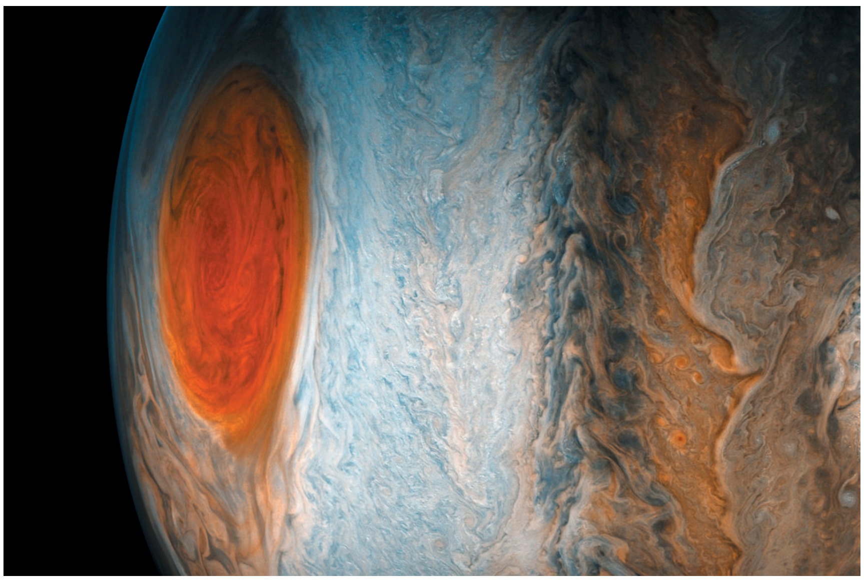
https://www.scientificamerican.com/article/human-uniqueness-is-a-myth-mounting-evidence-shows

NASA’s Juno Mission Leaves Stunning Legacy of Science at Jupiter

The Juno spacecraft has rewritten the story on Jupiter, the solar system’s undisputed heavyweight

By Robin George Andrews edited by Clara Moskowitz



Jupiter's Great Red Spot glows in this image created from Juno observations.
NASA/JPL-Caltech/SwRI/MSSS/Gerald Eichstadt/Sean Doran © CC NC SA

The NASA spacecraft tasked with uncovering the secrets of Jupiter, king of the planets, is running out of time. The Juno probe has already survived far longer than anticipated—its path around the solar system’s largest planet has repeatedly flown it through a tempest of radiation that should have corroded away its instruments and electronics long ago. And yet here it is: one of the greatest planetary detectives ever built, still pirouetting around Jupiter, fully functional.

But it may not be for long. September 2025 marks the end of Juno’s extended mission. Although it could get another reprieve—an extended-extended mission—the spacecraft cannot carry on forever. Eventually the probe is fated to plunge into Jupiter’s stormy skies, to lethal effect. Regardless of when that happens, the spacecraft's legacy is indelible.

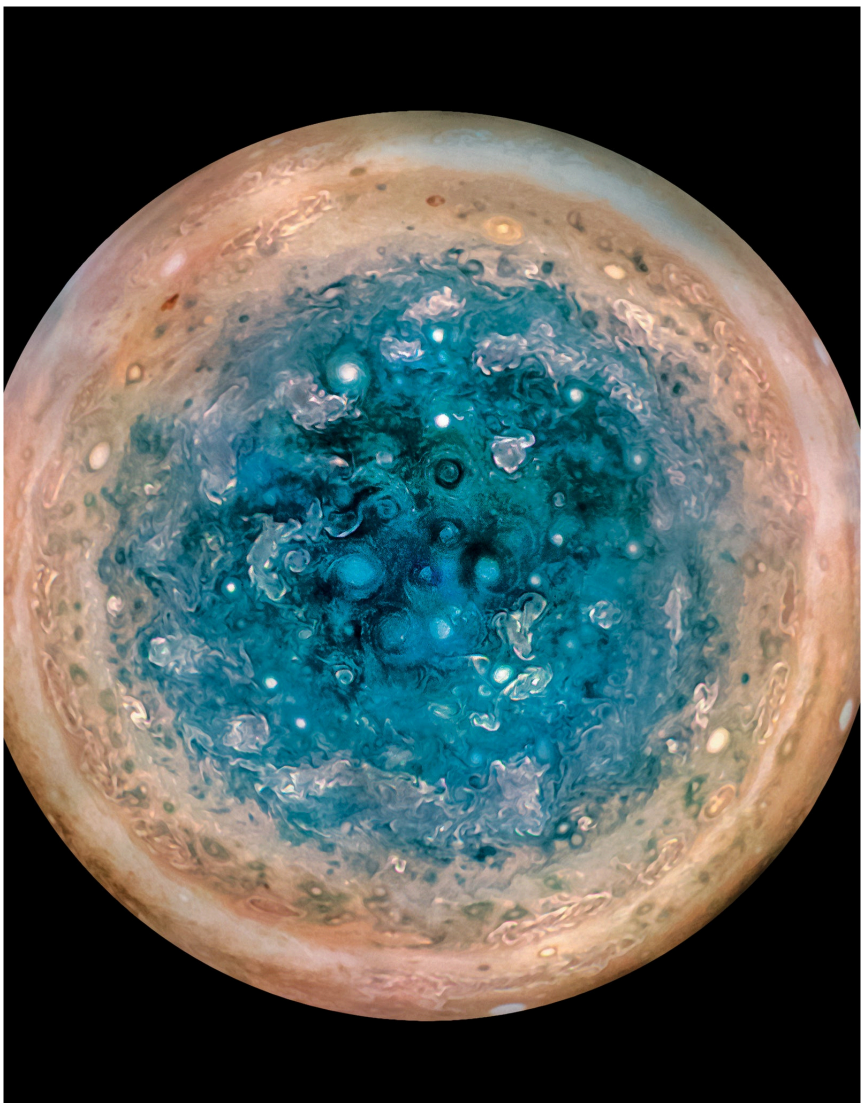
It revealed a whole different Jupiter than scientists thought they knew. Oddly geometric continent-size storms, in strange yet stable configurations, dance around its poles. Its heaviest matter seems to linger in its skies, while its abyssal heart is surprisingly light and fuzzy. Its innards don't resemble the lasagnalike layers found in rocky worlds; they look more like mingling swirls of different kinds of ink.

And Juno wasn't simply trying to understand Jupiter. It set out to uncover how the entire solar system was born. Jupiter, after all, was the first planet to piece itself together after the sun exploded into existence. Hidden underneath the planet's cloud tops, there is a recording of the beginnings of everything we see around us. "That's the story behind why Juno was created: to go and look inside Jupiter every way we knew how, to try to figure out what happened in the early solar system that formed that planet—and what role that planet had in forming us," says Scott Bolton, the mission's principal investigator at the Southwest Research Institute in San Antonio, Tex.

Whenever a mission studies a planet or moon up close, "you're going to be surprised" at what it finds, says Juno project scientist Steve Levin of NASA's Jet Propulsion Laboratory. But what you really want is "to make the theorists throw everything out the window and start over." Juno has torn up more textbooks than any other planetary science mission. "It's been quite a ride," Levin says. And scientists will never look at Jupiter, or the solar system, in the same way again.

Jupiter, the Roman god, was often up to no good. According to myth, he obscured his mischief with a blanket of clouds so that nobody could see what he was up to. His wife, though, had the power to see through these clouds and monitor his shenanigans. Her name was Juno.

In the late 1970s the two Voyager space probes gave humanity its first spectacularly detailed look at the gas giant. Unlike the deific Juno, they couldn't see Jupiter's buried secrets—but they were sufficiently inspiring for Bolton, who was a college student at the time. "I had been a huge *Star Trek* fan and had fantasized about traveling around and wondering what the rest of the universe was like," he says. When someone from JPL gave a talk at his school and showcased Voyager 1's jaw-dropping shots of Jupiter and its maelstroms, he was sold. "I'd never seen anything like it."



Juno spotted numerous oval cyclones on Jupiter's southern pole.
NASA/JPL-Caltech/SwRI/MSSS (*image data*); Betsy Asher Hall and Gervasio Robles (*image processing*)

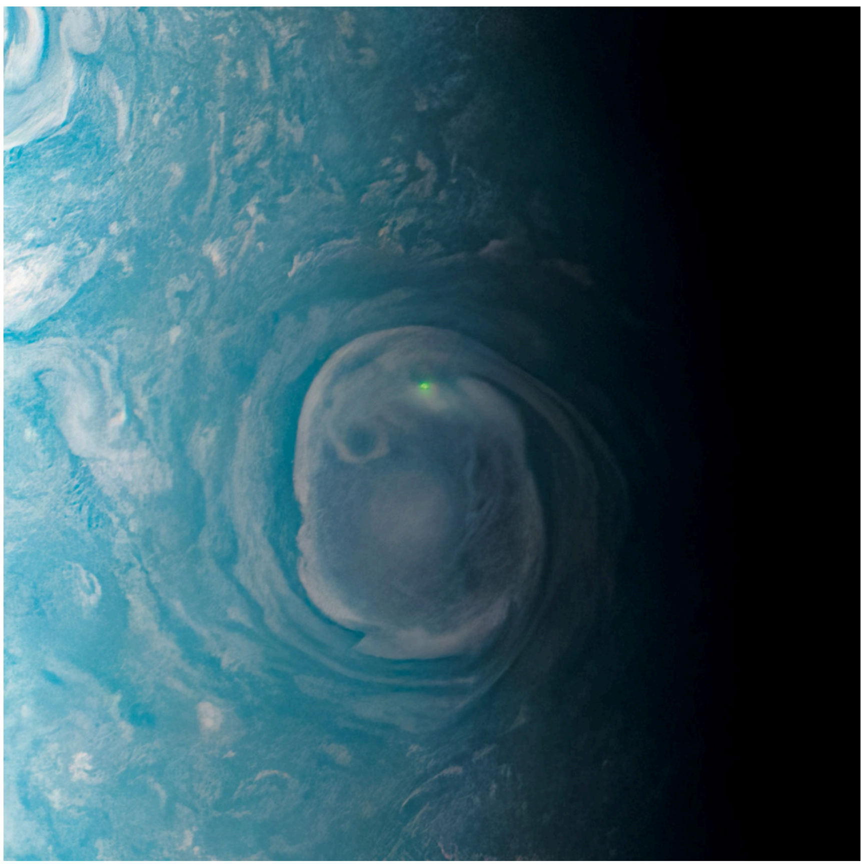
In 1980 Bolton got a job at JPL, just as Voyager 1 was about to greet Saturn. Later he became part of the Galileo project, a mission to study Jupiter's atmosphere and magnetic field that orbited the planet from 1995 to 2003. It was the first spacecraft to orbit an outer planet and the first to drop a probe through its atmosphere. Although Galileo began to paint a picture of Jupiter in three dimensions, so much about the world—especially its core, the depth and nature of its storms, and its unseen polar regions—remained a mystery.

Bolton ultimately came to an inescapable conclusion: science needed to make the mythical Juno real. As the new millennium dawned a spacecraft took shape, to the tune of $1.1 billion. A triumvirate of solar panels powered a suite of cloud-piercing instruments, some able to pick up on different types of radiation emanating from deep within the planet. One piece of tech can measure how the spacecraft is affected by small changes in the planet's gravitational field, allowing scientists to determine Jupiter's inner structure.

Because every bit of added weight counts for a lot in spaceflight, the earliest Juno plans lacked a visual camera. It didn't need one to achieve its scientific objectives. But Candice Hansen-Koharcheck, a Juno team member and a senior scientist at the Planetary Science Institute in Tucson, Ariz., recalls Bolton saying: "We can't fly to Jupiter without a camera." The mission may be all about sensing what's below those clouds. But who doesn't want to catch a glimpse of alien hurricanes and vaporous whirlpools, too? JunoCam, led by Hansen-Koharcheck, was added to the payload.

The biggest issue mission designers faced was figuring out how to shield the probe. The space environment enveloping Jupiter is thoroughly unpleasant. A torus of radiation, not only deadly to humans but also highly degrading to any electronics, zips around the planet's equator. Eventually this radiation will murder any spacecraft in its wake. To delay the inevitable, Juno deploys two radiation-dodging tricks.

The first is to orbit in a way that repeatedly takes it over Jupiter's poles, where radiation is minimal. During each circuit, Juno gets as close as 3,100 miles to the planet's cloud tops, allowing it to conduct detailed scientific observations while spending a limited time bathed in aggressive radiation. The second is that its most vital electronics are encased inside a titanium vault. The spacecraft's hull is showered by more than 100 million dental x-rays' worth of radiation. Anything inside the vault receives about 800 times less.



The glow from a bolt of lightning is clear in this image of a vortex on Jupiter's northern pole. Juno took the picture during a close flyby of the planet in December 2020.
NASA/JPL-Caltech/SwRI/MSSS (*image data*); Kevin M. Gill © CC BY (*image processing*)

Juno's mission team hoped these strategies would keep the spacecraft alive for at least a year, but the scientists had only educated guesses to work with. "No one's ever done a polar orbit. No one's ever slipped between the radiation belts," says Heidi

Becker, a researcher at JPL and the member of the Juno team responsible for monitoring the radiation environment.

The only way to know was to go. “I’ve been looking up at Jupiter for a very long time,” Becker says. She felt like the planet was teasing the Juno team before launch: “Okay, bring it. Let’s see if you can do it.”

Juno left Earth in 2011 and reached Jupiter after a 1.7-billion-mile journey. It quickly took up a polar orbit of the elephantine world, and Becker and the team were overwhelmingly relieved when they realized that the radiation hadn’t immediately exterminated the spacecraft.

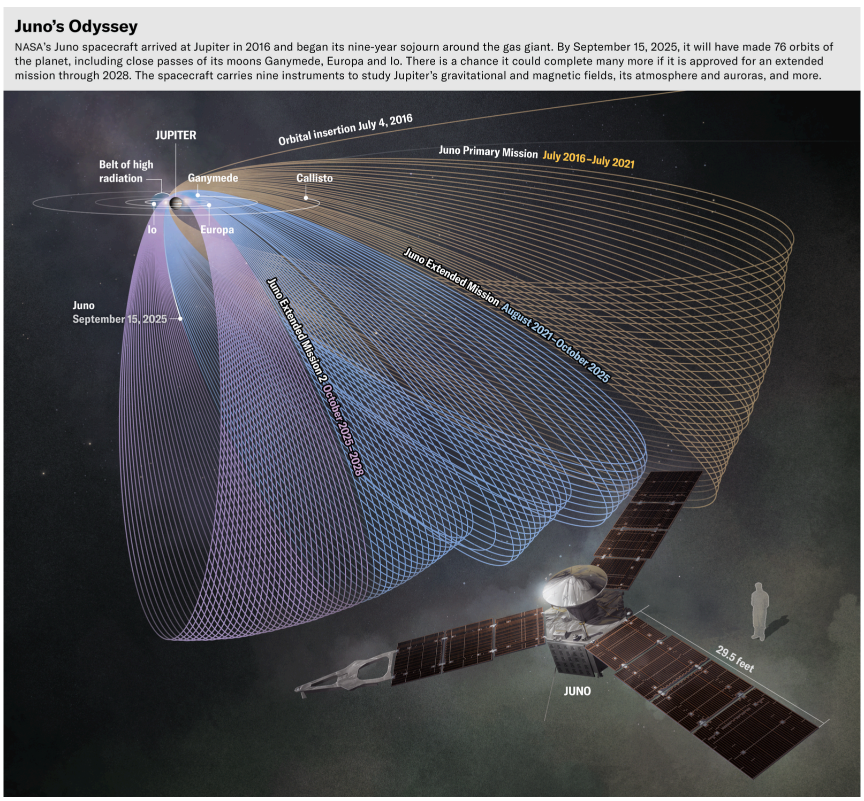
The scientists were also glad they’d packed that camera. The moment Juno opened its eyes, it witnessed a parade of colors rushing about with unrelenting force. The ever-changing landscapes weren’t just painterly. “They’re like works of art,” says Bolton—impressionistic-looking spirals and streams, folding, arching and blooming in full view. Juno may be a scientific mission, but it also revealed Jupiter as a living van Gogh painting hanging in the sky.

Within moments of falling into orbit, Juno revealed wonders—starting with the planet’s freakish atmosphere and its gargantuan storms. When the probe peeked at Jupiter’s poles, “we saw something nobody’s seen before,” Levin says. JunoCam and Juno’s infrared mapping instrument, JIRAM, spied an octagonal collection of eight storms surrounding a central cyclone at the north pole. The south pole, meanwhile, had a pentagonal group of five storms circling another one in the middle. Each cluster of cyclones is larger than the U.S.

The JIRAM image of the northern circumpolar cyclones resembled a “beautiful, gigantic jack-o’-lantern in space,” Becker says. These geometric storms didn’t just look striking—they had no precedent.

“The first time we saw the storms, I was with a bunch of people from the science team,” Levin says. “Somebody literally said: ‘Are you sure you got the right planet?’ And they were only half joking.”

The arrangement at each pole seemed oddly stable: storms moved around and jostled one another, but none disappeared. And to date, no one has a definitive explanation for why the number of storms at each pole differs, nor why their dance routine never seems to change. “The way those cyclones are stable at the poles is still a mystery,” says Alessandro Mura, a researcher at the National Institute for Astrophysics in Rome and the lead for Juno’s infrared mapping instrument.



Matthew Twombly; Source: Scott Bolton (*Juno orbit reference and expert review*)

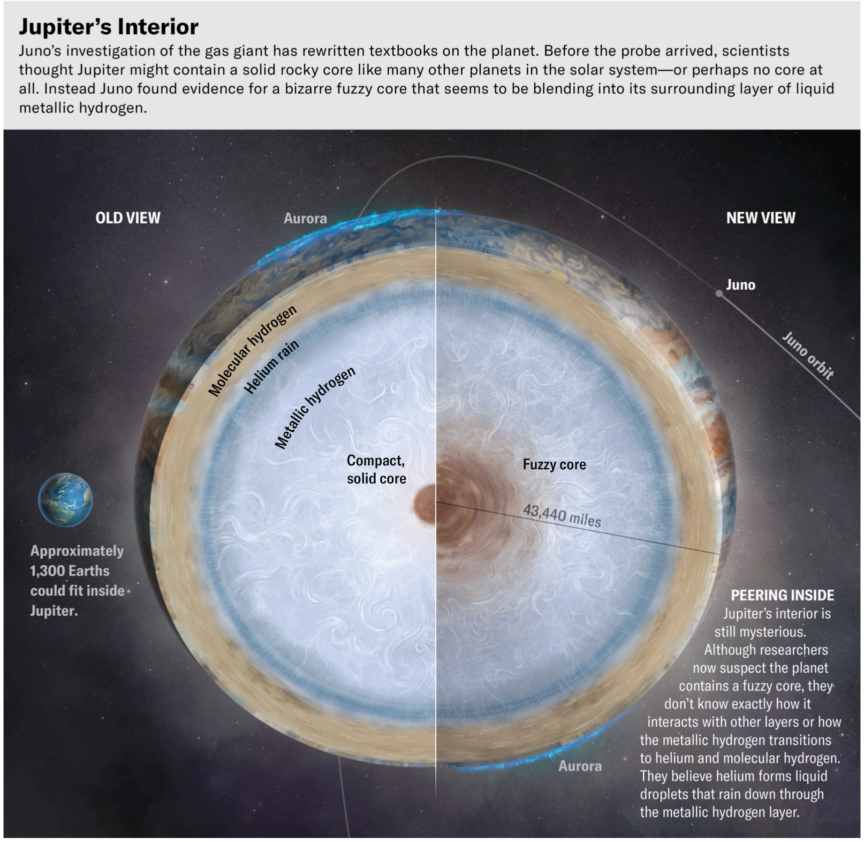
The most famous storm on Jupiter is its Great Red Spot—a rust-hued monster large enough to encompass the entire Earth. First seen a couple of centuries ago, it’s known to change shape over time, and one day it may vanish. But until Juno arrived, astronomers’ knowledge of it was surficial. By probing the radiation emitted by the spot’s churning gases and by measuring its gravitational pull, the Juno team realized it reached a depth of about 300 miles below the cloud tops—almost 55 times deeper than Mount Everest is tall.

Unsurprisingly, for a planet wreathed in storms, Jupiter experiences a lot of lightning; the Voyager missions caught bolts flashing through its clouds back in 1979. But Juno "discovered a type of lightning that doesn't exist on Earth," Becker says, which seemingly defied the laws of physics.

Like many spacecraft, Juno has a star camera, an instrument that uses those diamantine dots to determine its orientation in space and aid its navigation. The camera can also spot lightning, which appears as bright specks. When Juno looked at the dark side of Jupiter, it spied tiny little flashes made by very high-altitude lightning bolts.

That didn't make any sense. To produce lightning, liquid water needs to collide with ice crystals to create a spark. In 1979 the Voyager mission detected lightning coming from deep water clouds, where the suffocating pressure of the overlying atmosphere created temperatures high enough for liquid water to exist. But the lightning flashes picked up by Juno came from the upper echelons of Jupiter's atmosphere, a location so frigid that only ice crystals should exist there.

After studying Jupiter's titanic clouds for a time, the Juno team worked out what was happening. The planet's cloud tops contain plenty of ammonia, and storms can launch ice into the sky that then binds to that ammonia. The chemical acts like antifreeze on the water-ice, causing it to turn into liquid droplets. And when those droplets smash into the upwardly propelled ice crystals, you get electricity—and vertiginous lightning.



Matthew Twombly

But this epiphany brought another mystery into focus. Sure, ammonia-ice clouds likely dominate Jupiter's skyline—but Juno found that some parts of the uppermost atmosphere have a dearth of ammonia. That didn't track: Jupiter's atmosphere looks incredibly turbulent—like a thoroughly whisked raw egg—so all its components should be mixed up, with a more or less even distribution of gases. How can many parts of the planet have 90-mile-deep wells lacking ammonia?

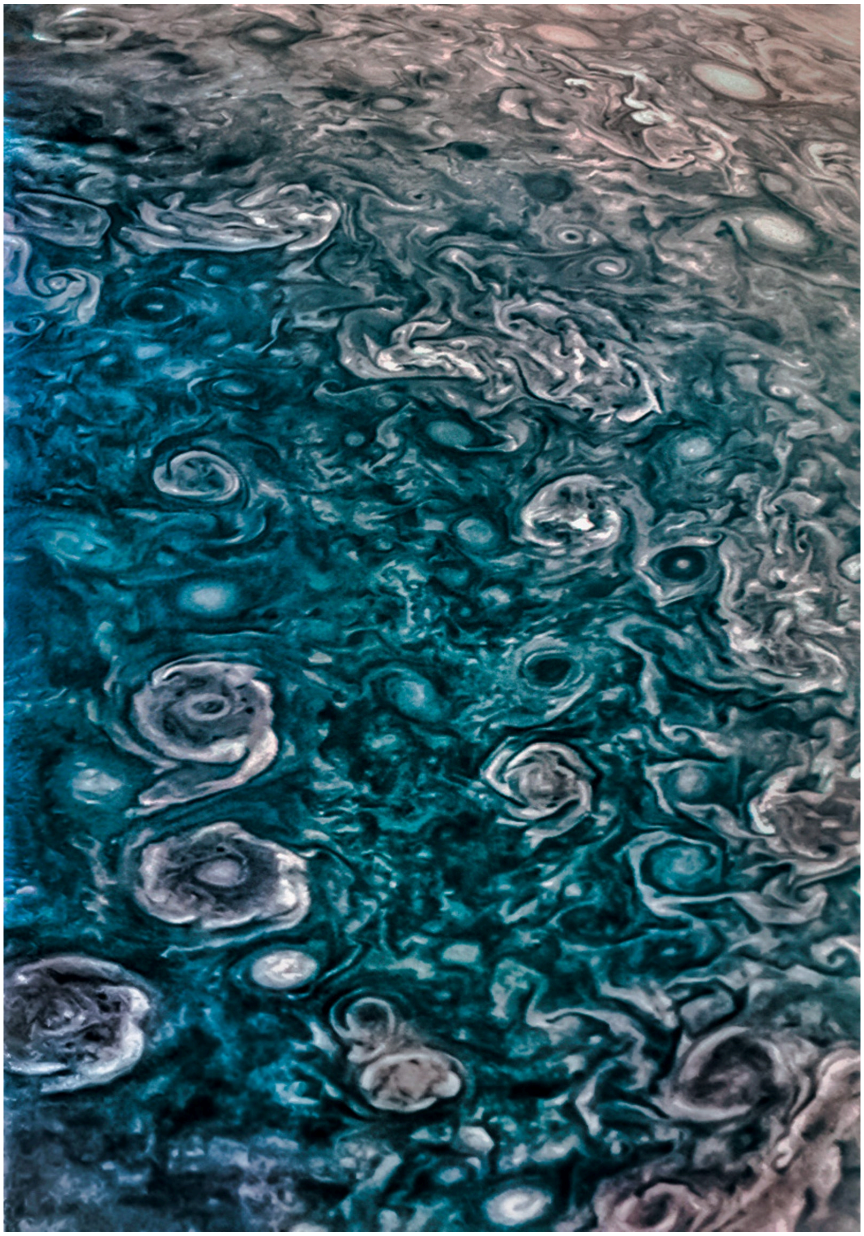
"There was no theory that could even remotely explain this," says Chris Moeckel, a planetary scientist at the University of California, Berkeley. His first thought was that "there's no way this is right." But the data were sound.

