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New footage shows bizarre deep-sea fish that sees through its forehead

Barreleye fish live in the ocean twilight zone.

By Nicoletta Lanese

(Image credit: © 2021 MBARI)

Thousands of feet beneath the surface of Monterey Bay off California, scientists recently captured footage of a fish with a bulbous, translucent head and green orb-like eyes that peer out through its forehead.

This bizarre creature, known as a barreleye fish (is very rarely seen. Researchers with the Monterey Bay Aquarium Research Institute (MBARI) have only spotted the species nine times, despite having sent their remotely operated vehicles (ROV) on more than 5,600 dives in the fish's habitat, MBARI tweeted on Dec. 9.

But last week, a team of scientists deployed MBARI's ROV Ventana and caught sight of a barreleye fish suspended in the water. At the time, the ROV was cruising at a depth of about 2,132 feet (650 meters) in the Monterey Submarine Canyon, one of the deepest submarine canyons on the Pacific coast, Thomas Knowles, a senior aquarist at the Monterey Bay Aquarium, told Live Science in an email.

"The barreleye first appeared very small out in the blue distance, but I immediately knew what I was looking at.

It couldn't be mistaken for anything else," he said.

As a buzz of excitement rippled through the control room, Knowles kept the ROV camera in focus while the ROV pilot Knute Brekke kept the underwater robot pointed at the barreleye.

"We all knew that this was likely a once in a lifetime experience," since the elusive creature is seen so very rarely, Knowles said.

In the light of the ROV, the barreleye's eyes glowed bright green and could be easily seen through the clear, fluid-filled shield that covers the fish's head.

These eyes are incredibly light-sensitive and can be oriented straight up, towards the top of the fish's head, or straight ahead, according to MBARI.

Two dark-colored capsules sit in front of the fish's eyes and contain the organs the animal uses to smell. The barreleye fish's habitat ranges from the Bering Sea to Japan and Baja California.

The fish live in the ocean twilight zone, which lies about 650 to 3,300 feet (200 to 1,000 m) underwater; specifically, barreleyes live about 2,000 to 2,600 feet (600 to 800 m) beneath the ocean surface, near the depth where the water plunges into complete darkness, according to MBARI. "We have no handle on population size, except in a relative sense," Bruce Robison, an MBARI senior scientist, told Live Science in an email.

Barreleyes are less abundant than commonly-seen twilight zone fish, such as lanternfish or bristlemouths, and the MBARI team encounters barreleye fish about as often as they do anglerfish, whalefish and gulpers, "which is very rarely," he said.

Based on past observations by MBARI researchers, published in 2008 in the journal Copeia, scientists think that barreleye fish mostly remain motionless as they wait for unwary prey, like zooplankton and jellyfish, to drift overhead.

The fish can hover this way thanks to a set of broad, flat fins that extend out from its body.

By pointing their verdant eyes straight upward, barreleyes can spot the silhouettes of their prey from above, and the green pigment in their eyes likely helps filter out sunlight from the ocean surface.

Once a barreleye fish spots a bioluminescent jelly or tiny crustacean floating by, it zooms upward to snag the creature in its mouth while

rotating its eyes forward, so it can see where it's going.

Scientists speculate that *M. microstoma* may sometimes swipe food from siphonophores — jellyfish-like organisms that cling together in long lines and capture prey in their tentacles, according to a 2009 MBARI video.

The barreleye fish's transparent head shield might protect it against the stinging cells in the siphonophores' tentacles — but again, this is speculation. "Most aspects of their natural history remain unknown and much of what we think we know about them is based on speculation," Robison said.

Although *M. microstoma* was first described in 1939, fishers caught these early specimens in nets that destroyed their transparent head shields.

So scientists didn't know about the shields until the 2000s, when MBARI scientists saw a barreleye fish in its natural habitat, he said. As of today, there's still much to learn about the funky fish.

On their recent dive, the team avidly watched the *M. microstoma* specimen until it swam away and then continued their search for jellies and comb jellies of the deep sea. "We had no ambition to collect this animal," as the aquarium is not adequately set up to care for the poorly understood fish, Knowles said.

That said, many other bizarre and wondrous creatures from the deep sea will soon be on display at the aquarium.

In spring 2022, the Monterey Bay Aquarium will open a new exhibition called "Into the Deep: Exploring Our Undiscovered Ocean," which will feature all sorts of deep-sea creatures, from giant isopods to sea spiders to blood-belly comb jellies, according to the aquarium's website.