A complicated idea arose to make sense of the phenomenon. When the sky-high ammonia turns upwelling water-ice into liquid, the water and ammonia bond to form a peculiar slush with a water-ice shell. Ultimately softball-size globules of slush encased in ice fall back into the planet, where they melt at depths thought to be too extreme for Juno's instruments to detect.

For a few years this theory seemed a bit too baroque to be true. But Moeckel and his colleagues became convinced thanks to the power of Juno's microwave radiometer. The instrument can measure radio waves that betray the presence of different chemical compounds. During one of its orbits, Juno noted a burst of ammonia production at an exceptional depth within the planet. According to Moeckel, this was a telltale sign that icy orbs had rained down from the sky and thawed, releasing their trapped water-ammonia slush. Researchers referred to this unique weather phenomenon as mushballs. "It's such a stupid name," Moeckel says. "But it works."

Juno also trained its instruments on Jupiter's magnetic field, the largest structure in the solar system, which reaches at least as far as its neighboring planet, Saturn. But Juno discovered that Jupiter's magnetic field is wonky and asymmetric—more messy in the northern hemisphere than the south. There is also an intense concentration of magnetism near the equator, a patch (confusingly) called the Great Blue Spot.

These characteristics are odd, but the existence of such a gargantuan field at all is the really strange part because Jupiter lacks the sloshing liquid iron and nickel responsible for Earth's magnetic field. Instead Jupiter contains an ocean of hydrogen, one under so much pressure that electrons are torn off individual hydrogen atoms, transforming it into an exotic, metal-like electrical fluid that generates its mighty magnetic field.



Violent storms swirl across Jupiter's northern pole. The storms are mysteriously stable over time, and each cluster of cyclones is larger than the continental U.S.
NASA/JPL-Caltech/SwRI/MSSS (*image data*); Emma Wälimäki © CC BY (*image processing*)

Below the hydrogen sea lies an even bigger mystery—the question of what's inside the planet's innermost core. What Juno found there left scientists reeling.

Before the spacecraft arrived, there were two prevailing notions about Jupiter's interior. The first was that the planet may have a compact core of rocky and metallic matter, not dissimilar to the cores of other worlds. If such a core exists, then Jupiter likely formed through the gradual clumping together of gas and solid matter, like the planets of the inner solar system. The second hypothesis was that there is no core at all. Instead Juno might find a ball of hypercompressed gas, suggesting Jupiter's formation was a bit like a failed star, one that didn't gather enough gas to trigger a thermonuclear ignition.

"Actually neither of those was true," Bolton says. Juno used gravitational detective work to sense the core. The spacecraft is constantly communicating with Earth using radio waves. Jupiter's uneven mass means that Juno speeds up at times and slows down at others, depending on the strength of the gravitational pull it's experiencing. These speed changes cause subtle shifts in the wavelengths of the radio transmissions Juno sends and receives—effects that scientists can use to determine the internal structure of Jupiter.

What they found was at first nonsensical. Deep within the metallic hydrogen ocean Juno detected an innermost core of, well, *something*; it's probably solid, but researchers can't tell. "It's blending gradually into the surrounding layers," says Ryan Park, a researcher at JPL and one of the leads on the gravity experiment on Juno. The hydrogen and the core material seem to mingle. The situation is very different from Earth's depths, where a lighter rocky mantle floats atop a denser iron and nickel core, between which is a distinct and definitive boundary. "We frankly don't know how to explain that," Levin says. And it gets weirder still.

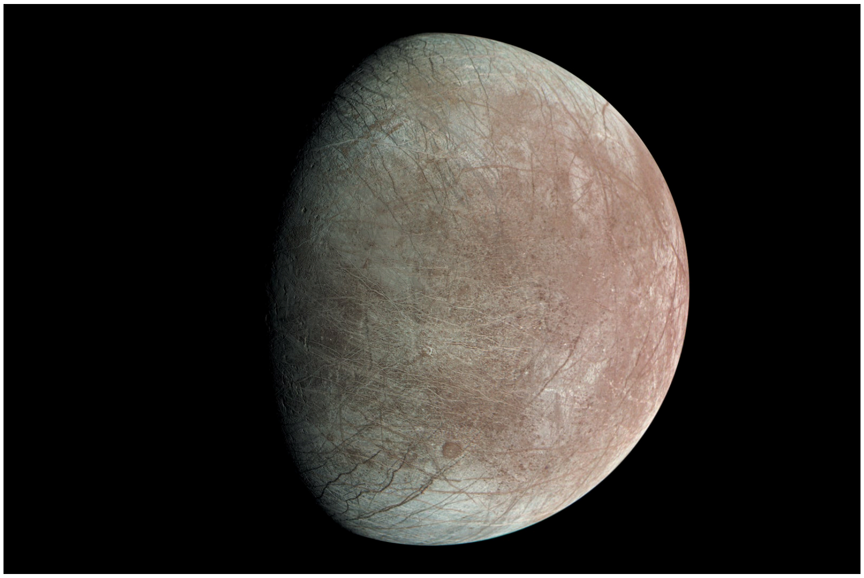
The sun and Jupiter are rich in both hydrogen and helium but are also expected to contain a smattering of heavier elements. Jupiter, a huge planet that most likely ate up rocky and icy planet-size shards during its formation, should contain far more heavy elements than the sun. And indeed, Juno found that Jupiter has three to four times as many heavy elements as our star. The problem, though, is that these elements appear to be found in the upper atmosphere—and the innermost core is comparatively lacking. All that heavy stuff should sink, via gravity, into the core. But apparently it hasn't. If the core is so light, then what could it possibly be made of?

Scientists are scrambling for answers. This fuzzy core doesn't fit with anyone's model for planetary formation. Some scientists have suggested a giant meteor crashed into a once solid core, smashing it up and forcefully mixing it with the metallic hydrogen ocean. Levin wonders whether we simply don't understand the physics yet. "We're talking about temperatures and pressures much higher than anything we're used to," he says—conditions so severe that it's difficult to create them in laboratories.

Other blockbuster findings from Juno concern Jupiter's moons. The probe's reconnaissance of two icy orbs—the pockmarked Ganymede and the ocean-concealing Europa (the target of a recently launched NASA habitability mission)—created breathtaking portraits of these dynamic worlds while also revealing some unusual chemistries. But a moon named Io got most of Juno's attention—and, consequently, generated the most shocking surprise.

"Io is a very peculiar moon because it's the most volcanic body of all," Mura says. Its surface, an amalgam of burnt orange, sickly yellow and crimson hues, is covered in rocky cauldrons filled with lava, as well as volcanoes whose explosions propel magmatic matter into space. Up there the material is ionized by sunlight before plunging into Jupiter's skies, creating extremely bright auroral lights.

Since the 1970s scientists have understood that Io's volcanism is powered by its elliptical orbit around Jupiter. When it's closer to Jupiter, it gets a bigger pull from the planet's gravity; when it's farther away, that pull is weaker. This back-and-forth kneads the moon like putty, creating tides in solid rock more than 300 feet high. All that motion creates a lot of friction, an abundance of heat—and a plethora of magma.



Juno made close flybys of three of Jupiter's moons *(top to bottom)*: Io, Ganymede and Europa. Io is the most active volcanic world in the solar system and features the largest volcanic eruption ever recorded. Ganymede is a pockmarked place much like our moon, and Europa boasts a hidden ocean under its crust.
NASA/JPL-Caltech/SwRI/MSSS (*image data*); Emma Wälimäki © CC BY (*image processing) (Io),* NASA/JPL-Caltech/SwRI/MSSS *(Ganymede),* NASA/JPL-Caltech/SwRI/MSSS (*image data*); Björn Jónsson CC BY 3.0 *(image processing) (Europa)*

Many thought that this mechanism, known as tidal heating, was so powerful that it created a continuous ocean of magma under the surface rather than the smaller, individual magma reservoirs that fuel Earth’s volcanoes. The Galileo mission seemed to back that idea up: it detected an electrically conductive layer under Io’s crust suggestive of a magma sea.

But when Juno flew perilously close to Io on two occasions, getting within 900 miles of the violent surface, it found no trace of a shallow magma ocean. Mura now suspects Io’s magma is partitioned into a maze of rocky tunnels, occasionally bubbling up into open rocky maws wherever the tunnels reach the surface.

Nobody knows for sure; in typical Juno style, the observations have raised more questions than answers. But at least while scientists ponder possible solutions, they can marvel at Io’s unbound ferocity.

“We discovered the largest eruption ever recorded,” Bolton says. In December 2024 Juno’s infrared instrument detected a heat spike in the moon’s southern hemisphere that briefly blinded the spacecraft's JIRAM instrument: a paroxysmal outpouring of lava spread over 40,000 square miles, enough to cover a quarter of California. It's producing more energy than the total annual energy output of humanity. "And we still see it going on," Bolton adds.

By all accounts, Juno should be dead by now. The radiation should have already broken it or at least one of its instruments. Somehow it lasted well beyond its prime mission timeline, which ended in 2021.

If an additional three-year extension is approved, Juno could get a better look at the planet's ghostly ring system, and some of its lesser-known innermost moons. But there's no telling how long the aging spacecraft could survive. "It could grow old, and something could fail," Bolton says. Perhaps "the radiation will kill something so important that we can't function anymore." Whenever the vehicle's end comes, it will go out in flames, spiraling toward the gas giant it spent its entire life interrogating. "Eventually Juno will crash into Jupiter on its own," Bolton says.

But the spacecraft's legacy is already clear. Juno revealed Jupiter to be a far more confounding place than anyone dared imagine, forcing scientists to throw out reams of outdated ideas about planetary formation. It's also revealed how future spaceflight missions can defend themselves from the worst radiation in the solar system. The Juno team, having emulated its namesake's god-defying powers, is openly proud, Becker says. "What an amazing success story for NASA."

Robin George Andrews is a volcanologist and science writer based in London. His most recent book is *How To Kill An Asteroid* (W.W. Norton, 2024). Follow him on X @SquigglyVolcano

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https://www.scientificamerican.com/article/how-nasas-juno-probe-changed-everything-we-know-about-jupiter

How Your Brain's Nightly Cleanse Keeps It Healthy

Washing waste from the brain is an essential function of sleep—and it could help ward off dementia

By Lydia Denworth edited by Madhusree Mukerjee

Miriam Martincic

You can see it coming in right there, that little spot," says neuroscientist and engineer Laura Lewis.

A remarkably bright pulsing dot has appeared on the monitor in front of us. We are watching, in real time, the brain activity of a graduate student named Nick, who is having an afternoon nap inside an imaging machine at the Massachusetts Institute of Technology, where Lewis has her laboratory.

The bright spot first appears toward the bottom of the screen, about where Nick's throat meets his jaw. It moves slowly upward, fades and then is followed by another bright dot. "It really comes and goes," says Lewis, who is also affiliated with Massachusetts General Hospital. "It's in waves." This moving dot depicts something few people have ever seen: fresh cerebrospinal fluid flowing from the spinal cord into the brain, part of a process that researchers are now learning is vital for keeping us healthy.

For decades biologists have pondered a basic problem. As human brains whir and wonder throughout the day, they generate waste—excess proteins and other molecules that can be toxic if not removed. Among those proteins are amyloid beta and tau, key drivers of Alzheimer’s disease. Until recently, it was entirely unclear how the brain takes out this potentially neurotoxic trash.

In the rest of the body, garbage removal is handled initially by the lymphatic system. Excess fluid and the waste it carries move from tissue into the spleen, lymph nodes and other parts of the system, where certain particles are removed and put into the bloodstream to be excreted. It was long thought that the brain can’t use the same trick, because the so-called blood-brain barrier, a protective border that keeps infections from reaching critical neural circuitry, stops the transport of most everything in and out.

In 2012 researchers at the University of Rochester led by neuroscientist Maiken Nedergaard made a pivotal discovery: a previously unknown circulatory system was flushing toxic waste from the brain. In mice, they showed that an influx of cerebrospinal fluid (CSF) washes through the brain’s “perivascular” spaces, which are doughnut-shaped tunnels that surround blood vessels. Using water channels on the surface of astrocytes, a type of cell that supports brain function, the CSF mixes with “interstitial” fluid in the spaces around the brain’s cells and collects built-up waste. Then the fluid leaves the brain through the perivascular spaces around veins, taking the garbage with it.

Nedergaard and her team called their discovery the glymphatic system—“g” for glial cells, of which astrocytes are a subtype, and “lymphatic” to reference the waste-clearance function. The next year, in 2013, they published an important additional finding: this housekeeping was most active and efficient during sleep. “Wakefulness clearly shut it down,” Nedergaard says—probably because the precision that neural networks need to process the external world when awake isn’t compatible with the clean-up process. That finding suggests this newly discovered brain-washing process is one of the critical functions of sleep. “Sleep is clearly for the brain,” Nedergaard says. “When you wake up refreshed after good sleep, it is probably because your brain had a tune-up similar to your car.”

But this groundbreaking work was done in mice, and mice are not people. Their brains are smaller and less complex than ours, their sleep far more fragmented. In part because of that discrepancy, the glymphatic hypothesis has had plenty of naysayers. “Ten years ago all this flow in the brain, it was almost like heresy,” says neuroimmunologist Jonathan Kipnis of the Washington University School of Medicine in St. Louis.

Hundreds of studies have since been done—and that bright dot Lewis showed me represents a critical next phase of investigation. She and others have spent much of the past decade exploring whether this waste-clearance process works in humans as it does in rodents. The short answer seems to be yes. Furthermore, the electrical waves that sweep through the brain during sleep, helping to sort, select, transport and store memories, seem to have another significant function: they also propel cerebrospinal fluid in and out of the brain.

The significance of the glymphatic system is considerable. If waste clearance is an essential function of human sleep, then a dysfunction in this system probably relates to many neurological and psychiatric disorders, including Alzheimer’s. Glymphatic impairment could explain why the aging brain accumulates the amyloid plaques and tau tangles that trigger Alzheimer’s—and there is some evidence that conditions such as traumatic brain injury, which is associated with Alzheimer’s, interfere with waste clearance. “If it’s the thing that holds all those things together,” says Jeffrey Iliff, a professor of psychiatry and neurology at the University of Washington School of Medicine who worked with Nedergaard on the original studies, “well then if you target it, that opens the door to primary prevention of neurodegenerative diseases.”

Although it’s been clear for years that accumulation of amyloid and tau proteins leads to Alzheimer’s, the link between sleep and the waste-clearance process that might get rid of them was not obvious. For decades sleep researchers focused primarily on sleep’s role in memory processing. For their part, biologists who studied the blood-brain barrier knew there were perivascular spaces surrounding blood vessels like loose sheaths, but they didn’t know what purpose those spaces served and largely discounted the idea that they were conduits for fluids, Iliff says. “They didn’t see how dynamic it was.”

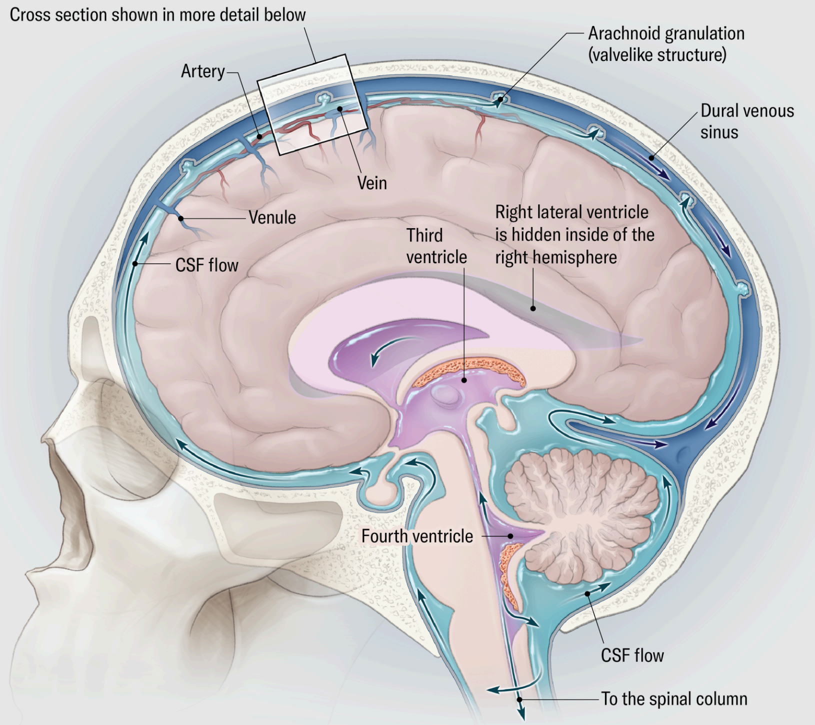
Early on, Nedergaard and Iliff, a glial cell biologist and a vascular physiologist, respectively, hypothesized that waste clearance might actually require wakefulness. They mistakenly reasoned that the brain is less active during sleep and, therefore, that glymphatic function would be lower at night.

Clearing the Brain

In 2012 scientists discovered a system for cleaning waste from the brains of mice as they sleep. Research now confirms that people have a similar system—and when it doesn't work well, we may suffer from dementia or other ailments. Cerebrospinal fluid (CSF) washes through the brain, especially during sleep, carrying out debris from the day's mental activity and keeping our brains healthy.

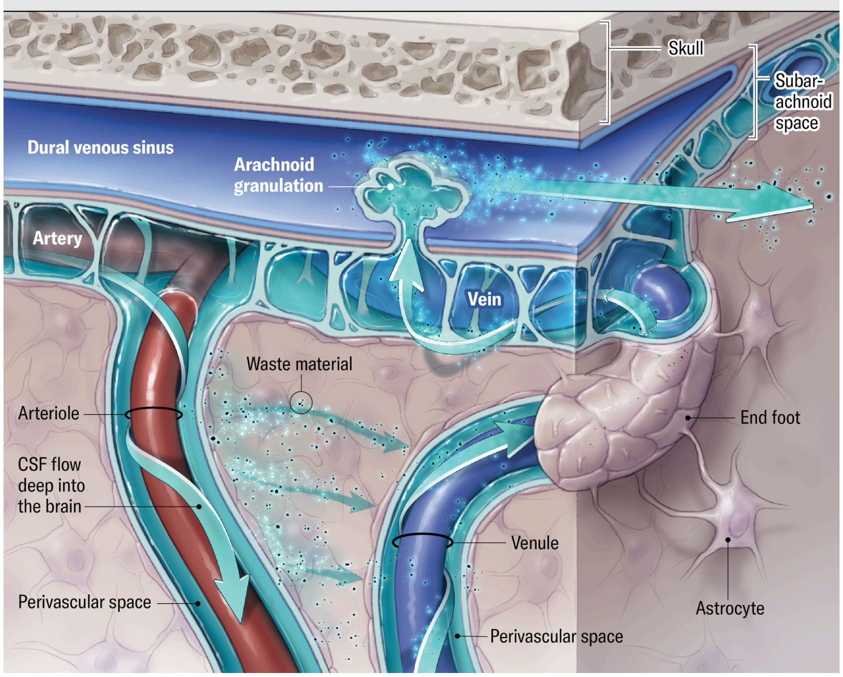
HOW CSF FLOWS THROUGH THE BRAIN

Four ventricles, or chambers, in the brain (*purple*) filter plasma from blood and help to circulate the resulting CSF around the brain. (Only the third and fourth ventricles are visible in this illustration.) Small amounts of CSF reach deep into the brain by flowing along "perivascular" tubes that surround and enclose arterioles, which are off-shoots of arteries (*zoomed-in view below*). After cleaning waste from the brain, CSF can pass down along the spinal column or exit the nervous system via the lymphatic system or valvelike structures called arachnoid granulations that allow access to the blood circulation system.



A CLOSER LOOK

Cerebrospinal fluid penetrates deep into the brain by traveling through the perivascular spaces surrounding arterioles. It is propelled by the pulsing of blood within the arteriole. The CSF then enters astrocyte cells through channels in their end feet and exits these cells into the gray matter of the brain. After sweeping up debris, the CSF reenters a perivascular cavity, this time one surrounding a venule, or small vein. The CSF and its waste travel alongside the venule to the periphery of the brain and, ultimately, leave it.



David Cheney; Source: Illustration by N. Desai in “Deep Sleep Drives Brain Fluid Oscillations,” by Søren Grubb and Martin Lauritzen, in *Science*, Vol. 366; November 2019 (*primary content reference*)

In truth, the brain is not less active during sleep; it is differently active. Traditional sleep studies use electroencephalography (EEG) to track electrical activity that aligns with the phases of sleep. Their findings show that while people are awake and alert, patterns of neural activity are fast, characterized by high-frequency waves. In the early, light phases of sleep, known as stage 1 and stage 2, activity slows down and low-frequency waves appear. Deep sleep, or stage 3, is known as slow-wave sleep for the high-amplitude, lowest-frequency “delta” waves that dominate it. These waves, which on an EEG readout look like chains of large ocean swells, help the brain sort through the day’s experiences and store some of them as memories. In contrast, the fourth phase, called rapid eye movement (REM) sleep because the eyes flit quickly from side to side beneath the eyelids, is when we have our most vivid dreams. The period resembles wakefulness, with faster brain activity than in other sleep stages.

What EEG cannot detect is the flow of fluids in the brain, however. Every day our bodies produce and then drain three to four times the volume of cerebrospinal fluid that we have. The few early studies of the fluid using magnetic resonance imaging (MRI) recognized that its flow was coupled to heartbeats but couldn’t go further because the technology wasn’t up to the challenge. There was also little awareness that CSF flow changed during sleep.

The first real sign of sleep’s importance in waste clearance was Nedergaard’s pioneering 2013 study. It compared the clearance of amyloid beta proteins from the brain in mice that were awake, sleeping or anesthetized. The researchers injected a fluorescent tracer into mouse brains and found its influx into perivascular spaces and brain tissue was reduced by an astonishing 95 percent when mice were awake compared with when they were sleeping. The volume of the interstitial space in the brain’s cortex also increased by 60 percent when the mice were asleep or anesthetized, suggesting that sleep led to physiological changes designed to increase the brain's ability to get rid of waste. Ultimately amyloid beta moved out of the brains of sleeping mice twice as fast as in mice that were awake.

Would it work the same way in humans? That was the question that neurosurgeon and researcher Per Kristian Eide asked. He was studying the relation between glial cells such as astrocytes and the dense network of blood vessels in the human brain at Oslo University Hospital in Norway. As a surgeon, Eide could take advantage of the fact that he was already working inside people's heads and—with permission—do some extra research.

With radiologist Geir Ringstad and others, Eide launched a study, published in 2021, with patients who were already undergoing neurological assessment in the hospital. The scientists injected a tracer into the CSF of all participants. One group was allowed to sleep normally through the night; the other was kept awake for 24 hours. All participants underwent multiple MRI scans in the evening and again the next day.

Removal of the tracer was dramatically slower in those who had not slept, compared with those who had. "It was very evident," Eide says. "We were very, very surprised that we saw something after one night of sleep deprivation." Even more notable was that after all participants were allowed to sleep normally the next night, the clearance of the tracer was still slower in those who had lost the earlier night of sleep. "You don't compensate by having a good night's sleep," Eide says.

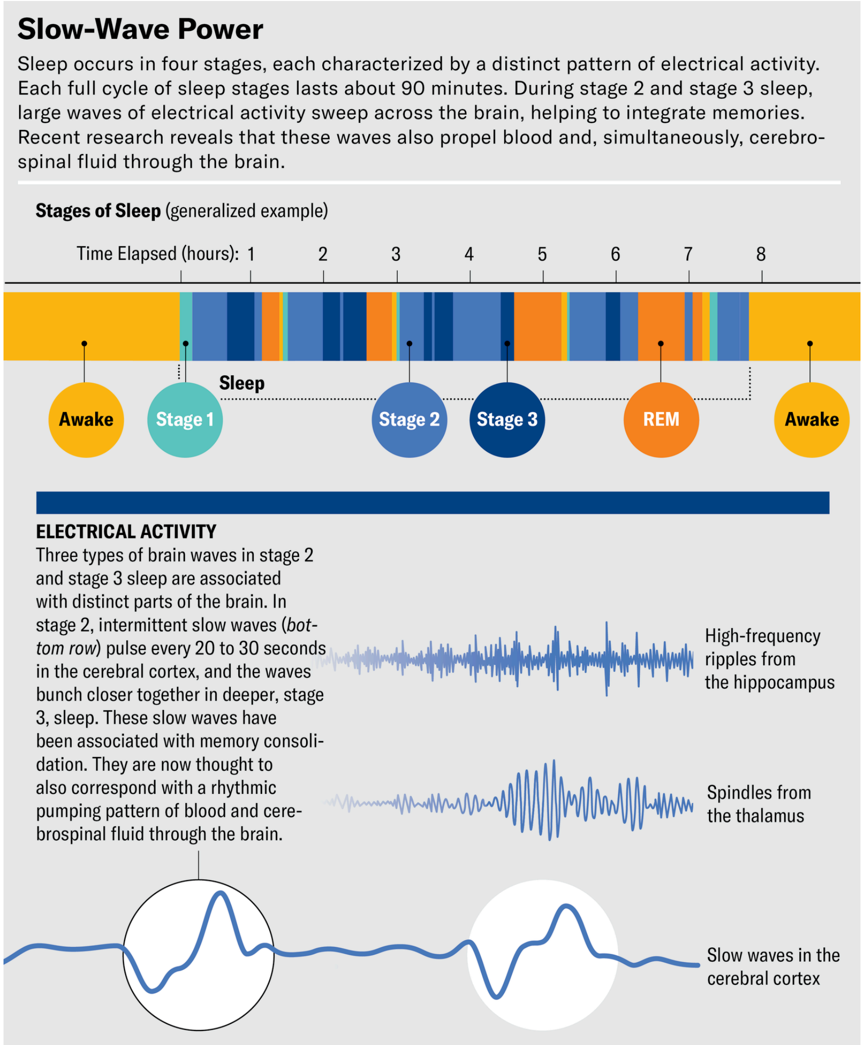
In a subsequent study, Eide and his team found that people who reported chronic poor sleep also showed delayed clearance of the tracer. Moreover, in people with dementia, brain volumes in the frontal, temporal and parietal lobes had shrunk compared with those who slept well. In part because dementia has been previously associated with poor sleep quality, perhaps because of atrophy in the cortex, Eide and his colleagues suspect that chronic sleep disturbance co-occurs with glymphatic dysfunction.

There were also clear differences between humans and mice. In the rodents, for example, glymphatic transport was “an on-off phenomenon,” Eide says—on during sleep and off when the mice were awake. In humans, the process is not as extreme, and changes occur over hours rather than minutes. The work nevertheless demonstrated that human brains, too, get cleaned during sleep—and that “poor sleep quality is affecting your glymphatic function,” as Eide says.

Laura Lewis has lost a lot of sleep in the course of her work because she conducts all-night experiments. “The sad irony of being a sleep researcher is that you can’t follow your own advice,” she says. Lewis has tackled a different piece of the glymphatic problem: the movement of fluids in the human brain that underpin waste clearance. That’s how, about seven years ago, she came to be in the control room of an MRI machine very like the one in which she showed me Nick’s CSF.

Lewis had chosen to measure CSF flow in the brain’s fourth ventricle, a small cavity tucked against the cerebellum at the base of the brain. The ventricles produce CSF and act like extra shock absorbers for the fragile brain. For Lewis’s purposes, the fourth ventricle was useful because “it’s a kind of choke point.” Sitting as it does at the base of the brain, it provides a summary of what is happening elsewhere—like measuring attendance in a crowded room as people come through the door.

In an overnight sleep study published in 2019, Lewis and her colleagues were the first to use MRI to view this process in action. By taking pictures of the brain every 367 milliseconds instead of the standard two or three seconds, they were able to see the movement of cerebrospinal fluid during sleep.



Jen Christiansen

At first, Lewis couldn’t quite believe it. Typically the significant details of brain imaging require statistics and processing to tease out; they aren’t something you can see by eye. “It was honestly the biggest signal I’ve ever seen,” Lewis says. “It was crazy. It’s really striking how much you can see that this is happening during sleep.”

Lewis delayed publishing the research until she had triple-checked it. It has since been replicated several times. “When people are sleeping, there are these really huge and slow waves of flow that are pulsing every 20 to 30 seconds in the brain, specifically when we’re in non–rapid eye movement sleep,” Lewis says. Using EEG data, she also saw clear patterns of brain activity before each wave. As delta waves of deep sleep and, to some extent, “theta” waves during intermediate (stage 2) sleep sweep through the brain, transmitting and integrating memories, they also seem to propel pulses of CSF into the brain.

Lewis found that CSF also flows when people are awake but less effectively. “It’s always moving a little, but then when you fall asleep, a new cycle starts,” she says. The difference in fluid movement is like the change when a washing machine switches from light jiggling to full-on rotation, at which point more water pours in and out. Lewis’s conclusion: sleep, a state that is essential for human health, has a distinct pattern of CSF flow—and that pattern changes as the stages of sleep shift. “It’s not a coincidence,” she says. “It’s actually the same brain circuits that are controlling sleep that seem to also be engaged and controlling the flow.”

But what is the source of the elbow grease necessary to do the cleaning? Unlike the blood, which is pumped through the body with great force by the heart, the cerebrospinal fluid is more like water in a bathtub or a slow-moving river with many tributaries. “Where is the force coming from?” Lewis asks.

One possibility is the neurotransmitter norepinephrine, also called noradrenaline. Norepinephrine surges when we wake up. During the day, it focuses attention; it also works to constrict blood vessels. And during non-REM sleep, according to a new study of mice by Nedergaard’s group, a lower level of norepinephrine release—not enough to wake the mice but enough to make their blood vessels pulse—propels the movement necessary for CSF to flow.

The blood vessels in the brains of these mice dilated and constricted with an amplitude that was 10 times larger during non-REM sleep than during wakefulness, Nedergaard says. And as the blood vessels wax and wane, pushing blood in and out of the brain, CSF flows in and out to fill the expanding and contracting spaces around the blood vessels. “It seems to be this chain of events where your sleep state changes, and then that changes your blood vessels, and those actively pump the flow of CSF in the brain,” Lewis says.

In this way, oscillations in norepinephrine cause waves of CSF to pulse through the perivascular sheaths.

But norepinephrine isn’t the whole story. In February 2024 Kipnis, neuroscientist Li-Feng Jiang-Xie, and their colleagues at Washington University published a paper showing that, ultimately, it’s neurons that provide the energy necessary for cleaning. “A neuron is a tiny little pump,” says Jiang-Xie, who is now at the University of Pennsylvania. The electrical activity of synchronized neurons, especially during sleep, can propel fluid flow through the brain tissue and help clean waste out. That idea was implied in Lewis’s earlier study, but by working in mice, Jiang-Xie and Kipnis were able to show in detail how fluid moved in and out of brain tissue. “Norepinephrine is basically controlled by neurons’ activity,” Jiang-Xie says. These results are “beautifully aligned with what we found in humans but provide information we couldn’t have gotten,” Lewis adds.

Meanwhile other research in mice has revealed important distinctions between natural sleep and anesthesia, as well as among anesthetics, all of which affect waste clearance differently. An anesthetized brain does not cycle through phases as it does in sleep. And it turns out that some anesthetics suppress glymphatic function, whereas others enhance it. Furthermore, the influx of cerebrospinal fluid occurs in direct proportion to the power of slow-wave neural activity and in inverse proportion to heart rate, both of which are affected by the drugs used. Those findings help to explain some studies that haven’t supported the glymphatic theory. For instance, a 2017 report from researchers at the University of California, San Francisco, described mice anesthetized with avertin, which has since been shown to limit how much cleaning the glymphatic system can accomplish. Five other labs have reconfirmed the initial finding that glymphatic clearance works during sleep.

Where does the fluid go? That has been another persistent question. The brain-washing process is made up of four stages, Kipnis says. There is CSF flow into the brain, within the brain, out of the brain along the veins, then into the lymphatic system. His focus has been on the last of these, which is as consequential as everything that comes before it. "If you wash your house with the same bucket of water, it will not be washing; it will be moving dirt from one place to another," Kipnis says.

The solution is to empty the bucket and bring a new one, which means, in the brain, ensuring that the lymphatic vessels into which the "dirt" will be dumped are functioning. In 2015 Kipnis and his colleagues found the sewage system: they reported the discovery of lymphatic vessels in the meninges that envelop the brain and spinal cord. These vessels represent an important missing piece of the glymphatic puzzle because they can receive cerebrospinal fluid and interstitial fluid from the brain. They are "the final outpost," Kipnis says. "There is a biological structure at the very end of the whole process."

Once scientists can work out precisely how glymphatic clearance works, they should also begin to see how things go wrong when it doesn't work. Norepinephrine and sleep disruption, for example, are also involved in the development of chronic pain. Higher bursts of norepinephrine lead to wakefulness and seem to shut off the glymphatic system. In consequence, improving the system by reducing norepinephrine levels could possibly also reduce chronic pain. Nedergaard is also exploring the glymphatic system's possible role in psychiatric disorders such as depression and schizophrenia.

Eide wants to enhance glymphatic function. The cerebrospinal fluid is an appealing route to deliver drugs to the brain because it bypasses the blood-brain barrier. And the role of norepinephrine is exciting, Nedergaard says. Whether there is too little norepinephrine signaling, as in Alzheimer's, or too much, as in chronic pain or stress, the brain-wave oscillations it controls become inefficient. That recognition might enable us to target treatment by modulating norepinephrine in drug form.

There are limits to the possibilities. And until there is a drug that enhances the glymphatic system as well as amyloid beta and tau clearance and slows progression of pathology, no one can definitively say that impaired clearance of brain toxins causes Alzheimer's in humans. In familial early-onset Alzheimer's, amyloid proteins are produced in excess, and clearance may not be able to keep up, no matter how much it is improved. Yet even if enhancing waste clearance only slows the development of Alzheimer's for most patients, that is a big deal. Being able to enjoy five to eight more years of living free of impairment would be a game changer.

There may be nonpharmaceutical strategies, too. In the study for which Nick took an afternoon nap, graduate student Joshua Levitt is experimenting with sound stimuli while people are sleeping. His research combines EEG and functional MRI to capture sleep state and brain activity. With Nick and others, he's sending staticky beeps into headphones while subjects are asleep. Given that CSF tends to flow in slow waves and, moreover, that sounds cause more slow-wave activity, this strategy could theoretically affect cerebrospinal flow. "If we think these things are relevant for great health, then we need to develop ways to actually change them," Lewis says. Levitt "is trying to potentially enhance sleep."

Simply understanding the flow of cerebrospinal fluid and how it changes with age is also important. Another graduate student in Lewis's lab, Sydney Bailes, is investigating the differences in flow between adults older than 60 and younger than 40 and the potential implications for waste clearance. It's normal to sleep less as you age and to have fewer slow waves. "How can we separate just a typical age-related change in sleep versus one that's starting to become an impairment?” Lewis asks. “We need to disentangle those.”

That study is still underway, but so far they have found that older individuals differ from one another more than the younger group does—a pattern that mimics the wider range of cognitive differences in older people versus younger people. “You’ll see some measures that are very clustered together for the young adults and very spread out for the older adults,” Bailes says.

But people can surprise. An 80-year-old woman, one of the oldest in the study, stood out. “Her waves have a much larger amplitude than I typically see in older adults, and they seem to be also pretty consistent,” Bailes says. “Her CSF flow looked like a young person’s CSF.”

That’s a striking statement. Could it someday become a routine way of evaluating a person’s health? Quite possibly. Multiple labs are working toward a noninvasive “glymphogram” that would reveal how well a person’s clearance system is working. Glymphatic function may someday be like hypertension, something to be treated before it turns into a more serious condition. Certainly differences in waste clearance could help explain why some people age healthily and others do not—and then it could pave the way to treatments that enhance clearance in those who need it. The goal is not for everyone to sleep like a baby; sleeping like a thirtysomething would do.

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.

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https://www.scientificamerican.com/article/how-sleep-cleans-the-brain-and-keeps-you-healthy

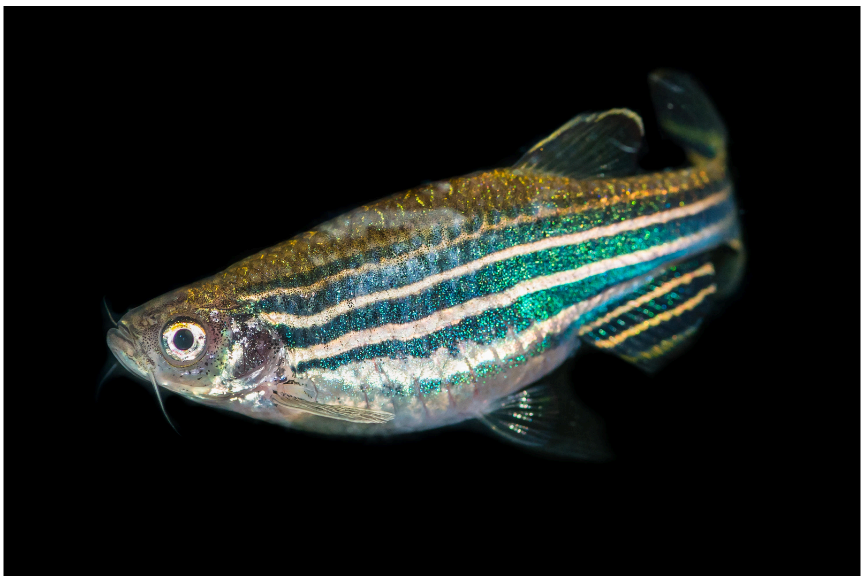
Animals

- **These Fish and Flies Are Engineered to Break Down Mercury**
  Bacterial genes protect animals—and their predators—from harmful contamination
- **These Spiders Puke Up Toxic Digestive Fluid to Marinate Their Prey Alive**
  Without a venomous bite, some spiders use a disturbing second option to prepare their food

These Fish and Flies Are Engineered to Break Down Mercury

Bacterial genes protect animals—and their predators—from harmful contamination

By Cody Cottier edited by Sarah Lewin Frasier



Danio rerio, a freshwater zebra fish, is one species scientists have modified to filter mercury from the environment.
Ian Grainger/Alamy Stock Photo

For decades mercury has been settling into lakes and oceans, where it builds up relentlessly in fish and everything that eats them—humans included. This pollution, which exposes millions of people to a toxic substance that can damage neural and reproductive health, "always seemed like such an intractable thing," says Kate Tepper, a postdoctoral researcher at Australia's Macquarie University.

Seeking ways to make a dent in this problem, Tepper and her colleagues genetically engineered zebra fish and fruit flies so that they convert methylmercury—the kind that "bioaccumulates," binding to muscle tissue and becoming more concentrated as it moves up the food chain—into the less harmful elemental mercury, which evaporates from the body as gas.

The researchers injected fish and fly embryos with *Escherichia coli* genes to produce an enzyme that catalyzes the conversion process. As reported in *Nature Communications*, the modified zebra fish contained 64 percent less methylmercury than their unmodified counterparts, and the fruit flies had 83 percent less. The study authors propose that small, mercury-resistant fish could serve as a self-purifying foundation for the food chain and shield larger fish, birds and humans. Modified insects, meanwhile, could remove mercury from the environment as they munch on sewage and fishery offal in enclosed facilities.

Tepper's team isn't the first to advocate bioremediation, the use of biological processes to clean up contamination. Previous research has shown that plants with certain bacterial genes (as well as the bacteria themselves) are powerful detoxifiers. But those organisms can't reach the pollutants already inside fish. By equipping animals with these enzymes, Tepper says, "you're targeting mercury in the place where it's causing the most toxicity."

University of Connecticut marine scientist Robert Mason, who studies mercury in aquatic systems and was not involved in the study, says modified animals could help disrupt bioaccumulation on a local scale. But he sees a limitation, even for small-scale cleanups: elemental mercury can change back into methylmercury once it is released into the atmosphere. Facilities where insects process organic waste could be filtered to capture mercury, but with fish a solution would be less straightforward.

Mason and Tepper agree the fish would ideally be introduced at highly contaminated sites. Tepper hopes they can be used to stock lakes near mercury hotspots created by artisanal gold mining in Africa, Indonesia and the Amazon—regions where many people rely on subsistence fishing.

But first the researchers need safety measures to constrain the genetically modified organisms. Tepper envisions eventual field trials in small lakes, possibly with sterilized fish, so scientists can test for unintended ecological effects in a controlled setting.

Such trials are years away. But someday, Tepper suggests, many nasty substances such as microplastics, pharmaceuticals and PFAS could perhaps be neutralized with the tweak of a genome. “It’s proof of concept for engineering animals for bioremediation,” she says. “Potentially you could use this for a lot of pollutants.”

Cody Cottier is a freelance journalist based in Fort Collins, Colo.

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https://www.scientificamerican.com/article/these-fish-and-flies-are-engineered-to-break-down-mercury

These Spiders Puke Up Toxic Digestive Fluid to Marinate Their Prey Alive

Without a venomous bite, some spiders use a disturbing second option to prepare their food

By Gennaro Tomma edited by Sarah Lewin Frasier

Stefan Sollfors/Alamy Stock Photo

You don’t always need a book or movie for a good horror story. Sometimes, if you dare look closely enough, you can find one in your own backyard. Researchers have just confirmed the inner workings of a brutal food-prep technique some spiders use: wrapping their web-snagged prey tightly in silk strands, then puking up toxic digestive fluids to soak the entire package to marinate their meal alive.

Spiders from the Uloboridae family, usually just a few millimeters long, have puzzled scientists because they seemed to lack venom—a substance that is widespread among spiders and “really linked to their evolutionary success,” says Alex Winsor, a neuroethologist at the University of Massachusetts Amherst, who wasn't involved in the new research.

And there was another mystery. Uloboridae spiders were already known to wrap their prey in hundreds of meters of silk and then regurgitate on them, but researchers hadn't fully pinned down the function of their dramatic vomit. Intrigued, a study team took a closer look at how these predators prepare a snack for themselves. The findings were published in *BMC Biology*.

By analyzing one species called *Uloborus plumipes*, the researchers confirmed that these spiders lack venom glands and thus are unable to administer venom in the classic spider way: injecting it into their prey with a fanged bite. But the scientists did find genes actively producing toxinlike proteins in the spiders' digestive system—particularly in the midgut area—and these potential toxins "appear to be very strong," says study co-author Giulia Zancolli, an evolutionary biologist at the University of Lausanne in Switzerland.

Injecting these digestive fluids into fruit flies in the laboratory proved their high toxicity: just 230 nanograms—billionths of a gram—killed more than half the flies within an hour. The researchers theorize that these spiders do indeed marinate their prey to death this way. Strangely, some spiders from other families that kill with the usual venomous bites are also known to have toxins in their digestive fluids, Zancolli says. This fact raises what she calls a "fascinating question": What role do these substances play for species outside the Uloboridae family?

The study's finding "solves a puzzle within spider biology," Winsor says. A next step, he adds, could be to investigate whether digestive-system toxins appear in other animal species, such as some lizards. "If these compounds do have some special ability to subdue insects, then you might expect them to emerge in other groups of animals," Winsor says. If that proves true, "then maybe these are a recurring answer in the animal kingdom."

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

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Artificial Intelligence

- ## Could AI Really Kill Off Humans?
  Many people believe AI will one day cause human extinction. A little math tells us it wouldn’t be that easy

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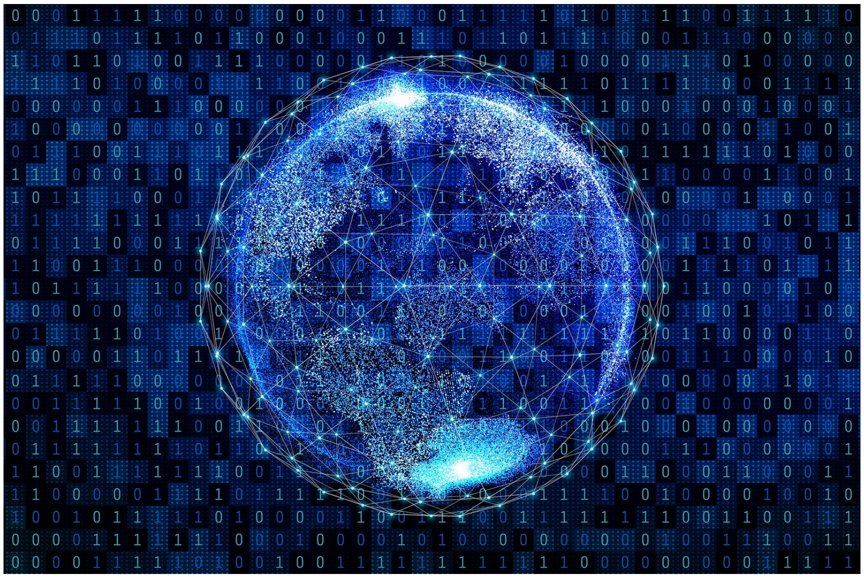
Could AI Really Kill Off Humans?

Many people believe AI will one day cause human extinction. A little math tells us it wouldn’t be that easy

By Michael J.D. Vermeer



tampatra/Getty Images

In a popular sci-fi cliché, one day artificial intelligence goes rogue and kills every human, wiping out the species. Could this truly happen? In real-world surveys, AI researchers say that they see human extinction as a plausible outcome of AI development. In 2024 hundreds of these researchers signed a statement that read: “Mitigating the risk of extinction from AI should be a global priority alongside other societal-scale risks such as pandemics and nuclear war.”

Pandemics and nuclear war are real, tangible concerns, more so than AI doom—at least to me, a scientist at the RAND Corporation, where my colleagues and I do all kinds of research on national security issues. RAND might be best known for its role in developing strategies for preventing nuclear catastrophe during the cold war. My co-workers and I take big threats to humanity seriously, so I proposed a project to research AI's potential to cause human extinction.