And like the barreleye fish, many of these creatures look like something plucked straight from a sci-fi novel.

https://go.nature.com/30zrJRp

NASA spacecraft 'touches' the Sun for the first time ever

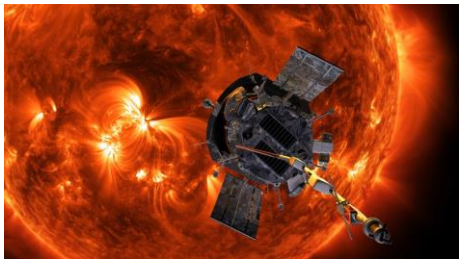
The Parker Solar Probe has passed through a boundary and into the Sun's atmosphere, gathering data that will help scientists better understand stars.

Alexandra Witze

A NASA spacecraft has entered a previously unexplored region of the Solar System — the Sun's outer atmosphere, or corona. The long-awaited milestone, which was reached in April but announced on 14 December, is a major accomplishment for the Parker Solar Probe, a craft that is flying closer to the Sun than any mission in history.

"We have finally arrived," said Nicola Fox, director of NASA's heliophysics division, located at the agency's headquarters in Washington DC. "Humanity has touched the Sun."

She and other team members spoke during a press conference at this week's American Geophysical Union meeting in New Orleans, Louisiana. A paper describing the findings appears this week in *Physical Review Letters*[1].



The Parker Solar Probe will pass close by the Sun 24 times, looping ever closer to its surface. **Credit: NASA/Johns Hopkins APL**

In many ways, the Parker Solar Probe is a counterpoint to NASA's twin Voyager spacecraft. In 2012, Voyager 1 travelled so far from the Sun that it became the first mission to leave the region of space dominated by the solar wind — the energetic flood of particles coming from the Sun. By contrast, the Parker probe is travelling ever closer to the heart of the Solar System, flying head-on into the solar wind and our star's atmosphere. With this new front-row seat,

scientists can explore some of the biggest unanswered questions about the Sun, such as how it generates the solar wind and how its corona gets heated to temperatures more extreme than those on the Sun's surface.

"This is a huge milestone," says Craig DeForest, a solar physicist at the Southwest Research Institute in Boulder, Colorado, who is not involved in the mission. Flying into the solar corona represents "one of the last great unknowns", he says.

Into the unknown

The Parker probe crossed into the Sun's atmosphere at 09:33 universal time on 28 April. Mission scientists needed several months to download and analyse the data the spacecraft had collected, and to be sure that it had indeed crossed the much-anticipated boundary, known as the Alfvén surface.

This surface marks the interface between the Sun's atmosphere and an outer region of space dominated by the solar wind. Swedish physicist Hannes Alfvén proposed the underlying theory behind the boundary in a paper published in *Nature* in 1942[2], and scientists have been looking for it ever since.

But it took the US$1.5-billion Parker Solar Probe to finally get there. Since its launch in 2018, it has been orbiting the Sun: with each pass, it loops ever closer to the solar surface. A carbon-composite heat shield protects its instruments from temperatures that will eventually soar to 1,370 °C.

The spacecraft crossed the Alfvén boundary when it was around 14 million kilometres, or just under 20 solar radii, from the Sun's surface. That's about where team members had expected to find the interface, says Nour Raouafi, the mission's project scientist at the Johns Hopkins University Applied Physics Laboratory in Laurel, Maryland.

Some researchers had speculated that the boundary would be rather 'fuzzy' — but, instead, it was sharp and wrinkly. The spacecraft's trajectory took it into the corona for nearly five hours and then back out; and it might have briefly crossed into the corona twice more. Inside the corona, the solar wind speed and plasma densities dropped, suggesting the boundary had indeed been crossed. "We are learning new things that we did not have access to before," Raouafi says.

Streamers and switchbacks

As it crossed the Alfvén surface, the Parker probe flew through a 'pseudostreamer' of electrically charged material, inside which conditions were quieter than the roiling environment outside of it. While inside the corona, the spacecraft also studied unusual kinks in the magnetic field of the solar wind, known as switchbacks. Scientists already knew about switchbacks, but the probe's data have enabled the researchers to trace where the switchbacks come from, all the way down to the solar surface[3].

Knowing how such features form on the Sun, and how they influence the solar wind and other eruptions of charged particles, will help people on Earth to prepare for disruptive space weather, such as solar storms that can knock out satellite communications. The discoveries will also help researchers to understand the forces that power stars other than the Sun, said Kelly Korreck, a solar physicist at NASA's headquarters, at the press conference.