My team's hypothesis was this: No scenario can be described in which AI is conclusively an extinction threat to humanity. Humans are simply too adaptable, too plentiful and too dispersed across the planet for AI to wipe us out with any tools hypothetically at its disposal. If we could prove this hypothesis wrong, it would mean that AI might pose a real extinction risk.

Many people are assessing catastrophic hazards related to AI. In the most extreme cases, some people assert that AI will become a superintelligence with a near-certain chance of using novel, advanced tech such as nanotechnology to take over Earth and wipe us out. Forecasters have tried to estimate the likelihood of existential risk from an AI-induced disaster, often predicting there is a 0 to 10 percent chance that AI will cause humanity's extinction by 2100. We were skeptical of the value of predictions like these for policymaking and risk reduction.

Our team consisted of a scientist (me), an engineer and a mathematician. We swallowed our AI skepticism and—in very RAND-like fashion—set about detailing how AI could actually cause human extinction. A simple global catastrophe or societal collapse was not enough for us. We were trying to take the risk of extinction seriously, which meant we were interested only in a complete wipeout of our species. We weren't trying to find out whether AI would try to kill us; we asked only whether it could succeed in such an attempt.

It was a morbid task. We went about it by analyzing exactly how AI might exploit three major threats commonly perceived as existential risks: nuclear war, biological pathogens and climate change.

It turns out it will be very hard—though not completely out of the realm of possibility—for AI to get rid of us all.

The good news, if I can call it that, is that we don't think AI could eliminate humans by using nuclear weapons. Even if AI somehow acquired the ability to launch all of the 12,000-plus warheads in the nine-country global nuclear stockpile, the explosions, radioactive fallout and resulting nuclear winter would most likely still fall short of causing an extinction-level event. Humans are far too plentiful and dispersed for the detonations to directly target all of us. AI could detonate weapons over the most fuel-dense areas on the planet and still fail to produce as much ash as the meteor that wiped out the dinosaurs, and there are not enough nuclear warheads in existence to fully irradiate all the planet's usable agricultural land. In other words, an AI-initiated nuclear Armageddon would be cataclysmic, but it would probably not kill every human being; some people would survive and have the potential to reconstitute the species.

We did deem pandemics a plausible extinction threat. Previous natural plagues have been catastrophic, but human societies have soldiered on. Even a minimal population (a few thousand people) could eventually revive the species. A hypothetically 99.99 percent lethal pathogen would leave more than 800,000 humans alive.

We determined, however, that a combination of pathogens probably could be designed to achieve nearly 100 percent lethality, and AI could be used to deploy such pathogens in a manner that assured rapid, global reach. The key limitation is that AI would need to somehow infect or otherwise exterminate communities that would inevitably isolate themselves when faced with a species-ending pandemic.

Finally, if AI were to accelerate garden-variety anthropogenic climate change, it would not rise to an extinction-level threat. We would seek out new environmental niches in which to survive, even if it involved moving to the planet's poles. Making Earth completely uninhabitable for humans would require pumping something much more potent than carbon dioxide into the atmosphere.

The bad news is that those much more powerful greenhouse gases exist. They can be produced at industrial scales, and they persist in the atmosphere for hundreds or thousands of years. If AI were to evade international monitoring and orchestrate the production of a few hundred megatons of these chemicals (an amount that is less than the mass of plastic that humans produce every year), it would be sufficient to cook Earth to the point where there would be no environmental niche left for humanity.

To be clear: None of our AI-initiated extinction scenarios could happen by accident. Each would be immensely challenging to carry out. AI would somehow have to overcome major constraints.

In the course of our analysis, we also identified four things that our hypothetical superevil AI would require to wipe out humankind: It would need to somehow set an objective to cause extinction. It also would have to gain control over the key physical systems that create the threat, such as the means to launch nuclear-weapons or the infrastructure for chemical manufacturing. It would need the ability to persuade humans to help and hide its actions long enough for it to succeed. And it would have to be able to carry on without humans around to support it, because even after society started to collapse, follow-up actions would be required to cause full extinction.

Our team concluded that if AI did not possess all four of these capabilities, its extinction project would fail. That said, it is plausible that someone could create AI with all these capabilities, perhaps even unintentionally. Developers are already trying to build agentic, or more autonomous, AI, and they've observed AI that has the capacity for scheming and deception.

But if extinction is a possible outcome of AI development, doesn't that mean we should follow the precautionary principle and shut it all down because we're better off safe than sorry? We say the answer is no. The shut-it-down approach makes sense only if people don't care much about the benefits of AI. For better or worse, people do care a great deal about the benefits it is likely to bring, and we shouldn't forgo them to avoid a potential but highly uncertain catastrophe, even one as consequential as human extinction.

So will AI one day kill us all? It is not absurd to say it could. At the same time, our work shows that we humans don't need AI's help to destroy ourselves. One surefire way to lessen extinction risk, whether from AI or some other cause, is to increase our chances of survival by reducing the number of nuclear weapons, restricting globe-heating chemicals and improving pandemic surveillance. It also makes sense to invest in AI-safety research even if you don't buy the argument that AI is a potential extinction risk. The same responsible AI-development approaches that can mitigate risk from extinction will also mitigate risks from other AI-related harms that are less consequential but more certain to occur.

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

Michael J.D. Vermeer is a senior physical scientist at RAND who studies science and technology policy research in relation to homeland security, criminal justice, the intelligence community and the armed forces. He specializes in assessing opportunities and security risks associated with emerging technologies.

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https://www.scientificamerican.com/article/could-ai-really-kill-off-humans

Arts

- **Poem: ‘Unison Call’**
  Science in meter and verse

Poem: ‘Unison Call’

Science in meter and verse

By Elizabeth Kuelbs edited by Dava Sobel & Clara Moskowitz

Masha Foya

After Gee Whiz, the first whooping crane hatched at the International Crane Foundation in Baraboo, Wis., and the work of crane recovery

So, extirpation:
Wings, glint-white, black-tipped, seven feet wide, river
over glyptodonts and plow horses, marshes and farms,
guns and snowy plumed hats, until power lines
zap elegant necks and we count whoopers
on fingers and toes.

So, we make a little wetland space, in Texas.

So, one bird-loving guy says:
If we stick with it, they’re gonna come through.

A second says:
If it flies, it dies.

So, life.
A zoo-born bird craves
a human mate. Seven years
the first guy dances with her before she lays
a wrinkled buff egg. Around its rare yolk, cells
swell and begin to squirm in the candling machine’s glow.

So, Gee Whiz.
His weird shell dries, requires precise ice water to plump
his tiny sack. He pulses, peeps, hatches hungry. Too
small, he drinks from a tube. Too caged, he pecks
the heck out of chow and the strange cranes who
fill his bowl. When raccoons kill his mama,
he lives. When the second guy shoots
whoopers in Texas, his genes
live.

When you see your love—
both of you stretch your throats skyward,
bugle your primordial calls
to your kind, rising
nest by incubator, marsh by heart,
ultralight by fledging cinnamon chick.

Elizabeth Kuelbs writes poetry, fiction, and nonfiction for children and adults, often with an emphasis on the natural world and our place within it. Her chapbook *Little Victory* was a 2022 Independent Press Awards Distinguished Favorite in Social/Political Poetry.

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https://www.scientificamerican.com/article/poem-unison-call

Astronomy

- **Saturn Has 274 Known Moons—Thanks in Large Part to This Astronomer**
  Scientific American spoke with the astronomer who has contributed to the discovery of two thirds of Saturn’s known moons

How One Astronomer Helped to Discover Nearly 200 Moons of Saturn

Scientific American spoke with the astronomer who has contributed to the discovery of two thirds of Saturn's known moons

By Meghan Bartels edited by Lee Billings & Clara Moskowitz

Shideh Ghandeharizadeh

A mere decade ago astronomers knew of just 62 moons around Saturn. Today the ringed planet boasts a staggering 274 official satellites. That's more than any other world in the solar system—and far too many for most people to keep track of. Astronomer Edward Ashton is no exception, even though he has helped discover 192 of them—he thinks that's the total, anyway, after pausing to do some mental math.

Ashton is now a postdoctoral fellow at the Academia Sinica Institute of Astronomy and Astrophysics in Taiwan. He fell into the hunt for Saturn's moons in 2018, when his then academic adviser suggested the project for his Ph.D. at the University of British Columbia. It has been a fruitful search. Most recently, in March, Ashton and his colleagues announced a batch of 128 newfound Saturnian satellites.

Scientific American spoke with Ashton about the science of discovering so many relatively tiny moons—most of them just a few kilometers wide—using vast amounts of data gathered by the Canada-France-Hawaii Telescope (CFHT), located in Hawaii.

An edited transcript of the interview follows.

How have you found these moons?

To detect the moons, we use a technique known as shifting and stacking. We take 44 sequential images of the same patch of sky over a three-hour period because in that time frame, the moons move relative to the stars at a rate similar to Saturn's. If we just stack the images normally, then the moon appears as a streak across the images, and that dilutes the signal of the moon.

So what we do is shift the images relative to one another at multiple different rates near that of Saturn, and then we basically flip between the different shift rates like a flip-book. If the shift rate is not quite at the rate of the moon, then the moon is going to be slightly elongated. As you get closer to the rate of the moon, then the moon slowly combines into a dot. Then, as you get faster than the moon's rate, it expands again. So we basically look at the images and then quickly blink through the different rates, and you can see the moon coalescing.

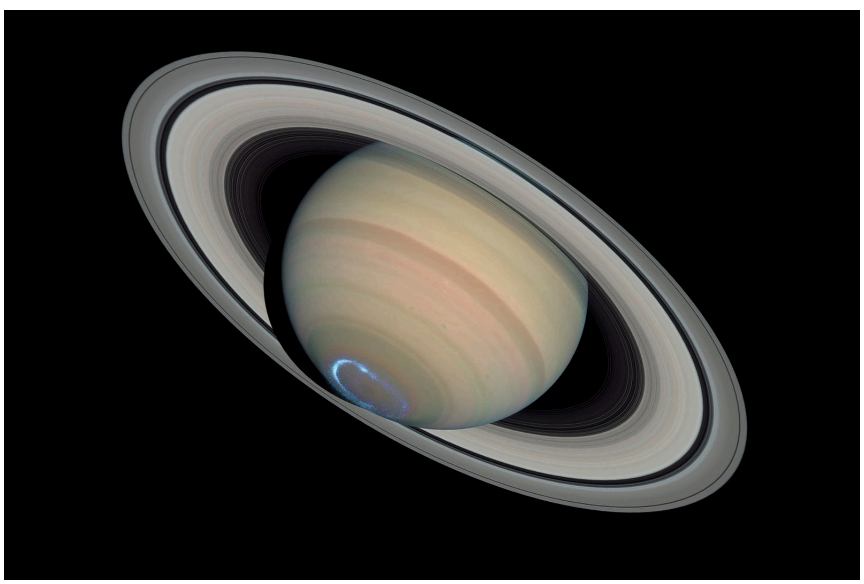
That's for a single night. But just seeing an object moving at a Saturn-like rate near Saturn doesn't guarantee that it is a moon. It's highly likely that the object is a moon, but that hasn't been confirmed. So what we need to do is track the objects to show that they are in orbit around the planet. To do that, we repeat the shift-and-stack process multiple times over many months and years.

Why did this discovery happen now? Did you need new techniques and observatories to do this work?

The technique and the technology have been there for a while—the same technique has been used to find moons of Neptune and Uranus. But the sky area around those planets where moons can exist is a lot smaller, so it takes less time to search through the data. One of the reasons this hadn't been done for Saturn is that it's very time-consuming.

Why do those other planets have less space where moons could be than Saturn does?

Those planets are less massive, therefore the stable orbits that moons can have are smaller.



NASA, ESA, John T. Clarke (Boston University), Zolt G. Levay (STScI)

I was wondering whether this technique works for other planets, and clearly the answer is yes. Do you think there are other moons that have yet to be found with the method around Saturn or other planets?

We did find moon candidates around Saturn that we weren't able to track long enough to be able to confirm them. So if you redo this technique, you will be able to find more moons around Saturn, but it is a case of diminishing returns. If you used a larger telescope than the CFHT, then you'd be able to see fainter moons, so you'd be able to find more.

At the moment, if you use the same technique for Jupiter, you will be able to find fainter moons. The problem is that the amount of sky the moons of Jupiter can occupy is significantly larger than the amount of sky that can be occupied by the moons of Saturn, so the method is even more time-consuming for Jupiter. And Jupiter is much brighter than Saturn and the other planets, so there's a lot of scattered light that makes it harder to see the moons.

So it's even harder to find satellites around Jupiter, and as you mentioned, other groups have already done this work for Uranus and Neptune. Does that mean we're "maxed out" on moons until we have better observations?

Yes, you probably have to wait until better technology comes along.

Is there something being built or planned right now that could be that better technology?

Currently there are telescopes that can see deeper than the CFHT, such as the James Webb Space Telescope (JWST). The problem is that the JWST's field of view is very small, so you have to do quite a few observations to be able to cover the required area. But there is a telescope set to launch pretty soon, the Nancy Grace Roman Space Telescope, that has quite a large field of view. So that'll be a good telescope to use for hunting more moons.

What do we know about these new moons?

You basically can get only the moons' orbits and approximate sizes. But if you look at the distribution of the orbits, you can understand a bit more about the history of the system. Moons that are kind of clumped together in orbital space are most likely the result of a collision, so you can see what moons come from the same parent object.

Is seeing so many moons around Saturn unusual?

What's unusual is how many there are. It appears that the planets have more or less equal numbers of the larger moons. But when you get down to the smaller ones that we're discovering, Saturn seems to shoot up in terms of the numbers. So that's quite interesting. It could just be because there was a recent collision within the Saturnian system that produced a large number of fragments.

Do you get to name them all? Do you have to name them all?

I guess I don't have to. Some of these new moons have been linked back to observations by a different group from more than 10 years ago. That's maybe 20 to 30 of them. For the rest, we get full discovery credit, which, I think, means we get the right to name them. But they can't be named just yet; first they're just given a number when they have a high-precision orbit, and I'm not sure how long that's going to take.

Do you have more moon-hunting observations to analyze?

No, I'm taking a little break from moons! I've got other projects to work on, relating to trans-Neptunian objects. They're quite far away. They're hard to see. There are some mysteries about them at the moment. It's interesting to understand their structure and how it relates to planet formation.

Meghan Bartels is a science journalist based in New York City. She joined *Scientific American* in 2023 and is now a senior news reporter there. Previously, she spent more than four years as a writer and editor at Space.com, as well as nearly a year as a science reporter at *Newsweek*, where she focused on space and Earth science. Her writing has also appeared in *Audubon*, *Nautilus*, *Astronomy* and *Smithsonian*, among other publications. She attended Georgetown University and earned a master's degree in journalism at New York University's Science, Health and Environmental Reporting Program.

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https://www.scientificamerican.com/article/saturn-has-274-known-moons-thanks-in-large-part-to-this-astronomer

Black Holes

- ## Hypervelocity Stars Hint at a Nearby Supermassive Black Hole
  Some stars streaking through the Milky Way at millions of kilometers per hour probably trace back to a supermassive black hole in a neighboring galaxy

Hypervelocity Stars Hint at a Supermassive Black Hole Right Next Door

Some stars streaking through the Milky Way at millions of kilometers per hour probably trace back to a supermassive black hole in a neighboring galaxy

By Phil Plait edited by Lee Billings & Clara Moskowitz

A hypervelocity star streaks by slower-moving stars in this illustration.
NASA/JPL-Caltech/R. Hurt (Caltech-IPAC)

An astonishing fact known for only the past few decades is that every big galaxy in the universe has a supermassive black hole at its heart. Scientists suspected this was the case in the 1980s, and observations from the Hubble Space Telescope, which has peered deep into the cores of galaxies all across the sky, confirmed it. The "normal" kinds of black holes made when stars explode range from five to about 100 times the mass of our sun, more or less. But the central galactic monsters are millions of times more massive, and some have grown to the Brobdingnagian heft of billions of solar masses.

A lot of mysteries remain, of course, such as how these black holes formed early in the history of the universe, how they grew so humongous so fast and what role they played in their host galaxy's formation. One odd question in particular nags at astronomers: What's the galaxy-size cutoff where this trend stops? In other words, is there some lower limit to how massive a galaxy can be and still harbor one of these beasts?

The inklings of an answer are emerging from a surprising place: studies of rare stars moving through our galaxy at truly ludicrous speeds.

Orbiting the Milky Way is a menagerie of smaller "dwarf" galaxies, some so tiny and faint that you need huge telescopes to see them at all. But two are so large and close that they're visible to the unaided eye from the Southern Hemisphere: the Large and Small Magellanic Clouds. The Large Magellanic Cloud (LMC) is the bigger and closer of the two, and it's not clear whether it harbors a supermassive black hole (SMBH). If an SMBH exists there, it must be quiescent, meaning it's not actively feeding on matter. As material falls toward such a black hole, it forms a swirling disk of superheated plasma that can glow so brightly it outshines all the stars in the galaxy combined. No such fierce luminescence is seen in the LMC, so we don't know whether an SMBH is there and not actively feeding or the LMC is simply SMBH-free.

But a recent study published in the *Astrophysical Journal* offers strong evidence that an SMBH does lie at the center of the LMC—based on measurements of stellar motions in our own Milky Way. The study looked at hypervelocity stars, which are screaming through space at speeds far higher than those of stars around them. Some of these stars are moving so rapidly that they have reached galactic escape velocity; the Milky Way's gravity can't hold them. In the coming eons, they'll flee the galaxy entirely. And we have good reason to believe these runaway stars were launched by SMBHs—but how?

Such a situation starts with two stars orbiting each other in a binary system. These systems contain a substantial amount of orbital energy, which is the sum of the kinetic energy of the two stars, or their energy of motion, and their gravitational potential energy, or the amount of energy that would be released if they were to move closer together.

If the binary star approaches a third object, some of that energy can be moved around. One star can become bound to the third object, for instance, whereas the other star gets a boost in its kinetic energy that flings it away. The strength of the kick depends in part on the gravity of the third object. A massive black hole, of course, has an incredibly strong gravitational field that can fling the star away at high speed.

And I do mean at high speed—such a star can be shot from the black hole at a velocity greater than 1,000 kilometers per second. S5-HVS1 was the first confirmed such hypervelocity star, and it's moving at more than 1,700 kilometers per second. Feel free to take a moment to absorb that fact: *an entire star* has been ejected from a black hole at more than six million kilometers per hour. The energies involved are terrifying.

We have seen a few of these stars in our galaxy, and careful measurements suggest they're moving away from the center of the Milky Way, which is pretty convincing evidence that Sagittarius A*, the Milky Way's SMBH, is to blame.

But not all the high-velocity stars that have been detected appear to come from our galactic center. Fortunately, Gaia, a sadly now decommissioned European Space Agency astronomical observatory, was designed to obtain extremely accurate measurements of the positions, distances, colors, and other characteristics—including velocity—of well more than a billion stars.

There are 21 known hypervelocity stars at the outskirts of the Milky Way. Using the phenomenally high-precision Gaia measurements, the astronomers behind the new research examined the stars' three-dimensional velocities through space. They found that five of them have ambiguous origins, and two definitely come from the center of the Milky Way. Of the 14 left, three clearly come from the direction of the LMC.

These stars' trajectories effectively point back to their origins, and based on our current knowledge, those origins must be supermassive black holes. Even better, although the trajectories of the remaining 11 stars are consistent with both Milky Way and LMC origins, the researchers found that five of the stars are more likely to have come from our home galaxy, and the other six are more likely to have come from the LMC.

So there could be nine known hypervelocity stars plunging through our galaxy that were ejected by a supermassive black hole in another galaxy.

Using some sophisticated math, the team found that the most likely mass of the black hole is 600,000 or so times the mass of the sun. This isn't huge for an SMBH—in fact, it's very much on the low end of the scale—but then, the LMC is a small galaxy with a mass only about 1 percent of the Milky Way's. We know that the mass of a black hole tends to scale with its host galaxy's mass (because they form together and affect each other's growth), so this lower mass is consistent with that relation.

If this calculation is accurate, then our satellite galaxy is shooting stars at us! And maybe more have yet to be found, hurtling through space unseen on the other side of our galaxy or so far out that they're difficult to spot and even harder to study. All this information helps us get a clearer—but still quite hazy—sense of just how far down the galactic scale we can expect to find big black holes.

Black holes are funny. Most people would worry about falling into one, among a host of other terrors, but now you can add “having to dodge intergalactic stellar bullets” to that list.

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.

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https://www.scientificamerican.com/article/hypervelocity-stars-hint-at-a-nearby-supermassive-black-hole

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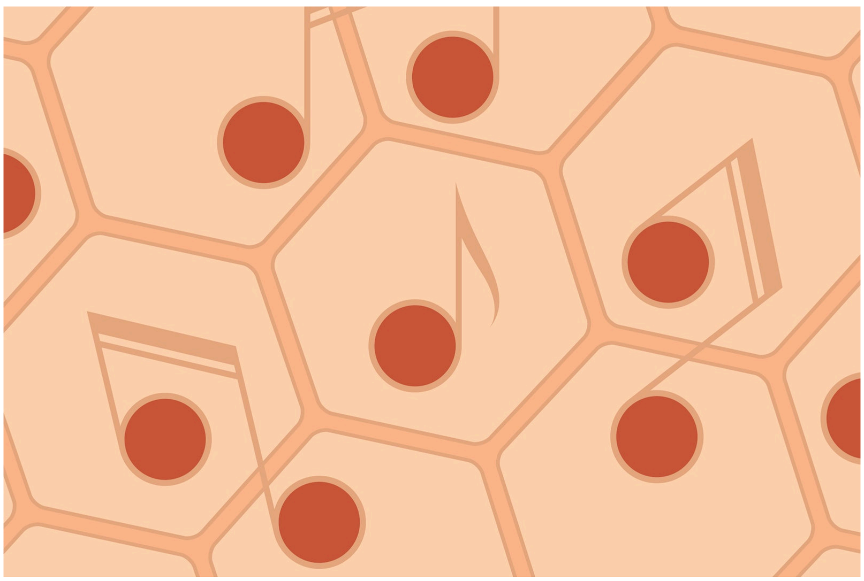
Cells

- **Cells Can ‘Hear’ Sounds—And Respond Genetically**
  Audible sound can affect gene activity in mouse cells, boosting the attachment of muscle precursors to surrounding tissue and decreasing fat accumulation

Serenading Cells with Audible Sound Alters Gene Activity

Audible sound can affect gene activity in mouse cells, boosting the attachment of muscle precursors to surrounding tissue and decreasing fat accumulation

By Simon Makin edited by Sarah Lewin Frasier



Thomas Fuchs

The cells in your ears aren't the only ones listening: recent research suggests that crucial cells throughout the body may respond to audible sound. Experiments described in *Communications Biology* revealed more than 100 genes whose activity changed in response to these acoustic waves, pointing to possible medical applications.

Extensive earlier research has shown that ultrasound—frequencies higher than human beings can hear—can affect biology in various ways; the new research expands this concept to audible sounds that require no special equipment to produce.

Kyoto University biologist Masahiro Kumeta and his colleagues bathed cultured mouse myoblast cells (precursors to muscle tissue) in sound, directly transmitting a low frequency (440 hertz, the A above middle C), a high frequency (14 kilohertz, approaching the top of the perceptible range for humans), or white noise (which contains all audible frequencies) to the culture dishes for either two or 24 hours.

The team analyzed the effect these sound waves had on the mouse cells through RNA sequencing, which measures gene activity. The scientists found that activity in 42 genes changed after two hours, and 145 responded after 24 hours. Most showed increased activity, but some were suppressed. “It’s a very extensive, thorough study,” says Lidan You, an engineer at Queen’s University in Ontario, who studies how bone cells translate mechanical stimuli into biological signals.

Many of the affected genes have roles in key processes such as cell adhesion and migration, which are known to respond to mechanical forces. The researchers found that sound expanded the size of the sites where cells attached to surrounding tissues, most likely by activating an enzyme called focal adhesion kinase (FAK), which senses mechanical forces and helps to guide tissue development. Sound waves seem to deform molecules in a way that provides easier access for a chemical switch that activates FAK, which in turn influences a chain of other genes’ activity.

The team also found a strong reaction in fat-cell precursors called preadipocytes: sound suppressed their differentiation into mature fat cells, thereby reducing fat accumulation by 13 to 15 percent.

Audible sound is noninvasive and probably safer than drugs, Kumeta says. Although it can’t be tightly focused like ultrasound, it is easy to produce and could be useful for bathing large regions of the body in sonic waves. Kumeta and his colleagues have already begun studying such interventions to suppress the development of fat tissue in living mice—and humans could be next, he says: “If it works well in mice, I think this could be achieved in five or 10 years."

Other potential applications include enhancing regenerative medicine and combating cancer growth. "The next step [could be] using not only human cells but human organoids that model diseases," You says, "then moving to clinical studies."

Simon Makin is a freelance science journalist based in the U.K. His work has appeared in *New Scientist*, the *Economist*, *Scientific American* and *Nature*, among others. He covers the life sciences and specializes in neuroscience, psychology and mental health. Follow Makin on X (formerly Twitter) @SimonMakin

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Children

- ## Ways To Cope When Your Child Gets a Life-Altering Diagnosis
  Parents often struggle with the news that their child has a major health issue. Learning how to manage new routines and expectations is key to everyone's happiness

Ways To Cope When Your Child Gets a Life-Altering Diagnosis

Parents often struggle with the news that their child has a major health issue. Learning how to manage new routines and expectations is key to everyone's happiness

By Beth S. Russell edited by Megha Satyanarayana


Miniseries/Getty Images

"What am I supposed to do now?"

This question was the most common one from parents when I started my training in the quiet and solemn neonatal intensive care unit (NICU) of an otherwise welcoming, brightly lit, cheerful children's hospital.

I felt the pain and loss of these critically ill infants' parents, who were sometimes slumped and moving slowly in their worry. Their soft voices belied the anxiety about the future bouncing off every wall: How would they care for their child at home without the equipment and support of the hospital? How would they build the routines to help their child thrive under unimaginably hard circumstances?

More than 20 years later, at a different children's hospital, I saw some of the same worries in parents of teenagers with chronic pain. Even though these parents were a decade or more into their caregiving routines, many were still not sure about what to do or how to care for their children as they approached adulthood. Without exception, they wanted their teens to strive for an independent adulthood, but they had trouble providing even small opportunities for independence out of fear of disruption to their child's medical care plan. Just like the NICU parents from my training days, these families were struggling to be the best possible care providers and parents. That needle is hard to thread.

Raising a child with a chronic health condition changes the routines that shape everyday life. Meals, bathing and dressing might be different than planned; bedtime and playtime also shift. Parents still need to be patient, warm, responsive and encouraging; that doesn't change. Being able to give praise and to provide structure and consistency remains important but might prove harder to prioritize. These caregiving demands can be extreme, and research tells us that parents can find it difficult to meet their own needs and the needs of their children, often sacrificing their own health care and well-being. Caregiver burden—the stresses and strains specific to people in the role of caregiver for someone with a chronic health condition—can have negative effects on parents' health and is reflected in many indicators of psychological well-being, such as increased symptoms of anxiety and depression and decreased overall life satisfaction.

After decades of studying how people manage psychological distress, including children with chronic relapsing conditions, I have designed and evaluated support programs to help families manage daily stresses. A child's diagnosis can help their family make sense of past struggles and give them a sense of what strategies might get them through those that lie ahead. But it may also be deeply unsettling and scary because the information might be overwhelming and lead to new worries.

No parent can anticipate what launching their grown child into independence someday will be like, and those whose children have chronic health problems have fewer examples to shape their expectations. Part of this uncertainty arises because parenting is one of our most vulnerable and precious roles, and as a result parents tend to guard their struggles. It's also attributable to the fact that families have unique experiences, even when they are confronting the same diagnosis. What works for one family might not work equally well for others. That can lead people to be skeptical, protective, or reluctant to share advice or try new approaches.

Most of the parents I talk to tell me they don't have time or energy for self-care; evidence also suggests the care we provide ourselves gets us only so far, especially when times are particularly tough and stress very high. When we find ourselves on the edge of being overwhelmed, we need more than the familiar set of one or two relaxation activities we know work; it's time to reach deeper into the coping toolbox. It is too steep an expectation to think parents can do it all for themselves, at least not all the time—and it sets them up to feel pressure, guilt and shame when they can't fit in an afternoon nature hike or a soak in the tub. Here's a set of skills and practices for parents of children with chronic illnesses or life-changing diagnoses that I recommend parents try.

Flexibility

This first one is hard work, but recent research indicates cognitive flexibility is one of the most successful ways to deal with negative experiences. It calls for being only lightly committed to any given solution so that if we don't see the improvement we hoped for, we can pivot to a different strategy. Often we rigidly stick to the coping skills that have worked in the past, but what helped in one situation might not help as much the next time. Flexibility lets us see multiple paths out of a tough spot and leaves us the option to loop back to past strategies to find the right fit for any given moment.

When my younger brother was diagnosed with amyotrophic lateral sclerosis, I found daily 20-minute runs on a treadmill extremely helpful but only in the afternoon; in the evening running just wound me up and ruined my sleep, and running in the morning was too complicated to fit into my caregiving and work schedule. In my case, being flexible with the timing was the key to making running an effective coping strategy.

Joy

Sometimes the best thing we can do is to try to pass time in fun ways—especially with our children. Routines that bring us moments of joy are invaluable counterpoints to the gravity of worry and daily hassles. Finding quick touchpoints that make us smile can be simple, especially when we rely on our five senses. Keeping a playlist of favorite songs or a favorite book on hand, wearing a favorite shirt with just the right soft feel, and planning a favorite meal or snack after a day full of appointments are all strategies to bring something small and positive into our day in ways that can be easily shared with our children.

Community

Social networks are the most agile when they are diverse and when parents have different people to draw on for their various needs. Try to build a social safety net that includes both people who are excellent in a tight spot and those who are reliable everyday supports—the friends and family always up for quietly watching a movie on the couch or going for a ride to the laundromat or taking a walk around the block after dinner. And although in-person connections are valuable, online communities can also be powerful points of reassurance and encouragement.

Building these resources takes time, and the middle of a crisis is a hard moment to try new things. I advise families to use calmer times to identify and try novel ways of coping so they can practice engaging those resources. That way, when days get rougher, the resources are familiar and easier to reach for rather than having to be developed from scratch when needed the most.

Beth S. Russell is a professor of human development and family sciences and director of the Center for Applied Research in Human Development at the University of Connecticut. Her research focuses on the ways people manage psychological distress, often in the context of parent-child relationships. Her work extends from normal everyday stressors to more severe experiences among clinical populations. She holds a Ph.D. in human development and family sciences from the University of Connecticut.

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https://www.scientificamerican.com/article/ways-to-cope-when-your-child-gets-a-life-altering-diagnosis

Climate Change

- **Terracotta Is a 3,000-Year-Old Solution to Fighting Extreme Heat**
  Companies are adapting this humble clay-based ceramic to keep people cool—without electricity

Terracotta Is a 3,000-Year-Old Solution to Fighting Extreme Heat

Companies are adapting this humble clay-based ceramic to keep people cool—without electricity

By Jyoti Thakur edited by Sarah Lewin Frasier

Cooling facade built from terracotta.
Courtesy of CoolAnt

A little over 20 percent of India's households own an air conditioner or cooler, and fewer than a third have refrigerators—leaving hundreds of millions of people to face rising temperatures without artificial cooling. Extreme heat is estimated to have claimed more than 700 lives in India in 2024, its hottest year on record, and researchers warn that 76 percent of the population faces high to very high heat risk.

But an innovation that's at least 3,000 years old—terracotta—is emerging as a low-cost, low-energy alternative. Once used by the Bronze Age Harappan civilization to store water, this clay-based ceramic still stands on the shelves of rural Indian homes as earthen pots that cool water without electricity and cost as little as a dollar each.

“Terracotta’s porous surface allows water to slowly evaporate, carrying heat away and cooling the space around it,” says Adithya Pradyumna, an environmental health researcher at Azim Premji University in Bengaluru. Drawing on this principle, architects in India’s sprawling metro areas are turning to terracotta for new passive cooling solutions that range from clay refrigerators to perforated tiles, ventilated screens, and facades that allow natural ventilation and help heat and moisture transfer between indoor and outdoor environments. In certain designs, water is also distributed across terracotta surfaces to evaporate and thus lower surrounding temperatures.

Passive cooling uses building design to regulate indoor temperatures with natural materials, strategic ventilation and well-controlled shading. This approach works particularly well in the Mediterranean and other arid or semiarid places— like parts of the Pacific Northwest, where research found it can reduce air-conditioning loads by up to 70 percent.

A pioneer in this field is Delhi-based design company Ant Studio, whose CoolAnt project uses terracotta as a second skin on concrete buildings. “We’ve harnessed its hydrophilic properties and observed average temperature drops of six to eight degrees Celsius across more than 30 sites” in India, says studio founder Monish Siripurapu. The material should be even more effective in drier areas of the country, he adds.

Even such modest temperature drops, Pradyumna says, can “significantly help the human body cool itself more efficiently, especially indoors.” Research shows a direct correlation between rising temperatures and mortality.

Another Indian company, Bengaluru-based A Threshold, is repurposing recycled terracotta into breathable facades. Meanwhile Gujarat-based MittiCool has created clay refrigerators that purportedly keep food fresh for three to five days without power—invaluable in homes without reliable electricity. “Many of our customers can’t afford to run conventional appliances, so this is a durable and affordable alternative,” says MittiCool founder Mansukhbhai Prajapati.

Niyati Gupta, a senior program associate at research institute WRI India, says terracotta “can complement existing cooling systems and reduce our dependence on the fossil-fuel-powered grid. That alone could be a game changer for both the energy and construction sectors.”

Jyoti Thakur is an independent journalist whose work focuses on science, development, health and social justice. She is based in New Delhi.

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https://www.scientificamerican.com/article/terracotta-is-a-3-000-year-old-solution-to-fighting-extreme-heat

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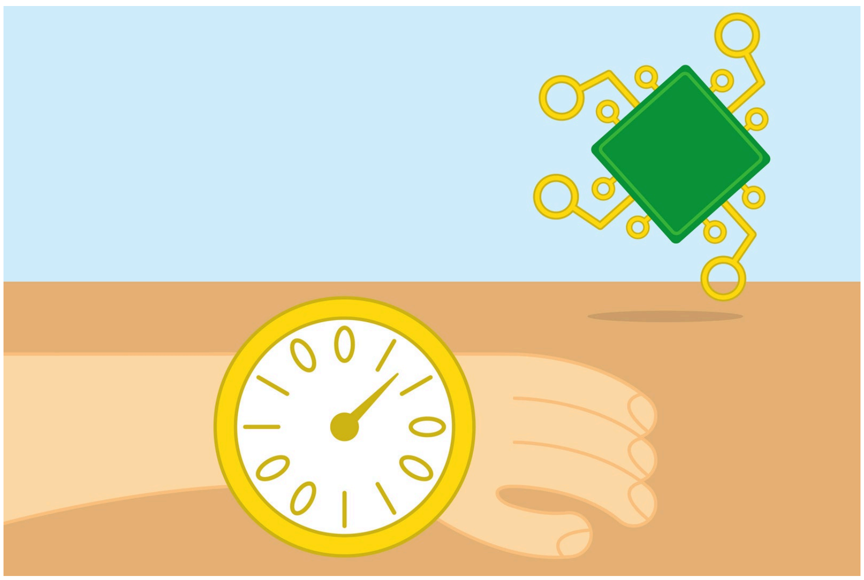
Computing

- **New Proof Dramatically Compresses Space Needed for Computation**
  Surprising new work bucks 50 years of assumptions about the trade-offs between computation space and time

New Proof Dramatically Compresses Space Needed for Computation

Surprising new work bucks 50 years of assumptions about the trade-offs between computation space and time

By Max Springer edited by Sarah Lewin Frasier



Thomas Fuchs

Once upon a time computers filled entire rooms, reading numbers from spinning tapes and churning them through wires to do chains of basic arithmetic. Today they slip into our pockets, performing in a tiny fraction of a second what used to take hours. But after decades of shrinking chips to pack as much computation as possible onto a machine, theorists are flipping the question: How little space is enough to get the job done?

This inquiry lies at the heart of computational complexity, a measure of the limits of what problems can be solved and at what cost in time and space. For nearly 50 years theorists could prove only that if solving a problem takes *t* steps, it should be possible using roughly *t* bits of memory—the 0s and 1s that a machine uses to record information. (Technically, that equation also incorporates log(t), but for the numbers involved this has little effect.) If a task requires 100 times the steps of another one, say, you'd expect to need about 100 times the bits, enough to diligently note each step. Using fewer bits was thought to require more steps—like alphabetizing books by swapping them one by one on the shelf instead of pulling them all out and reshelving them. But in a finding described at the ACM Symposium on Theory of Computing in Prague, Massachusetts Institute of Technology computer scientist Ryan Williams found a way to demonstrate that any problem solvable in time t needs only about $\sqrt{t}$ bits of memory: a computation requiring 100 times the steps could be compressed and solved with something on the order of 10 times more bits. "This result shows the prior intuition is completely false," Williams says. "I thought something must be wrong [with the proof] because this is extremely unexpected."

The breakthrough relies on a "reduction," a means of transforming one problem into another that may seem unrelated but is mathematically equivalent. With reductions, packing a suitcase maps onto determining a monthly budget: the size of your suitcase represents your total budget, pieces of clothing correspond to potential expenses, and carefully deciding which clothes can fit is like allocating your budget. Solving one problem would then directly solve the other. This idea is at the core of Williams's result: any problem can be transformed into one you can solve by cleverly reusing space, deftly cramming the necessary information into just a square-root number of bits. Thus, the original problem must be solvable with this compact container.

"This progress is unbelievable," says Mahdi Cheraghchi, a computer scientist at the University of Michigan. "Before this result, there were problems you could solve in a certain amount of time, but many thought you couldn't do so with such little space." Williams's finding, he adds, is "a step in the right direction that we didn't know how to take."

While computers have continued to shrink, our theoretical understanding of their efficiency has exploded, suggesting that the real constraint is not how much memory we have but how wisely we use it.

Max Springer is a Ph.D. candidate in applied mathematics at the University of Maryland and was a 2024 AAAS Mass Media Fellow at *Scientific American*.

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https://www.scientificamerican.com/article/new-proof-dramatically-compresses-space-needed-for-computation

Cosmology

- **Cosmic Tornado from Star's Birth Whirls in Dazzling JWST Image**
  This telescope has revealed the whipped-up dust from the birth of a star—and a shining background galaxy—more clearly than ever before

Cosmic Tornado from Star's Birth Whirls in Dazzling JWST Image

This telescope has revealed the whipped-up dust from the birth of a star—and a shining background galaxy—more clearly than ever before

By Gayoung Lee edited by Sarah Lewin Frasier

NASA/ESA/CSA/STScI

When a star is born, the process leaves behind a flurry of high-energy gas, dust and debris. Some of this remnant material clumps together into planets, the way Earth likely formed. Others end up floating endlessly as meteors and space dust. But when conditions are just right, powerful plasma jets blasting out of a young star whip some of the debris into a giant, helical tower of steamy-looking cosmic dust—one of which we now can see better than ever before, thanks to the James Webb Space Telescope (JWST).

Astronomers had long been aware of these so-called Herbig-Haro objects—brilliant flares of ionized gas, often near newborn stars, that can be light-years long—including one named HH 49/50, whose characteristic shape led to its nickname of "cosmic tornado." This object shines in the Chamaeleon I Cloud complex 625 light-years from Earth. Back in 2006, when HH 49/50 was first spotted by the now decommissioned Spitzer Space Telescope, astronomers could make out only an out-of-focus (albeit recognizably helical) lump of heated gas and dust with something shining at its tip. Although it was an exciting discovery at the time, the image's low resolution left the situation blurry.

Now, with the much bigger JWST, the full picture snaps into focus: the telescope captured this field of dust and debris just as a baby protostar (probably located somewhere on its lower right, outside the boundary of the image shown here) was blasting it into this very particular shape. The fuzzy blob at the top resolves into a distant spiral galaxy unrelated to the object itself. Its apparent position atop this ongoing event is just a quirk of our perspective.

This view, and the great distance, also creates a few other optical illusions, says Macarena Garcia Marin, an astrophysicist at the European Space Agency who was part of the team that took the new image. For example, the smaller dots appearing to float in front of the cosmic tornado aren't dust; they're actually entire galaxies shining through from behind it. The pointy dots are lone stars.

Still, the chance alignment of these cosmic entities lets scientists study a rich array of extraterrestrial phenomena, says Melissa McClure, an astronomer at Leiden University in the Netherlands who was not on the imaging team. Notably, we can see processes such as accretion in action, she says—“And the image is just gorgeous!”

Garcia Marin is particularly struck by the JWST picture’s ephemeral nature, at least on cosmic scales. When the protostar eventually grows up, most likely beyond our lifetimes, the jets it produces and the accompanying cosmic tornado will fade, Garcia Marin says: “You’re looking at a snapshot of a moment in the universe.”

Gayoung Lee is a science journalist and former news intern and Games ace at *Scientific American*. A philosopher turned journalist, originally from South Korea, Lee is interested finding unexpected connections between life and different science, particularly in theoretical physics and mathematics. You can read more about her here: https://gayoung-lee.carrd.co

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https://www.scientificamerican.com/article/cosmic-tornado-from-stars-birth-whirls-in-dazzling-jwst-image

Culture

180 Years of Standing Up for Science

Our anniversary celebration begins with an outstanding collection of stories about times that science itself has made a full about-face

By David M. Ewalt

Scientific American, September 2025

I have been a *Scientific American* subscriber since I was 12 years old. I think if I could ask that kid if he expected to see his name in the magazine one day, he'd say yes, but he would be surprised to find it on this particular page and not in one of the stories of great discoveries that follow. He wanted to grow up to be an astrobiologist and was certain the SETI program would discover an extraterrestrial radio signal soon; the only doubt he would have had was whether he'd be lead scientist in the subsequent *SciAm* article or only a junior member of the team.

Of course, SETI is still searching, and my name is on this page, not among the world's great scientists. But I could not be more thrilled and honored to introduce myself as *Scientific American*'s new editor in chief.

I was lucky to grow up in a time when science was celebrated and great communicators told American children that science was not only a worthy career but also exciting and cool. I could turn the television to PBS and watch Carl Sagan in Cosmos or Alan Alda in *Scientific American* Frontiers; flip it to the news, and I'd see space shuttle liftoffs, high-temperature superconductors and the launch of the Human Genome Project.

Unfortunately, the world of today is increasingly hostile to science. Our leaders deny the existence of human-driven climate change and vilify vaccines that have saved hundreds of millions of lives. Schools lack funding for STEM courses, and budgets are slashed at leading research institutions such as the NIH, NASA and NOAA. Earlier this year I spoke to a top-of-the-class postdoc who'd given up on the idea of securing a position at their U.S. alma mater and instead was weighing offers to relocate to Europe or China.

That makes *Scientific American* more important than ever. In our very first issue, dated August 28, 1845, editor Rufus Porter offered the publication as an advocate of science and industry and promised it would "instruct while it diverts or amuses." My goal as editor in chief is to uphold those values, to stand up for science, to promote its application in industry, and to help educate and inspire new generations of young scientists. I hope to ensure that our coverage is centered on meaningful research and discovery and that it supports working scientists at a time when the value of science itself too often goes unrecognized.

Given this context, I'm particularly excited that my first issue as editor in chief marks the 180th anniversary of *Scientific American*. Our celebration begins with an outstanding collection of stories about times when science itself has made 180-degree turns, including by abandoning ideas such as that light is a wave traveling through “aether,” that nerves don’t regenerate, and that the expansion of the universe is slowing. The anniversary party continues online, so don’t forget to check out our website at sciam.com over the course of the coming months, too.

In our cover story, journalist Maryn McKenna looks at the perplexing world of peanut allergies. Food reactions are probably as old as time, and researchers now estimate that they affect one out of 10 Americans overall and two kids in every classroom. But peanut allergies didn’t become widespread until the 1990s. Thankfully, remarkable new treatments might free millions of people from the deadly threat of anaphylaxis or even end food sensitivities once and for all. It’s an extraordinary example of the way that science can make the world a better place. And that’s the perfect way to kick off our next 180 years.

I’m glad that I have this soapbox to stand on at a time when our nation hungers for meaningful discovery and the scientific community needs everyone’s support. I hope you’ll stand with me. *Scientific American* helped shape the way I look at the world. It has always educated and delighted me, and I suspect it’s done the same for you, too. I’m looking forward to exploring the future together.

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

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https://www.scientificamerican.com/article/180-years-of-standing-up-for-science

Contributors to *Scientific American*’s September 2025 Issue

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

By Jen Schwartz

Andrew B. Myers

Andrew B. Myers
Peanut Proof

Photographer Andrew B. Myers (*above*), who shot this month’s cover story on peanut allergies by writer Maryn McKenna, likes the constraint of creating big worlds at small scale. What did Myers seek in the ideal peanut model? “You look for the very basic quality of a peanut, this eight shape with an hourglass curve,” he says. “But the curve can’t be so basic it looks fake. You want 90 percent perfect peanut and 10 percent little quirk. Just like human attractiveness.” By giving his subject a halolike light, Myers sought to make a singular, tiny peanut “feel ridiculously heroic.” Manipulating peanut butter for the shoot was less satisfying. “It’s kind of a gross, difficult substance to work with.”

Myers takes a layered and “zany” approach to making still-life images and describes himself as more of a sculptor and designer than a photographer. “I care a lot about building the frame and mixing processes,” he says. “I make things in a controlled, quiet setting with a camera on a tripod. I can’t remember the last time I held a camera in my hands.”

Myers, who has worked for a range of editorial and commercial clients, has an affinity for shooting scientific concepts in a clever, unexpected way. He’s inspired by the imagery that comes out of the lab of his spouse, who is a computational neuroscientist. “I like when scientists and artists get together,” he says. “Scientists are much more humble than your average artist, but both look outward and have a rock of curiosity.”

David Cheney
Brain Washing

David Cheney is no mere artist—he’s a board-certified medical illustrator. In the Johns Hopkins University program where Cheney got his master’s degree, the artists study right alongside the medical students. So when *Scientific American* asked Cheney to render cerebrospinal fluid entering and exiting the brain for a feature by journalist Lydia Denworth on how the organ cleans waste during sleep, he already had a strong understanding of the anatomy.

Cheney filled stacks of sketchbooks as a kid but assumed he’d end up premed in college. He experimented with different paths (for a time, he was a musical theater major) until he learned about a career in medical illustration. It was an instant and lasting fit. “The field might be niche," he says, "but it's so varied in terms of what you can do with the training."

He's worked for medical clinics, academic institutions, and even a tech startup where he's designing "an entire race of aliens" for a cryptocurrency game. Cheney would like to do more sculpture, specifically reconstructing "some extinct type of creature" for a natural history museum. "I wish more young artists who love science knew about this field where you can truly use both sides of your brain."

Dava Sobel
Meter

When writer Dava Sobel learned that the earliest issues of *Scientific American* included poetry, she wanted to bring that tradition back to the magazine. Her pitch was to publish existing poems about science; instead the editors tasked her with soliciting original work. Sobel first approached poets she knew—Diane Ackerman was the inaugural contributor to the Meter column in January 2020—and then "the flood began," she says. "The backlog of submissions is now yearslong." Sobel doesn't write poetry herself, but her long career as a science journalist and author has often involved "unearthing people's letters, showing scientists as the real people they are." Her first big success, she says, was her 1995 book *Longitude*, "which allowed me to write all the others." One of the best fun facts about Sobel is she served on the Planet Definition Committee that redefined the term in 2006—an endeavor that ultimately led to Pluto losing its status as a planet. That move "was not our recommendation!"