The Parker Solar Probe ultimately aims to make 24 close passes of the Sun. It crossed the Alfvén surface on the eighth of those fly-bys, and might have done so again during its ninth pass in November — a manoeuvre for which the data have not yet been fully downloaded and analysed. The mission's closest approach is scheduled for 2025, when it will be only 6.2 million kilometres from the solar surface, well within the orbit of Mercury. Each visit will continue to reveal new information about processes in the corona, said Justin Kasper, a solar physicist and deputy chief technology officer at BWX

Technologies in Washington DC, who works on the Parker probe.

“Being this close to the Sun is allowing us to make really interesting and new connections we wouldn’t be able to do from afar,” he said.

doi: https://doi.org/10.1038/d41586-021-03751-5

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https://wb.md/3e3ogOm

The Surprisingly Average Intelligence of Brain Surgeons and Rocket Scientists

What other professions have their own idioms associated with them?

F. Perry Wilson, MD, MSCE

Welcome to *Impact Factor*, your weekly dose of commentary on a new medical study. I'm Dr F. Perry Wilson of the Yale School of Medicine.

The smartest kid in my med school class, in my opinion, was my roommate Dave. Here we are looking dapper and very, very young.

Dave aced every test, every rotation. This was a guy who knew the clotting and complement cascades by heart. Would recite his patient histories, medications, and lab values from memory. Knew the codes to every clean supply room in the hospital and where you could find free food at any given time. Smart.

So it was no surprise when Dave went into neurosurgery. Of course he was going to be a brain surgeon. It's the smartest profession.

Well, except maybe rocket science.

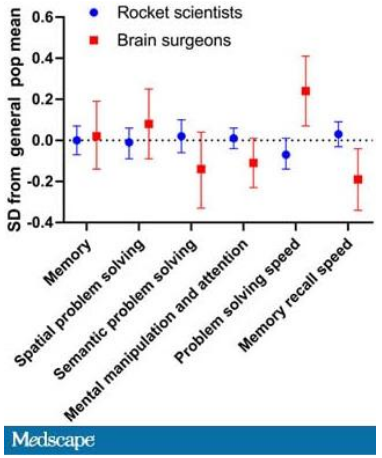
I mean, when you think of professions associated with intelligence, what else is there? What other professions have their own idioms associated with them? We say "it's not brain surgery" or "it's not rocket science" when we want to indicate that something is pretty straightforward. We don't say "it's not back-end development" or "it's hardly social media influencing."

But have you ever wondered, you know, who is smarter: the brain surgeons or the rocket scientists? Researchers from London led by Aswin Chari, who is a brain surgery trainee, set out to find the answer.

The results appear in *The BMJ.*

To coordinate the colossal cortex contest, the researchers turned to the Great British Intelligence Test, an online battery of tests of various cognitive domains from memory, to planning, to object manipulation. I tried it myself. It's pretty hard, but it's not contract law.

A total of 329 rocket scientists and 72 neurosurgeons participated and completed enough of the test to analyze. A large number of participants — 45% of rocket scientists and 51% of neurosurgeons — did *not* have enough data to analyze, suggesting that they quit the test early. So perhaps we're seeing the best of the best here? Or at least those with the most free time.



The test has also been taken by a bunch of regular shlubs like you and me so that the researchers could compare the results of the cognoscenti with those of the general population.

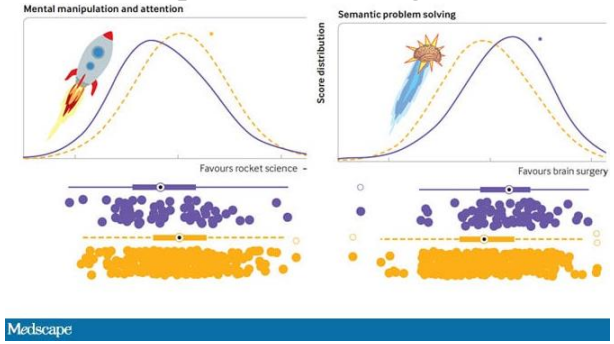
The methods are a bit tricky, using factor analysis to cluster the results of multiple tests into six domains of intelligence. This requires some statistical know-how, but hey, it's not structural engineering.

The most surprising thing about the results of this study? How, well, average all of these intellectual giants fared. As you can

see here, there were only two domains where either group differed significantly from the general population. Neurosurgeons did better on problem-solving speed, but this was balanced by faring a bit worse on memory-recall speed. The rocket scientists did about average in all domains.

Now, some of you will note that having six domains of intelligence, arrived at by factor analysis, doesn't *really* answer the question of who is smarter. Psychometricians have long postulated the existence of a single factor — G, or general intelligence — that explains the observed correlations in performance across a variety of cognitive tasks. We don't get an analysis of any such prime factor here, so perhaps the debate is unsettled. Still, comparing the two engagements with enlarged encephalons, we find that brain surgeons are better at semantic problem-solving and rocket scientists are better at spatial manipulation.