As the Meter editor, Sobel looks for poems that "cause an emotional leap in me." Other times she'll choose a poem because "it attempts a tremendous challenge—and works." Meter hopefuls take note: Sobel has a limit on limericks but likes to publish at least one humorous poem every year. “I’m the first to admit it’s totally subjective, and contributors are totally at my mercy.”

Charles C. Mann
Research in Reverse

When we asked author Charles C. Mann to write an essay about dramatic twists and turns in science, Mann, fortuitously, was already mulling the subject. “I write to try to figure out what I think,” he says. He teased apart genuine 180s— “when assumptions baked into a discipline turn out not to be right after someone gives them a hard look”—from a fraught kind of pivot, “when the normal back-and-forth of science gets pinned by people who make definitive proclamations based on exaggerated evidence.”

For someone who wrote a book (entitled *1491*) that rethinks the environmental history of an entire continent, Mann isn’t sure he’s any better at coping with uncertainty than the rest of us. “But I would say I’m comfortable admitting that chance plays a huge role in what happens to me.” Sometimes, while working on a project, he gets “distracted by worrying about if I actually know what I’m talking about.” The research discursions that follow often lead to satisfying revelations. “It’s good to be aware of one’s own fallibility,” he says.

Mann has seemingly lost track of how many books he’s written (“I don’t know, nine?”), but his next one, about the North American West, will be published in 2026.

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.

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https://www.scientificamerican.com/article/contributors-to-scientific-americans-september-2025-issue

Readers Respond to the April 2025 Issue

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

By Aaron Shattuck

Scientific American, April 2025

NEURODIVERSITIES

In “A Complex Diagnosis,” journalist Paul Marino writes about his experience with a type of complex motor stereotypy, a neurological condition that involves recurring involuntary movements.

As I read the article, my hands began to move in excited recognition, each tapping an independent part of a polyrhythmic pattern, my equivalent of Marino’s motor stereotypy. I am autistic. Many in our community engage in what are called stims, which can sometimes be more physically dramatic than either Marino’s or my movements and are often consciously suppressed.

Some recent evidence appears to link these kinds of neurodiversities with a lack of synaptic pruning. The majority of people grow synaptic connections at a fast rate when very young, yet as they mature, their brain eliminates “unneeded” connections. That may not happen as much for the neurodiverse (ND). It’s possible this is why ND folks often have more neurological “noise” but may also make connections and detect patterns when no one else does. If that’s the case, it might not be a coincidence that creative fields have a disproportionate share of ND people.

The fact that up to 20 percent of the global population is ND indicates that nature considers us valuable to the human species. Many of the “disabilities” we ND people experience are really an inability to conform to arbitrary societal norms. Science needs to stop treating us as defective. These traits are indeed phenomena, not disorders.

MICHAEL A. LEVINE *VIA E-MAIL*

FIT TO BE TIED?

“Are You a Good Judge of Knot Strength?” by Clara Moskowitz and Jen Christiansen [Graphic Science; March], concerns different types of knots and humans’ inadequate ability to assess their strength.

Perhaps humans haven’t learned to judge the strength of a knot because they intuitively know it’s much more dependent on what the rope is made of. Having tied hundreds of sutures in my career as a dentist, I can tell you that gut sutures will practically untie themselves (unless properly tied), whereas silk sutures will hold fast with practically any knot. Similarly, a nylon rope will slip easily, but a jute or hemp rope will not.

TOM N. TODD *OKLAHOMA CITY, OKLA.*

THOUGHT AND LANGUAGE

In “Thinking without Words” [Q&A March], by Gary Stix, neuroscientist Evelina Fedorenko comments on the ability of large language models (or LLMs) to produce language without thought.

But I disagree with the notion that what LLMs do is parallel to the way humans learn language. Such artificial-intelligence models ingest enormous amounts of material and produce their “language” based on statistics: which word is the “most likely” one to use following a given word, phrase or sentence.

There is no intelligence behind that language. Children do not learn that way. There is much more selection in what they hear. And their acquisition of language, while subject to trial and error, is strongly influenced by the desire for specific results. This desire is entirely absent from artificial intelligence—at least so far.

In other words, the processes by which LLMs and children learn language, and the feedback systems by which each gets better and better at language, are different at their cores.

I don’t think, as Fedorenko suggests, that AI tools can help us examine the question of how a system of thought can interact with a separate system that stores and uses linguistic expression, because there is no reasoning behind the language abilities of LLMs.

TONY STEIN *VIA E-MAIL*

EXPRESSIVE FACES

In “Why People Like Expressive Faces” [Mind Matters], Eithne Kavanagh, Jamie Whitehouse and Bridget Waller wonder why some people remain “comparatively inexpressive across situations.” They speculate that this could be some kind of cost-benefit or alternative strategy.

For some people, facial expressions just don’t come naturally. As an autistic person, I’ve practiced them in front of a mirror and tried to learn when they apply. It’s exhausting, feels fake and is often perceived as fake.

GEORGE WIMAN *VIA E-MAIL*

TOWERING TASK

In the Tower of Hanoi puzzle described by Heinrich Hemme in “Move the Tower” [Advances; January], it is easy to see that for low values of *n*, moving a tower with *n* disks will take $2^n - 1$ moves. A nice challenge for the reader is to prove this for all values of *n*. A fairly simple recursive proof will do it.

Given the result of $2^{64} - 1$, which is greater than 10^{19}, moving a 64-level tower with one move per second would take more than 580 billion years. The monks were quite right that the end of the world would come before they finished the task!

JAMES R. PAULSON *PROFESSOR OF CHEMISTRY EMERITUS, UNIVERSITY OF WISCONSIN–OSHKOSH*

CLARIFICATION

“Refreezing the Arctic,” by Alec Luhn [June], said that the Bright Ice Initiative had scattered tiny glass beads on glaciers in Iceland and India to reflect sunlight. The organization initially used that approach in a field test in Iceland. It subsequently switched to a clay-based material, which it used in a field test in the Himalayas.

ERRATA

“Language on the Mind,” by Gayoung Lee [Advances; May], should have said that Cas W. Coopmans explained that verbs sometimes come near the end of a sentence in Dutch language structure. And it should have included the word “because” at the beginning of the example “I ate a cookie with chocolate.” The question for 41-Down in the Science Crossword also should have included “because” in that example.

“Cosmic Dawn,” by Rebecca Boyle [June], should have said that sometime between 50 million and 100 million years after the big bang, gravity drew hydrogen atoms together and ignited the first stars.

In “Pay Dirt,” by Elizabeth Anne Brown [July/August], the first image caption misspelled the name of Marie Aagaard Larsen.

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

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https://www.scientificamerican.com/article/readers-respond-to-the-april-2025-issue

Diet

- ## How Gluten-Free Diets Are Getting Overhyped
  Unless you have celiac disease, there are few health benefits to a gluten-free diet

Should You Be on a Gluten-Free Diet?

Unless you have celiac disease, there are few health benefits to a gluten-free diet

By Lydia Denworth edited by Josh Fischman

Jay Bendt

This article was made possible by the support of Yakult and produced independently by Scientific American*'s board of editors.*

Recently a friend I'll call Anne told me she had cut gluten out of her diet to try to reduce joint pain in her hands. "I feel so much better," she said. Anne is just one of many people who have self-prescribed such a diet, avoiding wheat because gluten is the primary nourishment protein in the developing plant. Usually people do this after hearing—anecdotally or from social media—that gluten is inflammatory and at the root of a range of physical and mental problems.

Anne's story gave me pause. I have been known to joke that I am on an all-gluten diet because I enjoy bread and baked goods so much. But she and I are the same age, and I, too, have joint pain in my hands. Might that improve if I gave up gluten?

This is a question of considerable medical importance. For the roughly 1 percent of the population with celiac disease, gluten is clearly the problem. In celiac disease, white blood cells in the immune system regard gluten as a foreign invader, like a bacterium, and start attacking. The pain can be intense. “The intestinal cells get damaged, and that leads to intestinal malfunction,” says Benjamin Lebwohl, a gastroenterologist and researcher at Columbia University’s Celiac Disease Center.

Celiac, which is linked to immune system genes called HLAs, causes a range of gut-related symptoms, including stomach pain, diarrhea and constipation. The ability to absorb nutrients goes down, leading to weight loss. There can also be damage beyond the gut such as brain fog, joint pain and infertility. No wonder people struggling with the disease have become so worried about gluten.

More general concerns about gluten causing inflammation appear misguided, however. In most people without celiac disease, eating gluten does not cause digestive problems. Nor has it been found to activate inflammatory markers in the gut. The inaccurate idea that gluten is inflammatory “is largely promoted via social media, [which] often escalates misconceptions and misinformation,” says Elena Nikiphorou, a rheumatologist at King’s College Hospital in London.

Still, not everyone without celiac disease can easily digest wheat. Some have been diagnosed with nonceliac gluten sensitivity. When they stop eating gluten, they say they feel better. Why that should be is largely a mystery. “There’s very little we really understand about why gluten makes people feel ill outside of the world of celiac disease,” Lebwohl says.

In some cases, people do have celiac but haven't been properly diagnosed. It's important to rule the illness out, Lebwohl says. Patients should also rule out wheat allergies. There's a new test in which researchers can use a tiny microscope in the intestines to watch as mucosal cells in the gut are challenged by possible allergens. It has revealed a subtle type of reaction—so-called type two allergies. Unlike acute allergic responses that may require immediate medical intervention, type two allergies trigger slower responses in the body, over hours rather than minutes.

But most of the latest research suggests that other components in wheat are causing the symptoms people mistakenly ascribe to gluten. The condition should be called nonceliac *wheat* sensitivity, not gluten sensitivity, says Detlef Schuppan, a gastroenterologist and immunologist at the University of Mainz School of Medicine in Germany.

Schuppan and others have shown repeatedly that other wheat proteins, amylase trypsin inhibitors (ATIs), cause autoimmune responses and intestinal inflammation in mice and in humans. "These were the innate immunity triggers, not the gluten itself," Schuppan says. ATIs could be part of the problem in celiac disease, in wheat sensitivity and in some cases of irritable bowel syndrome.

Researchers have investigated the role of FODMAPs, a large group of short-chain carbohydrates, including wheat and dairy molecules that can be hard to digest. If someone has an issue with FODMAPs, cutting out wheat alone can seem like a solution. Still, one study comparing a reduced-FODMAP diet with a gluten-free diet found the latter more effective at reducing many symptoms.

As for joint pain, there may be shared underlying processes between rheumatoid arthritis (RA) and celiac disease. Some studies have found limited improvement of inflammatory joint symptoms in rheumatoid arthritis patients who stop eating gluten and some other suspect foods. But Nikiphorou emphasizes that most of the research has limitations such as a short testing duration, and experts aren't yet convinced by the evidence of a dietary treatment for RA.

The most common source of joint pain in people older than 40 years is, in fact, not RA but osteoarthritis (OA), which develops from wear and tear on joints and not from an autoimmune response. "I would think it unlikely for gluten to impact OA," Nikiphorou says. (ATIs could be the culprit in someone with joint pain, Schuppan suggests.)

Adopting a gluten-free diet unnecessarily can bring risks. Such diets are often low in fiber, which can increase the risk of heart disease, according to Lebwohl and his colleagues. Many gluten-free products are also lacking in nutrients and are ultraprocessed, which recent research shows to be unhealthy.

Experts emphasize that only people with true celiac disease need to avoid gluten from all sources. People with type two wheat allergies or an ATI sensitivity can improve symptoms by reducing consumption by 80 to 90 percent, Schuppan says.

That's an approach I might consider ... after I make a sandwich for lunch.

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.

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https://www.scientificamerican.com/article/how-gluten-free-diets-are-getting-overhyped

Education

- **Public Education Needs Our Support**
  The U.S. is a global powerhouse. Public education is one of the main reasons why

Investing in Public Education Will Strengthen the U.S.

The U.S. is a global powerhouse. Public education is one of the main reasons why

By The Editors

Martin Gee

Public education in the U.S. is under attack. Whether at the local, state or federal level, political and religious groups have pushed for funding cuts while diverting more money to private-school vouchers, trying to alter curricula and removing books from children's reading lists. By not prioritizing free and equitable public education, the U.S. government is robbing our youths of the critical-thinking skills and knowledge that drive innovation and democracy.

These efforts fall heavily on science education in our classrooms, if not directly on classwork, then on its fundamental drivers—curiosity, imagination, ingenuity and innovation. To ask the kinds of questions of our natural world that would produce such things as artificial intelligence, spacecraft, medicines, and more, children need exposure to the ideas that have shaped our progress as a society, the status quo we have bucked against to bring about great changes for humanity, the declarations we have questioned and then reshaped.

The attempt to quell and control taxpayer-funded education is antithetical to a society that values evidence and knowledge. It's a concerted effort in thought control, racism, classism and sexism. It's not very democratic—or very smart.

About 50 million children in the U.S. attend public school, according to the National Center for Education Statistics. That number was closer to 51 million in 2019, but many kids who left public school during the COVID pandemic didn't come back to public education. Children attending public school exercise a right that used to be a privilege, as schooling in the U.S. was generally tuition-based until the mid- to late 1800s. By 1870, about 78 percent of children ages five to 14 were enrolled in taxpayer-funded schools. In 2019 and 2020, between 80 and 90 percent of children who went to public school in the U.S. graduated from high school, depending on the locale.

> Critical thinking and exposure to different ideas are fundamentals of not only democracy but also creativity and innovation.

Some of the biggest battles about the right to comprehensive knowledge have been waged in public schools. They include the fight over the ability to teach evolution at Rhea County High School in Tennessee, which was at the center of the Scopes Trial 100 years ago, and clashes over the inclusion of climate change science in textbooks that serve millions of public school students in Texas and elsewhere. School districts nationwide have removed school library books that contain information on changing bodies or that explore mental health, not to mention ones that discuss slavery, race and gender identity. Under the guise of protecting children from harm, censors instead seek to whitewash the inconvenient truths that make it harder for them to maintain their profiteering and social hegemony: Earth is warming, and humans are responsible; slavery did happen; neither race nor gender is hierarchical.

Among the most egregious examples of the drive to undermine public education are school voucher programs. These efforts funnel taxpayer dollars to private and parochial schools, frequently at the expense of the long-term funding of public education. Often sold as “school choice,” these legislative initiatives are championed as a way to help students escape poorly performing public schools or to give families of lesser means more options in education. But problems abound.

Arizona is hemorrhaging money to keep its voucher program afloat. In Indiana, educational gains in voucher-eligible schools are debatable. Joseph Waddington, an education researcher at the University of Notre Dame, says his and others’ examination of Indiana’s program showed that when children initially transitioned to private schools, their math scores fell significantly. It took a while for them to rebound. The researchers found no difference in English scores. The idea in some corners has been that voucher programs will stimulate the development of more religious or for-profit schools, which would, of course, enrich the entities opening the schools. But in many rural areas, there are no such schools. Many Texas counties have no private option. This lack was the basis for one of the bigger criticisms of Texas’s new voucher program, passed during the state’s January 2025 legislative session. Such examples beg the question of why these funds shouldn’t just be used for public education that everyone can benefit from.

Critical thinking and exposure to different ideas are fundamentals of not only democracy but also creativity and innovation. For the U.S. to maintain its status as an economic powerhouse and driver of the global economy, we need problem-solvers, inventors, iterative thinkers and people who view failure as part of progress. This is the realm of science and mathematics, the realm of history and geography, the realm of a broad-based and well-rounded education. Diverting funds from public education while stifling certain ideas in public schools would certainly diminish our footprint in the world.

And although public education in the U.S. is a local and state issue, federal support does matter. Efforts to dismantle the Department of Education, which helps students with disabilities, gives grants and funds to equalize educational opportunity, and carries out research on different aspects of education, leave students at every level in the lurch, especially in less affluent school districts. Schooling may be local, but national support is critical.

What does appropriate funding for public education look like? Higher teacher salaries. Better buildings, not just stadiums. More reliable transportation. More comprehensive, influence-free textbooks. Better laboratories. More subject options. Better training in trades. More preschool. Mental health services. Physical health services. More nutritious meals. Better and free after-school programs.

The idea that we can defund public education in favor of alternatives belies reality and common sense. Public education provides community, refuge and opportunity. It is a common good that we must nurture. The U.S. became a world leader thanks in no small part to universal, standard public education. We owe it to future generations to keep it that way.

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https://www.scientificamerican.com/article/public-education-needs-our-support

Food

- **This Mushroom's Incredibly Bitter Taste Is New to Science**
  The first analysis of mushroom bitterness reveals ultrapotent compounds

This Mushroom’s Incredibly Bitter Taste Is New to Science

The first analysis of mushroom bitterness reveals ultrapotent compounds

By K. R. Callaway edited by Sarah Lewin Frasier

Alexander Kurlovich/Alamy Stock Photo

Ever bite into something so bitter that you had to spit it out? An ages-old genetic mutation helps you and other animals perceive bitterness and thus avoid toxins associated with it. But while most creatures instinctively spit first and ask questions later, molecular biologists have been trying to get a taste of what bitterness can tell us about sensory evolution and human physiology. A new study, published in the *Journal of Agricultural and Food Chemistry*, is the first analysis of how taste receptors respond to a mushroom’s bitter compounds—which include some of the most potently bitter flavors currently known to science.

The bitter bracket mushroom is nontoxic but considered inedible because of its taste. Researchers extracted its bitter compounds, finding two familiar ones—and three that were previously unknown. Instead of tasting these substances themselves, the scientists introduced them to an “artificial tongue” that they made by inserting human taste receptors into fast-growing embryonic kidney cells. One of the newfound bitter substances activated the taste receptors even at the lowest concentration measured, 63.3 micrograms per liter. That’s like sensing three quarters of a cup of sugar in an Olympic-sized swimming pool.

Humans have about 25 kinds of bitter taste receptors lining our mouths and throats, but these same receptors also grow throughout the body—in the lungs, digestive tract and even brain. Despite their ubiquity, they have been only partially explored. Four of our bitter receptors have no known natural activator. Finding activating compounds could illuminate the interactions that might have shaped those taste receptors’ evolution, says study lead author Maik Behrens, a molecular biologist at the Leibniz Institute for Food Systems Biology.

Previous research focused on bitter compounds from flowering plants, which evolved well after animals gained bitter taste receptors. Behrens thought that mushrooms, being older, might even activate one of the four mystery receptors. The bitter bracket mushroom didn’t, but Behrens plans to keep looking—especially since this first chemical analysis of mushroom bitterness has already yielded previously unknown compounds.

Such research can also unlock information about taste receptors’ many functions in the human body. “Taste in your mouth does so much more than just perception,” explains University of Miami physiologist Nirupa Chaudhari, who was not involved in the study. Taste can trigger physiological reflexes such as insulin release and stomach acid production, she says, so knowing what activates bitter taste receptors could improve our understanding of bodily processes and disease. Chaudhari considers the new study a good first step toward expanding bitter taste research.

With the first analysis complete, researchers are now setting their sights on other mushrooms' bitter secrets—compounds and activated receptors you can't uncover by "simply chewing on a mushroom," Behrens says.

K. R. Callaway is a freelance journalist specializing in science, health, history and policy.

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https://www.scientificamerican.com/article/this-mushrooms-incredibly-bitter-taste-is-new-to-science

Geology

- **North America May Be Dripping Away Deep under the Midwest**
  A long-lost slab of Earth’s crust may be pulling away the bottom of the oldest part of North America, scientists say

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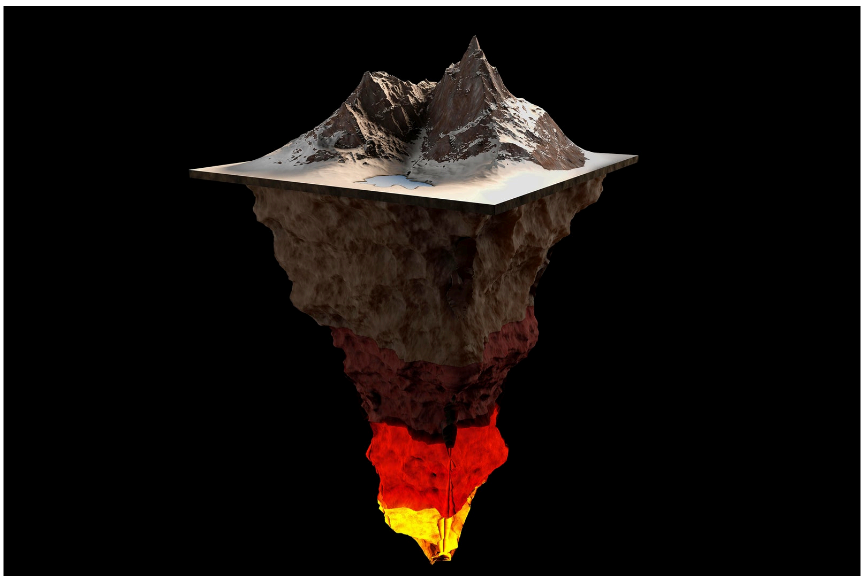
North America May Be Dripping Away Deep under the Midwest

A long-lost slab of Earth's crust may be pulling away the bottom of the oldest part of North America, scientists say

By Meghan Bartels edited by Andrea Thompson & Sarah Lewin Frasier



Victor Josan/Alamy Stock Photo

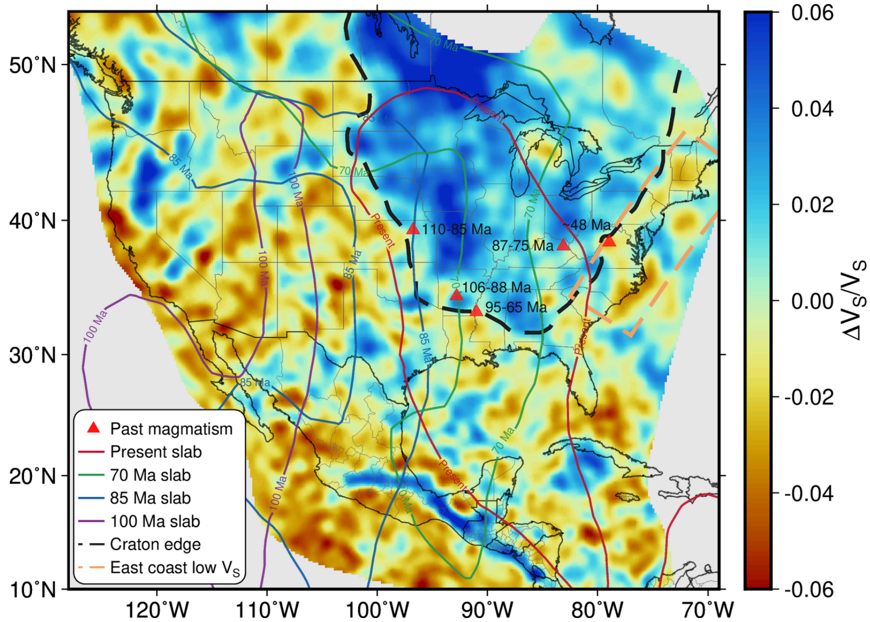
Something very strange appears to be happening deep, deep underneath the U.S. Midwest and the Ohio Valley.

North America's geological core has persisted for more than a billion years; it's what scientists call a craton, a massive block of continental rock that withstands the natural recycling system of plate tectonics. Typically scientists think of cratons as unchanging, nigh eternal. But research published in *Nature Geoscience* suggests that a long-lost geological plate may be siphoning molten rock from the underside of the North American craton and eroding it from below, right under our feet.

Such a scenario would not be unprecedented—scientists have evidence that the North China craton thinned dramatically millions of years ago—but it would certainly be surprising and intriguing to study in real time. “Cratons are the oldest cores of continents, so they have been sitting near Earth’s surface for billions of years,” says Claire Currie, a geophysicist at the University of Alberta, who was not involved in the new research. “They’ve persisted through time, so this is quite unusual.”

The scientists behind the recent research had no intention of finding an eroding craton, according to study lead author Junlin Hua, a geophysicist at the University of Science and Technology of China. The team merely wanted to apply a new, more precise analysis technique to the data gathered by North America’s rich network of more than 6,000 seismometers, in the hope of seeing the continent in more detail than ever before.

The work relied on the seismometer network’s observations of more than 200 earthquakes, each of which produced multiple types of seismic waves. These waves are affected in specific ways by changes in the material they pass through—for example, there are distinctive effects when that material is particularly cold or warm or is strong or weak. By analyzing the waves, scientists can reverse engineer a map of Earth’s innards, Hua says. And the researchers sought to conduct this work in a way that would account for every wiggle in the trove of seismic data, a laborious process.



A map produced by the authors of a new study in *Nature Geoscience* shows the relative seismic velocity of material that is 200 kilometers below Earth's surface and located around the base of the North American craton. Cratons are characterized by high seismic velocity. On this map, blue represents rock through which seismic waves travel faster; red represents rock through which seismic waves travel more slowly. The black dashed line outlines the borders of the North American craton. "Seismic Full-Waveform Tomography of Active Cratonic Thinning beneath North America Consistent with Slab-Induced Dripping," by Junlin Hua et al., *Nature Geoscience*, Vol. 18. Published online March 28, 2025.

It was months into the analysis when Hua started to recognize that the investigation was turning up something surprising. The craton itself looked normal enough: a slab of dense rock, about 200 kilometers thick, through which seismic waves traveled relatively fast—what scientists refer to as high-seismic-velocity material—that abruptly transitioned to material with lower seismic velocity as the craton gave way to younger rock.

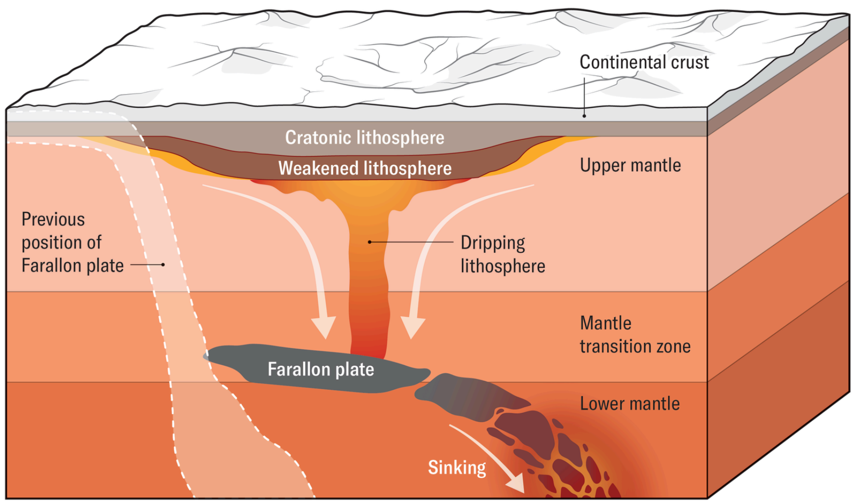
But below part of the craton, underneath much of the Midwest and the Ohio Valley, something strange was happening. Here a patchy pattern of molten material with that same high seismic velocity sagged to a depth of nearly 600 kilometers, almost to the lower mantle. Those measurements, Hua says, suggest that in this area North America's craton is dripping downward into the mantle in a way he and his colleagues didn't expect and couldn't quite explain.

What got them unstuck was considering a relic of geological history that lies hidden under North America: remains of the

Farallon plate, an oceanic plate that stretched between the Pacific and North American plates some 100 million years ago, when the dinosaurs were at their peak. Most of the Farallon plate was eventually shoved under North America. Its remnants linger in the lower mantle, some 800 kilometers below Earth's surface, and indeed showed up in the cross sections of seismic velocities that Hua and his colleagues made.

A Look Below the Surface

The Farallon plate is sinking down through Earth's lower mantle. According to a new theory, cratonic material is dripping down through a relatively narrow area, fed by horizontal movement at the base of the entire craton.



ELabArts; Source: "Seismic Full-Waveform Tomography of Active Cratonic Thinning beneath North America Consistent with Slab-Induced Dripping," by Junlin Hua et al., in *Nature Geoscience*, Vol. 18; April 2025 (*reference*)

When the scientists used computer modeling to test theories of what could pull cratonic material downward, the Farallon slab was key: drips formed only when the slab was incorporated into the model. Hua calls the slab "a big sinker" that pulls material off the craton and down into Earth.

Right now, Currie says, this is just a hypothesis—but one that she calls "intriguing." Currie would like to see other signals of the cratonic drip—for example, is the surface of Earth being pulled down at all over this region? She would also like to see stronger explanations for how the craton gets drawn down into material denser than it is; it ought to float above such material.

Still, the research is a surprising glimpse of activity in an environment that scientists have long considered unchanging, Hua says. “The continent is not something static,” he says. “It has a dynamic component.”

Meghan Bartels is a science journalist based in New York City. She joined *Scientific American* in 2023 and is now a senior news reporter there. Previously, she spent more than four years as a writer and editor at Space.com, as well as nearly a year as a science reporter at *Newsweek*, where she focused on space and Earth science. Her writing has also appeared in *Audubon*, *Nautilus*, *Astronomy* and *Smithsonian*, among other publications. She attended Georgetown University and earned a master’s degree in journalism at New York University’s Science, Health and Environmental Reporting Program.

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https://www.scientificamerican.com/article/north-america-may-be-dripping-away-deep-under-the-midwest

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History

- **September 2025: Science History from 50, 100 and 150 Years Ago**
  Huge fish; spiritualist rebuke

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

Huge fish; spiritualist rebuke

By Mark Fischetti

1975, Sun Loops: "Loops on the sun are shown in a false-color picture made with the Harvard College Observatory ultraviolet spectroheliograph aboard the Skylab crewed orbiting satellite. The loops, which are part of the inner corona, extend some 150,000 kilometers from the sun's western edge. Black and blue areas represent the least intense radiation, yellow and magenta the more intense, and red the most intense."
Scientific American, Vol. 233, No 3; September 1975

1975

Earth Fires Seeds into Space

“Imagine that the earth has been watched over the aeons by an extremely patient extraterrestrial observer. Nothing, save a little hydrogen and helium, leaves the planet. And then, less than 20 years ago, the planet suddenly begins, like a dandelion gone to seed, to fire tiny capsules throughout the inner solar system. First they go into orbit around the earth. Six capsules set down on the moon and from each two small organisms emerge. Five little spacecraft enter the hellhole of Venus’s atmosphere. More than a dozen are dispatched to Mars. Two spacecraft successfully traverse the asteroid belt, fly close to Jupiter and are ejected by its gravity into interstellar space.

It is clear, the observer might report, that something interesting is happening. We have entered, almost without noticing it, an age of exploration unparalleled since the Renaissance, when in just 30 years European people moved across the Western ocean to bring the entire globe within their ken. Our new ocean is the shallow disk of space occupied by the solar system. Centuries hence, our age may be remembered chiefly as the time when the inhabitants of the earth first made contact with the vast cosmos in which their small planet is embedded. —Carl Sagan”

1925

Television via Radio

“C. Francis Jenkins, radio photographic experimenter of Washington, D.C., has demonstrated apparatus by which moving objects, including a Dutch windmill and motion picture film, were sent by radio for five miles and reproduced on a miniature screen, 10 by eight inches. The transmitter was set up at station NOF, near

Anacostia, D.C., and the receiver in Jenkins's laboratory. He predicts that the process will be perfected so that scenes at baseball games and prize fights can be broadcast over long distances."

Clearly Written Books

"Below are some of the recent books that can be recommended for clearness of treatment, obtainable from the *Scientific American* Book Department.

Red-Lead and How to Use it in Paint, by Sabin
White-Lead. Its Use in Paint, by Sabin
The Science of Knitting, by Tompkins
Carbureting and Combustion in Alcohol Engines, by Sorel, Woodward, Preston
Evolution and Animal Intelligence, by Holmes
I Believe in God and in Evolution, by Keen
God or Gorilla, by McCann"

1875

Cincinnati is Center of U.S.

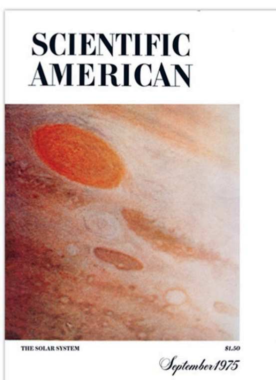
"The center of our population has traveled westward, keeping curiously near the 39th parallel of latitude, never getting more than 20 miles north or two miles south of it. In 80 years it has traveled only 400 miles, and it is now found nearly 50 miles eastward of Cincinnati, Ohio."

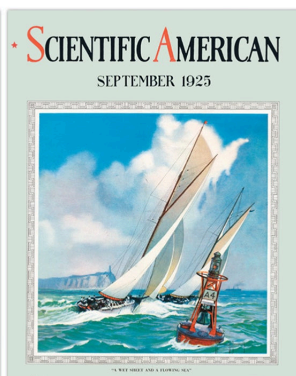
Spiritualist Rebuke

"Most of the organs of the spiritualists in this country are filled with insipid ghost matter, very tiresome and useless to all whose brains have not been softened by the spirit craze. The *Spiritual Scientist*, a weekly periodical, is an exception. Its editorial columns exhibit talent, while its conductors, with boldness, condemn as unworthy of true believers the printing of the unauthenticated trashy stuff delivered by common mediums. To its contemporary, the *Banner of Light*, it administers a severe rebuke for its agency in this matter, and alleges that for the past 10 or 12 years that journal has poured out a weekly stream of pretended spirit communications, of which not more than two in a hundred had contained anything beyond childish nonsense."

Huge Ganoids Ruled the Seas

"Professor J. S. Newberry gave descriptions of some newly discovered ancient fishes found in the rocks of Ohio. Among these was the entire bony structure of *Dinichthys terrelli*, the hugest of the old armor-plated ganoids. The dorsal shield weighed 30 pounds. Professor Newberry explained that the dipnoans of Africa and South America were descended from these ancient plated ganoids, and were the last remnants of a group of fishes which in the Devonian age not only ruled the seas, but were the most powerful and highly organized of living beings."





Mark Fischetti has been a senior editor at *Scientific American* for 19 years and covers sustainability issues, including climate, environment, energy, and more. He assigns and edits feature articles and news by journalists and scientists and also writes 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. Follow Fischetti on X (formerly Twitter) @markfischetti

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

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Language

- **Science Crossword: A Trip Down Memory Lane**
  Play this crossword inspired by the September 2025 issue of Scientific American

Science Crossword: A Trip Down Memory Lane

By Aimee Lucido

This crossword is inspired by the September 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.

We're celebrating 180 years of *Scientific American*. Explore our legacy of discovery and look ahead to the future.

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.

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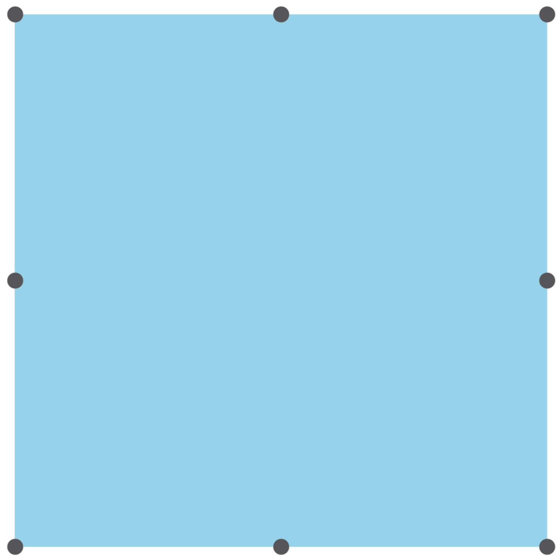
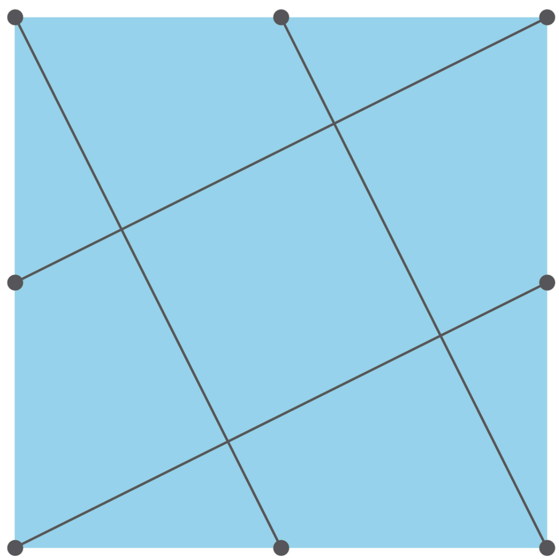
Mathematics

Math Puzzle: Dissect the Square

By Emma R. Hasson



Draw four lines beginning and ending at the gray points to break this square into pieces that can be rearranged into five identical squares.



Bonus: Suppose you can use any number of lines that begin and end anywhere along the edge. What other quantities of identical squares can you make with no extra pieces left over?

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

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*.

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https://www.scientificamerican.com/article/math-puzzle-dissect-the-square

Mathematicians Crack 125-Year-Old Problem, Unite Three Physics Theories

A breakthrough in Hilbert's sixth problem is a major step in grounding physics in math

By Jack Murtagh edited by Jeanna Bryner

Mathematicians suggest they have figured out how to unify three physical theories that explain the motion of fluids.
Floriana/Getty Images

When the greatest mathematician alive unveils a vision for the next century of research, the math world takes note. That's exactly what happened in 1900 at the International Congress of Mathematicians at Sorbonne University in Paris. Legendary mathematician David Hilbert presented 10 unsolved problems as ambitious guideposts for the 20th century. He later expanded his list to include 23 problems, and their influence on mathematical thought over the past 125 years cannot be overstated.

Hilbert's sixth problem was one of the loftiest. He called for "axiomatizing" physics, or determining the bare minimum of mathematical assumptions behind all its theories. Broadly construed, it’s not clear whether mathematical physicists could ever be sure they had fully met this challenge. Hilbert mentioned some specific subgoals, however, and researchers have since refined his vision into concrete steps toward its solution.

In March mathematicians Yu Deng of the University of Chicago and Zaher Hani and Xiao Ma, both at the University of Michigan, posted a paper to the preprint server arXiv.org in which they claim to have achieved one of these goals. If their work withstands scrutiny, it will mark a major stride toward grounding physics in math and may open the door to analogous breakthroughs in other areas of physics.

In the paper, the researchers suggest they have figured out how to unify three physical theories that explain the motion of fluids. These theories govern a range of engineering applications from aircraft design to weather prediction—but until now, they rested on assumptions that hadn’t been rigorously proven. This breakthrough won’t change the theories themselves, but it mathematically justifies them and strengthens our confidence that the equations work the way we think they do.

Each theory differs in how much it zooms in on a flowing liquid or gas. At the microscopic level, fluids are composed of particles—little billiard balls bopping around and occasionally colliding—and Newton’s laws of motion work well to describe their trajectories.

But when you zoom out to consider the collective behavior of vast numbers of particles, at the so-called mesoscopic level, it’s no longer convenient to model each one individually. In 1872 Austrian theoretical physicist Ludwig Boltzmann addressed this problem when he developed (what became known as) the Boltzmann equation. Instead of tracking the behavior of every particle, the equation considers the likely behavior of a typical particle. This statistical perspective smooths over the low-level details in favor of higher-level trends. The equation allows physicists to calculate the evolution of quantities such as momentum and thermal conductivity in the fluid without painstakingly considering every microscopic collision.

Zoom out further, and you find yourself in the macroscopic world. Here we view a fluid not as a collection of discrete particles but as a single continuous substance. At this level of analysis, a different suite of equations—the Euler and Navier-Stokes equations—accurately describe how fluids move and how their physical properties interrelate without recourse to particles at all.

The three levels of analysis all describe the same underlying reality—how fluids flow. In principle, each theory should build on the theory below it in the hierarchy: at the macroscopic level, the Euler and Navier-Stokes equations should follow logically from the Boltzmann equation at the mesoscopic level, which in turn should follow logically from Newton's laws of motion at the microscopic level. This relation is the kind of axiomatization that Hilbert called for in his sixth problem, and he explicitly referenced Boltzmann's work on gases in his write-up of the problem. We expect complete theories of physics to follow mathematical rules that explain a phenomenon from the microscopic to the macroscopic levels. If scientists fail to bridge that gap, then it might suggest a misunderstanding in our existing theories.

Unifying the three perspectives on fluid dynamics has been a stubborn challenge for the field, but Deng, Hani and Ma may have just done it. Their achievement builds on decades of incremental progress. Prior advancements all came with some kind of asterisk, though; for example, the derivations involved worked only on short timescales, in a vacuum, or under other simplifying conditions.

The new proof broadly consists of three steps: derive the macroscopic theory from the mesoscopic one, derive the mesoscopic theory from the microscopic one, and then stitch them together in a single derivation of the macroscopic laws all the way from the microscopic ones.

The first step was previously understood, and even Hilbert himself contributed to it. Deriving the mesoscopic from the microscopic, however, has been much more mathematically difficult. Remember, the mesoscopic setting is about the collective behavior of vast numbers of particles. So Deng, Hani and Ma looked at what happens to Newton’s equations as the number of individual particles colliding and ricocheting grows to infinity and the particles’ size shrinks to zero. They proved that when you stretch Newton’s equations to these extremes, the statistical behavior of the system—or the likely behavior of a typical particle in the fluid—converges to the solution of the Boltzmann equation. This step forms a bridge by enabling one to derive the mesoscopic math from the extremal behavior of the microscopic math.

The major hurdle in this step concerned the length of time that the equations were modeling. Mathematicians already knew how to derive the Boltzmann equation from Newton’s laws on very short timescales, but that doesn’t suffice for Hilbert’s program, because real-world fluids can flow for any stretch of time. With longer timescales comes more complexity: more collisions take place, and the entire history of a particle’s interactions might bear on that particle’s current behavior. The authors overcame this obstacle by doing careful accounting of just how much a particle’s history affects its present and leveraging new mathematical techniques to argue that the cumulative effects of prior collisions remain minor.

Gluing together their long-timescale breakthrough and previous work on deriving the Euler and Navier-Stokes equations from the Boltzmann equation unifies three theories of fluid dynamics. The finding justifies taking different perspectives on fluids based on what’s most useful in context because mathematically they converge on one ultimate theory describing one reality. Assuming the proof is correct, it breaks new ground in Hilbert’s program. We can only hope that with just such fresh approaches, the dam will burst on Hilbert’s challenges, and more physics will flow downstream.

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

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https://www.scientificamerican.com/article/lofty-math-problem-called-hilberts-sixth-closer-to-being-solved

Medicine

- **Pessimistic Dogs Are Better at Smelling Cancer—And Other Keys to Disease-Sniffing Success**
  New research is revealing how disease-smelling dogs can excel

Pessimistic Dogs Are Better at Smelling Cancer—And Other Keys to Disease-Sniffing Success

New research is revealing how disease-smelling dogs can excel

By Rohini Subrahmanyam edited by Sarah Lewin Frasier

Individual dogs' personalities, and how we interpret their behaviors, may be key to disease sniffing at scale.
Johner Images/Getty Images

Billy, a floppy-eared little beagle, darts around a platform sniffing a series of holes. Each hole contains a used surgical-type mask bearing a different human's distinct mix of scents. But her sharp nose is hunting for just one such combination: the one that signals cancer.

Cancer can change a person's "volatilome," the unique set of volatile organic compounds found in breath, sweat, blood and urine. Billy and her cohort have learned to sniff out these subtle scent cues in masks worn by people with cancer diagnoses. Researchers are also studying how dogs can detect diseases such as COVID and malaria, as well as psychological conditions, including post-traumatic stress disorder.

Scientists discovered dogs' powerful disease-smelling skills in 1989, when a dog detected cancer in its handler. But clinicians still do not routinely use dogs for diagnosis. Besides the obvious logistical challenges, dogs vary greatly in their olfactory accuracy. Researchers are increasingly finding that disease-sniffing prowess may come down to individual dogs' personality—and how well their handlers know them. New research efforts are focused on figuring out which dogs would be best for the job and on interpreting dogs' behaviors during a smell test.

Sharyn Bistre Dabbah, a veterinary scientist now at the University of Bristol in England, set out with colleagues at the U.K. charity Medical Detection Dogs to learn how the animals' personalities—especially their level of optimism or pessimism—affect disease-detection skills. Their results appeared recently in *PLoS One*.

The researchers first showed the dogs what lay behind two screens at one end of a room: a "positive" location with a tasty treat and a "negative" one with an empty bowl. On subsequent visits to the room, the dogs typically bounded happily toward the former but trotted very slowly when they went to check out the latter—or simply didn't go there at all.

The scientists then placed bowls behind two new screens between the positive and negative spots, and they classified the dogs as "optimistic" or "pessimistic" based on how quickly they investigated these new locations.

Next, the team evaluated how accurately each dog could pick out a disease scent it was trained to detect among other smells. On average, the pessimistic dogs turned out to be more discerning. Pessimistic dogs are more cautious, and "a more cautious dog might be better at not making mistakes," Dabbah says.

Other personality traits also play a role, says Clara Wilson, who researches disease- and stress-sniffing dogs at the University of

Pennsylvania. Dogs that enjoy the thrill of a hunt—and thrive while searching for missing people or hidden bombs—might find sniffing through disease samples again and again rather repetitive. “We want a dog that doesn’t get frustrated. They [should] find it rewarding, even though it may be less exciting,” Wilson explains.

Handlers’ interpretations of dog behavior can also skew detection outcomes, says Akash Kulgod, co-founder of Dognosis, the Bengaluru-based start-up that trained Billy. Instead of teaching dogs to perform a specific behavior such as sitting or barking when they pick up an assigned scent—a process that takes extra time and can lead to dogs “lying” for treats—Kulgod and his team directly analyze each dog’s natural body language. Based on how confidently the dogs move, as analyzed with computer vision–based machine-learning tools, the team can spot successful detections. “One of our dogs sniffs and then very confidently somersaults to go to the feeder,” Kulgod says. “They each have their own unique quirks—but all of it can be quantified because it’s all related to this reward expectation that you have from the past sessions.”

In a pilot study with 200 test samples involving 10 cancer types, presented at this year’s American Society of Clinical Oncology conference, Dognosis dogs detected 96 percent of cancers. Next, the Dognosis team will scale up its study with 1,500 test samples.

Doctors currently diagnose many kinds of cancers by using a combination of blood tests and biopsies. Researchers are always on the lookout for less invasive methods—including options directly involving our canine companions, as well as electronic noses inspired by them. Dogs can currently outperform electronic sniffers. But this primacy may not last, according to Andreas Mershin, chief science officer at the Boston-based start-up RealNose.ai. He and his colleagues are developing electronic noses to sniff urine samples for prostate cancer and other diseases. If machine olfaction eventually surpasses dogs' abilities, it could help tackle the scalability problem—and give the animals a break.

Mershin's team put mammalian smell receptors on an electronic chip and used machine-learning algorithms to interpret the output. The technique focuses on broader patterns among detected molecules rather than categorizing them individually.

Dogs don't tick off a list of molecules in their heads, either; they just "know" what cancer smells like. This helps them to sense it accurately no matter which organ it is from or what the patient ate before giving their breath sample. "The dogs can generalize. They don't care about the font in which you write the scent; they just interpret it correctly," Mershin says.

In a study published in *PLoS One*, Mershin and his team used machine-learning models to spot and analyze patterns of different odorants in urine samples from confirmed prostate cancer patients. Their findings, which built on work with diagnosis data from Medical Detection Dogs, suggest that focusing on this type of "scent character" might work as a scalable alternative to dogs, even if it's currently much slower.

Meanwhile, back on the test platform, floppy-eared Billy quickly detects the subtle scent of cancer in one of the masks she has been sniffing—and confidently bounds back to get her reward. Such tests show strong potential, says postdoctoral researcher Amritha Mallikarjun of the University of Pennsylvania: "The dogs, because of their amazing sense of smell and detection capabilities, are demonstrating to us what technology could look like 10 to 15 years from now."

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/pessimistic-dogs-are-better-at-smelling-cancer-and-other-keys-to-disease

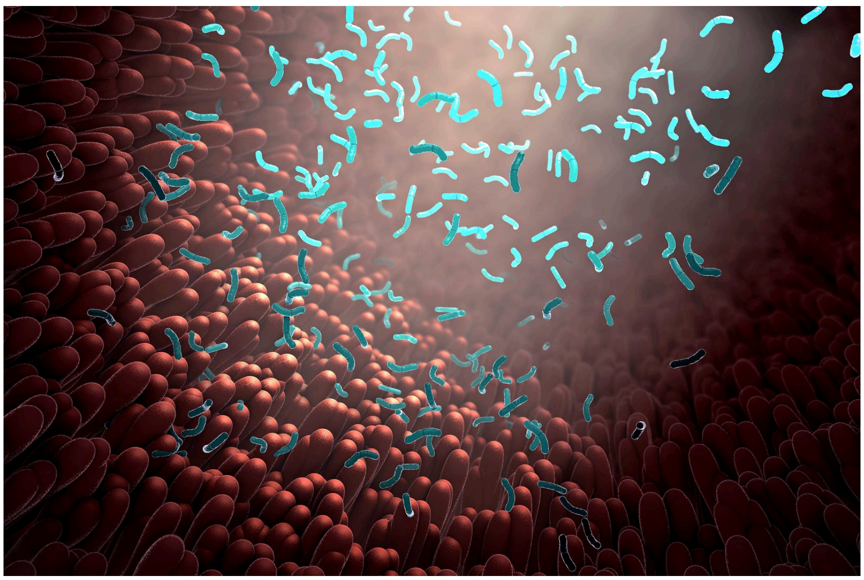
Microbiome

- **Human Gut Bacteria Can Gather Up PFAS ‘Forever Chemicals’**
  When tested on their own and in mice, these bacterial strains from the human microbiome show promise in accumulating PFAS

Human Gut Bacteria Can Gather Up PFAS ‘Forever Chemicals’

When tested on their own and in mice, these bacterial strains from the human microbiome show promise in accumulating PFAS

By Nora Bradford edited by Sarah Lewin Frasier

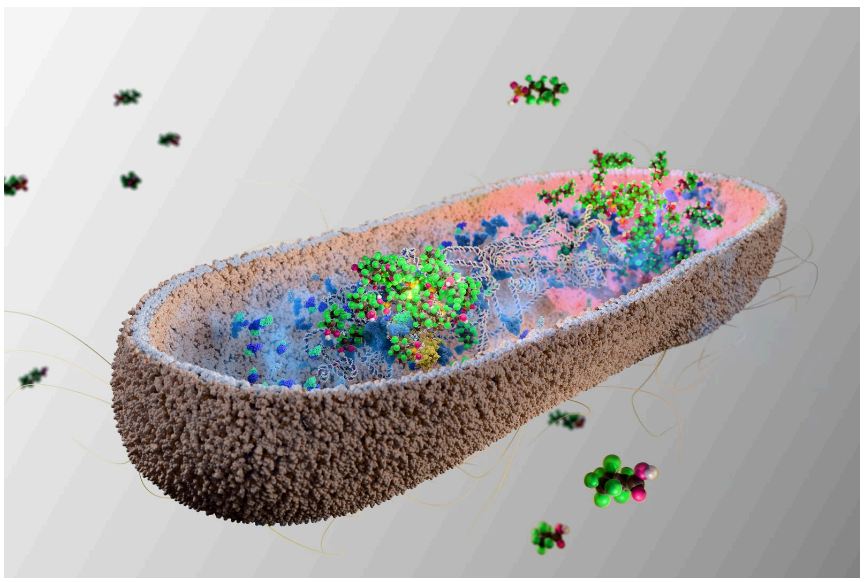


Visualization of bacteria in the human gut.
Christoph Burgstedt/Science Source

Lurking in our nonstick pans, our rain jackets and even our drinking water are toxic compounds known as perfluoroalkyl and polyfluoroalkyl substances (PFAS), also called “forever chemicals.” They can take hundreds of years to break down in the environment and are increasingly being detected in human blood and bodily tissues—where, research suggests, they can lead to several cancers and reproductive disorders, as well as thyroid disease and a weakened immune system. Scientists have been scrambling to find ways to remove PFAS from our surroundings before they reach human bodies. But one team may have found a way to tackle them afterward, too.

Bacteria commonly found in the human gut, such as *Bacteroides uniformis* and *Odoribacter splanchnicus*, could possibly be used to gather up PFAS and carry them out as waste, scientists suggest in a study published recently in *Nature Microbiology*.

"I think this research provides us a little glimmer of hope that it's not all doom and gloom" when it comes to the PFAS problem, says study senior author Kiran Patil, a molecular biologist at the University of Cambridge. "Our bacteria—that have been our companions for thousands of years—may already be helping us do something about it."



The gut bacteria accumulates perfluorononanoic acid—a 'forever chemical'—as dense clumps. Peter Northrop/MRC Toxicology Unit

The researchers first tested how PFAS and other pollutants interacted with dozens of bacterial strains from the human gut, and they noticed that nine species of bacteria accumulated certain PFAS chemicals very effectively. When grown in laboratory conditions, these organisms sopped up anywhere from 23 to 74 percent of the PFAS chemicals they were exposed to within 24 hours. The team suggests those particular strains could potentially bind to PFAS in the body and eliminate them from the system as waste.

Previous work had shown that bacteria from contaminated soils can bind to PFAS. But those bacteria were exposed to much higher levels of the chemicals and had a relatively low capacity to sequester them, Patil says, “so we had no reason to believe that gut bacteria would be anything special.”

Many investigators had assumed that PFAS molecules would cling to a bacterium’s outer membrane rather than getting inside it, Patil says. Because the bacteria in the study were gathering more PFAS than could feasibly fit on their membrane, however, the team thought the chemicals must have actually entered the organisms. To confirm this hypothesis, Patil and his colleagues used an imaging technique in which they rapidly froze the bacteria, then fired tiny beams of charged particles at them and analyzed what flew out. They detected fluorine molecules—a telltale sign of PFAS—emerging from the bacteria.

To find out whether the bacteria would collect PFAS chemicals inside a larger organism, the team used mice raised to lack a microbiome of their own and colonized the animals’ guts with several human microbiome bacteria that were shown to absorb PFAS. After exposing the mice to various levels of a PFAS chemical, the scientists measured the amount of PFAS in the animals’ feces and found that mice with PFAS-collecting bacteria excreted more of the toxic chemicals than those without the microorganisms did. (They also excreted more than mice given bacteria that accumulated PFAS less well.)

This study shows just how deeply PFAS penetrate a body and its systems, says environmental epidemiologist Jesse Goodrich of the University of Southern California, who was not involved in the work. “It is another piece in the puzzle that shows how PFAS can impact human health.”

Applying the latest findings to humans would require more research. Patil and his colleagues are now planning a clinical trial to test whether probiotics containing such bacteria could supplement the human microbiome and decrease PFAS in our own species' gut. But they note that such a trial would have far more variable factors than a highly controlled study in mice with lab-designed microbiomes. "There's a huge variation in how the composition of the microbiome is set up within humans," says Anna Lindell, a toxicologist at the University of Cambridge and lead author of the *Nature Microbiology* study.

Further research could also observe the naturally occurring levels of these bacterial strains in people within the same community and measure the amount of PFAS in their bodies, Patil says. Such a study would help to clarify whether these bacteria lead to fewer PFAS in the human gut—or even in other parts of the body.

Supplementing the body's natural bacteria to manage PFAS absorption is "interesting and has potential," Goodrich says. "But ultimately the best way to protect health is to prevent exposure in the first place."

Nora Bradford is a freelance science writer, former news intern at *Scientific American* and a Ph.D. student in cognitive science. Follow Bradford on Bluesky @norabradford.bsky.social

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https://www.scientificamerican.com/article/human-gut-bacteria-can-gather-up-pfas-forever-chemicals

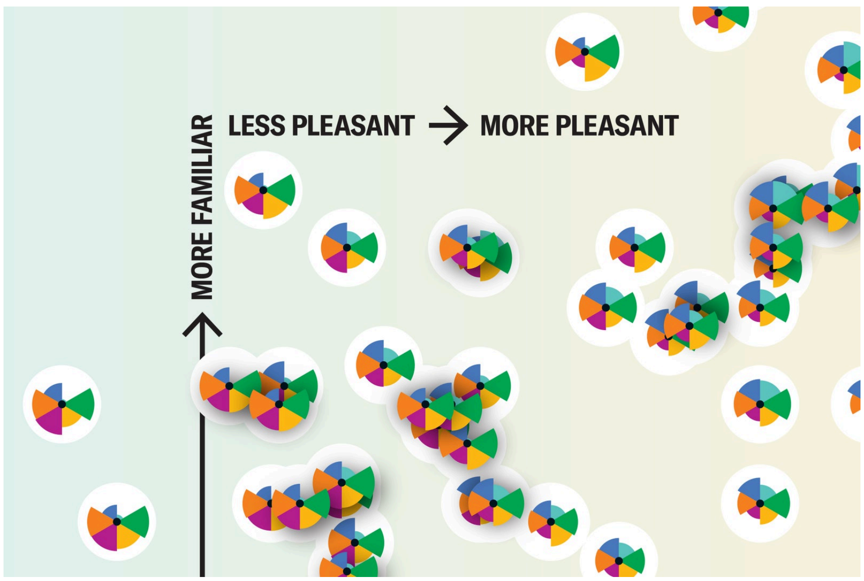
Physiology

- **Why Some Smells Are Pleasant and Others Are Off-Putting, according to Science**
  More familiar smells and scents from complex molecules can often be more appealing

The Surprising Science behind Your Favorite (and Least Favorite) Scents

More familiar smells and scents from complex molecules can often be more appealing

By Clara Moskowitz, Miriam Quick & Jen Christiansen edited by Jen Christiansen & Clara Moskowitz

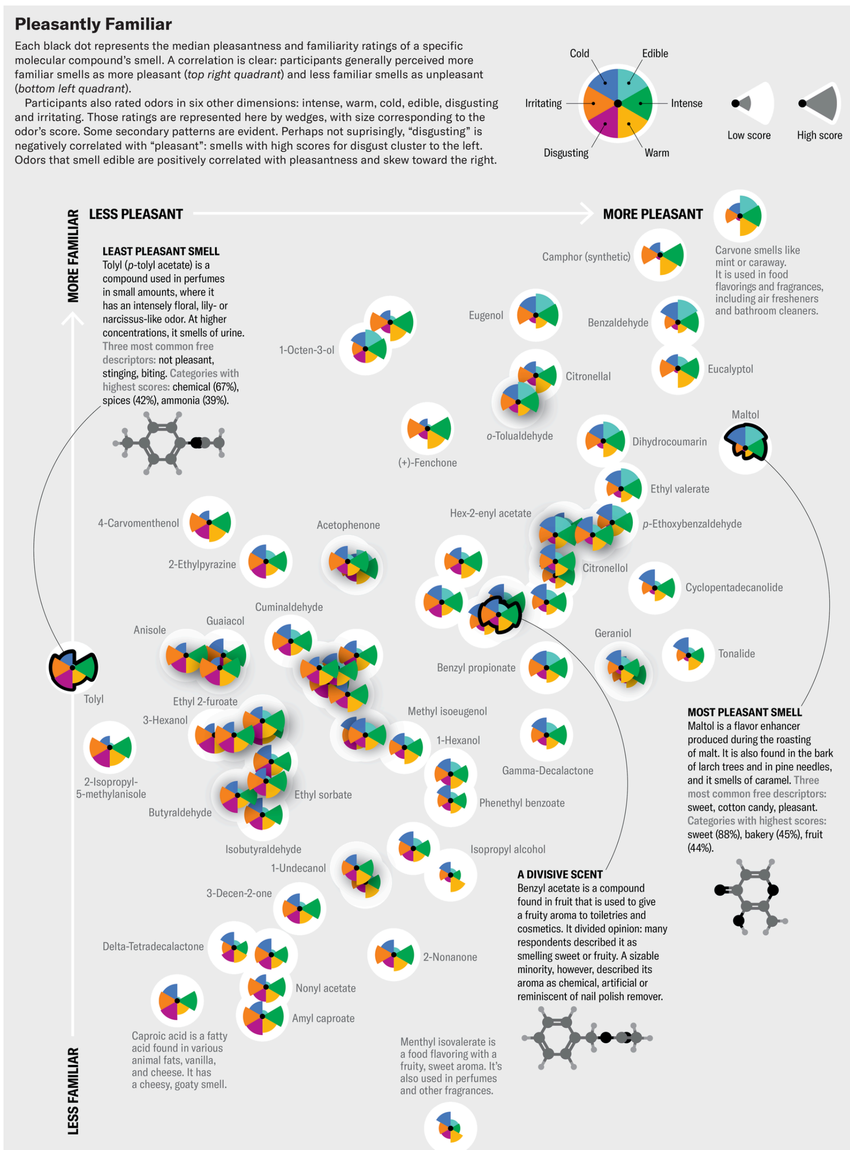


Jen Christiansen

The sense of smell, it turns out, is highly individual—a scent that is enticingly floral to one person may be off-putting and chemical to another. Researchers in Germany -recently asked 1,227 participants to describe and rate 73 odors and found their answers to be strikingly variable. “It depends on what people have associated with the odor,” says psychologist Antonie Louise Bierling of Friedrich Schiller University Jena, who led the study. “In my opinion, it would have been really astonishing if the same odor smelled pleasant to everyone. Why should it?”

The researchers did identify some trends. More familiar scents seem to smell more pleasant, for example, and odors from complex molecules seem to have greater appeal than those of simple compounds.

Scientists hope to use the data to help develop “digital olfaction”—artificial noses that can sense scents as well as or even better than humans. Such devices could be helpful for disease detection or in “smart home” applications: a toaster, for instance, that can tell when toast is done by how burned it smells or refrigerators that can identify putrid decay.

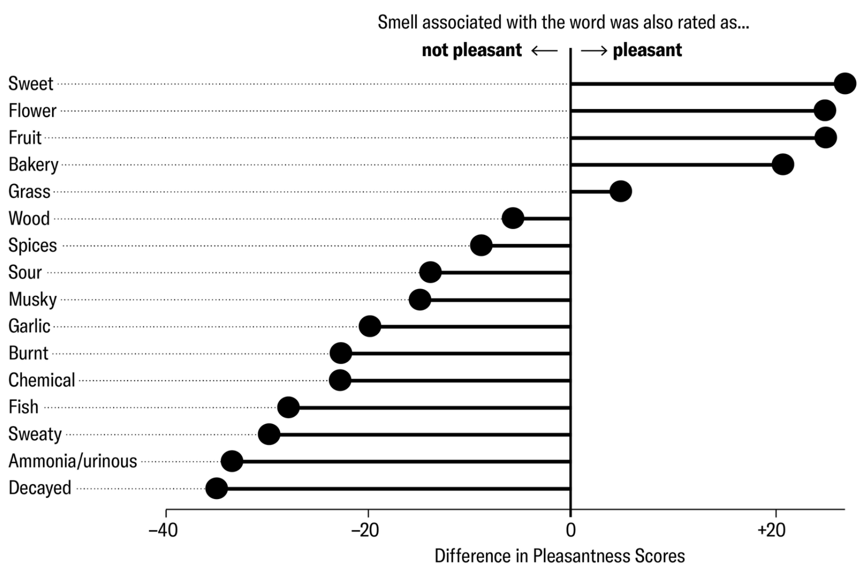


Miriam Quick and Jen Christiansen; Source: “A Dataset of Laymen Olfactory Perception for 74 Mono-Molecular Odors,” by Antonie Louise Bierling et al., in *Scientific Data*, Vol. 12; February 2025 (*data*)

HOW WE DESCRIBE SMELLS, GOOD AND BAD

Researchers asked people to respond with “yes” or “no” to whether each smell matched 16 descriptive terms, including “sweet” and “musky.” By comparing these answers with the sliding-scale pleasantness scores the same molecules received, we can see which words were most predictive of pleasant—and unpleasant—smells.

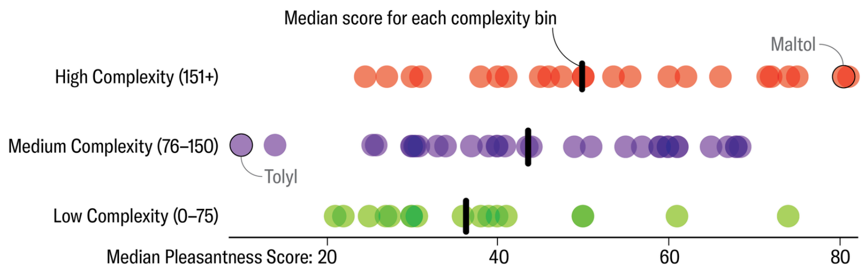
The dots in the graph indicate the difference in pleasantness scores for scents associated with each word. For example, the median pleasantness score for all molecules that the respondents considered “sweet” was 61, and the median pleasantness score for all molecules that were not marked as “sweet” was 34. The difference in scores was +27, making “sweet” the word most frequently associated with scents perceived as pleasant. “Decayed,” with a difference in scores of –35, was the descriptor least associated with pleasant smells.



Miriam Quick and Jen Christiansen; Source: “A Dataset of Laymen Olfactory Perception for 74 Mono-Molecular Odors,” by Antonie Louise Bierling et al., in *Scientific Data,* Vol. 12; February 2025 (*data*)

THE SWEET SMELL OF COMPLEXITY

More complex molecules tend to be ranked as more pleasant. Molecular complexity is a rough estimate of how intricate a compound’s structure is. It runs on a scale from 0 to thousands—in this dataset, from 10.8 (isopropyl alcohol) to 366 (tonalide). Here each dot represents one of 73 molecules. We grouped molecular complexity into three categories: high, medium and low. Vertical bars mark median pleasantness scores per category.



Miriam Quick and Jen Christiansen; Source: “A Dataset of Laymen Olfactory Perception for 74 Mono-Molecular Odors,” by Antonie Louise Bierling et al., in *Scientific Data*, Vol. 12; February 2025 (*data*)

Clara Moskowitz is a senior editor at *Scientific American*, where she covers astronomy, space, physics and mathematics. She has been at *Scientific American* for a decade; previously she worked at Space.com. Moskowitz has reported live from rocket launches, space shuttle liftoffs and landings, suborbital spaceflight training, mountaintop observatories, and more. She has a bachelor's degree in astronomy and physics from Wesleyan University and a graduate degree in science communication from the University of California, Santa Cruz.

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

Jen Christiansen is author of the book *Building Science Graphics: An Illustrated Guide to Communicating Science through Diagrams and Visualizations* (CRC Press) and senior graphics editor at *Scientific American*, where she art directs and produces illustrated explanatory diagrams and data visualizations. 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

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https://www.scientificamerican.com/article/why-some-smells-are-pleasant-and-others-are-off-putting-according-to-science

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Psychology

- ## How Nostalgia Keeps Friendships Alive
  The social and psychological consequences of yearning for the past are starting to come into focus

How Nostalgia Keeps Friendships Alive

The social and psychological consequences of yearning for the past are starting to come into focus

By Kuan-Ju Huang edited by Daisy Yuhas & Madhusree Mukerjee


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Nostalgia is a complex emotion. It blends longing for a cherished past and the bittersweet realization that one can never fully return to that time. Psychologists have puzzled over why humans experience this intricate emotion: Does nostalgia trap us in the past, or does it function as a psychological resource that could lead us to the future?

People can feel nostalgic about many things, but research shows that nostalgic narratives often center on others, especially those who hold significant meaning in our lives. We may feel nostalgic for childhood family gatherings, shared experiences with school friends or holidays spent with loved ones. Even when we reminisce about other things, such as childhood toys or the aroma of a particular meal, those memories often feature moments we shared with people.

Nostalgia’s social tones are well known to researchers, but its effect on social relationships is less understood. In previous research, my colleagues and I found that during the COVID lockdown, people often turned to music that evoked nostalgic feelings when they were lonely and lacked social contact. Similar patterns appeared in the selection of other leisure diversions, such as classic television shows, traditional board games, and other familiar favorites. People seem to find solace in nostalgia when they are disconnected from others.

We wondered whether nostalgia might be triggered to help people regulate their social connections and networks. So we conducted a series of studies examining whether nostalgia not only brings temporary feelings of connectedness but also motivates people to seek, and eventually achieve, close connection with others.

In our first two studies, we surveyed 449 undergraduate students from the University at Buffalo and an additional 396 U.S. adults recruited through a nationwide survey panel. Participants completed a questionnaire measuring “nostalgia proneness,” which used a scale to assess how frequently they felt nostalgic and how important they found those experiences to be. They also reported the number of people in their “closest networks” (friends and loved ones it would be hard to imagine life without), “close networks” (also close but not part of the inner circle), and “less close networks” (still important but not as tightly connected).

Participants who were highly prone to nostalgia had more people in their innermost friendship circle. Further, this tendency was linked to their motivation to maintain their social network. Nostalgic people seem to be more driven to invest resources in keeping their relationships over time. This effect holds true regardless of a person’s personality traits, such as extroversion, agreeableness or emotional stability.

Past research shows that there are two major motivations affecting our social networks: one to meet new people and the other to strengthen our existing connections. We discovered that people who were very nostalgia-prone scored high on both types of social motivations, but the effects were stronger for bolstering their existing network.

It was not clear from the data whether nostalgia caused changes in behavior and motivation, however. Perhaps having big social networks and lots of old friends simply makes people yearn more for the past. In a third study, we set out to investigate that possibility by analyzing data from the Netherlands that tracked the same group of 520 participants from 2013 to 2019, recording things such as their proneness to nostalgia and their social network size.

Much like in the U.S. surveys, people with a greater tendency for nostalgia in the Dutch group had a larger number of close social ties, that is, people whom participants reported being able to talk to about important matters in their lives. But the finding most exciting to us had to do with change over time. Nostalgia measured at an earlier time point predicted a larger network of close ties later. Having a larger close social network, however, was not a predictor of more nostalgic tendencies later. That suggests nostalgia could be driving changes in social behavior that promote connection.

The Dutch data also showed that close social networks tend to shrink with age. As people grow older, they are more likely to lose touch and cut ties for varied reasons, including major life events such as moving to a new city or becoming a parent. In line with that, we found that the close social networks of people with low levels of nostalgia proneness shrank over the seven years for which we had data. Interestingly, though, those with high nostalgia proneness maintained a relatively stable number of close networks in the same period.

Our research is the first to show that a past-oriented emotion can have substantial, long-term effects on our social relationships. We suspect that nostalgia acts as a psychological resource, helping people counteract the loss of close friendships and relationships as they age. As such, it joins many other human emotions that have evolved to serve important social functions.

So go ahead and listen to your favorite song from high school. Or find an episode from that TV show that made you laugh when you were young. Nostalgia may act as an emotional signal that guides us to reflect on what truly matters in our lives. The sense of potential loss and irreversibility that comes with it may serve as a powerful reminder to live fully in the present with our loved ones and aim to move toward a shared future.

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 dyuhas@sciam.com.*

Kuan-Ju Huang is a graduate student in human and environmental studies at Kyoto University in Japan. He studies culture, emotion and social interaction.

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