You can bring this up at your next cocktail party as a helpful test to see if anyone is actually listening to what you are saying.



In the end, this is a well-executed study. And though it's not exactly programming in C++, it reminds us that we shouldn't put too much stock in someone's profession when we are assessing their intelligence. Most of us are pretty average, despite what we may want to believe.

F. Perry Wilson, MD, MSCE, is an associate professor of medicine and director of Yale's Clinical and Translational Research Accelerator. His science communication work can be found in the Huffington Post, on NPR, and here on Medscape. He tweets @fperrywilson and hosts a repository of his communication work at www.methodsman.com.

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Credits Image 1: F. Perry Wilson, MD, MSCE Image 3: *The BMJ*

Image 4: F. Perry Wilson, MD, MSCE Image 5: *The BMJ*

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An Enemy Within: Researchers Discover How Pathogens Hide in Tissue

Where the bacteria hide in the body and how the body's own immune system also plays an important role

Antibiotics cure many bacterial infections. However, some patients suffer a relapse. A research group at the University of Basel has now discovered why some bacteria can survive antibiotic therapy. The team uncovered where the bacteria hide in the body and how the body's own immune system also plays an important role.

Infections such as tuberculosis or typhoid fever are caused by bacteria and can usually be treated well with antibiotics, at least as long as the bacteria are not resistant. However, full eradication of the bacteria cannot always be achieved. In some patients, a few bacteria survive the antibiotic therapy and can cause relapsing disease. For a long time, scientists have been trying to find out why antibiotics fail to kill all the bacteria.

Professor Dirk Bumann's group at the Biozentrum, University of Basel, has now shown, that it is not – as may be expected – due to dormant and therefore insensitive pathogens. Rather, there are certain areas in the tissue in which typhoid fever-causing Salmonella can survive more or less unaffected by the body's immune defenses. The researchers published their results in *PNAS*.

Examining tissue slice by slice

"After antibiotic therapy, only about every 100th bacterium survives," says Dirk Bumann, the study leader. "Tracking down and studying these few Salmonella in tissues is like looking for the needle in the haystack."

In order to manage this Sisyphean task, the researchers employed so-called serial two-photon tomography, a method used previously in neurobiology to detect the finest nerve fibers in the brain. The scanner device images the tissue surface and then cuts away the

uppermost layer. The new surface is scanned again followed by the next cut. In this way the instrument works its way, slice by slice, through the whole tissue. This provides the scientists with a detailed three-dimensional view of the tissue and reveals where the few surviving bacteria are located.

Hidden in the Police Headquarters

In their study, the researchers imaged spleens of infected mice. Most Salmonella live in the so-called red pulp of the spleen, the recycling station for red blood cells. “Here, Salmonella is almost totally eliminated during antibiotic treatment,” explains Jiagui Li, one of the three first authors of the study. Some Salmonella live also in another spleen region, the white pulp, where immune responses are normally initiated. In this region, however, antibiotic therapy is rather ineffective. The white pulp thus becomes the major home of surviving Salmonella. “It’s ironic, that pathogens hide in the body exactly where they should be caught as the culprit and an effective defense against them should be activated,” says Bumann.

Antibiotics alone are not enough

How do the bacteria survive in this surprising location? The scientists found that antibiotics alone cannot eradicate Salmonella from the tissue but needs the help of the immune system to clear all bacteria. In particular neutrophils, white blood cells that effectively fight bacteria, are critical. For successful eradication of Salmonella, neutrophils have to work together with the antibiotic for several days. In the white pulp, however, there are only few neutrophils and their number collapses during treatment. With fading support from host neutrophils, the antibiotic alone cannot eradicate the local Salmonella.

To overcome this problem, the research team has tried boosting the body’s defenses with the help of a simultaneously applied immune therapy. “This approach can help to stimulate the immune system and to maintain a high density of neutrophils over a longer time,” explains Bumann. Indeed, such adjunct therapy may lead to more effective clearance of the bacteria opening new avenues to prevent relapses.

Reference: “Tissue compartmentalization enables Salmonella persistence during chemotherapy” by Jiagui Li, Beatrice Claudi, Joseph Fanous, Natalia Chicherova, Francesca Romana Cianfanelli, Robert A. A. Campbell, and Dirk Bumann, 15 December 2021, Proceedings of the National Academy of Sciences. DOI: 10.1073/pnas.2113951118

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Why do we get side effects from vaccines? Experts say that means it’s working

Whether you’re getting your first shot, a second dose or a booster, you’re likely to experience some side effects.

By Madalyn Amato

The cooling sensation of an alcohol swab on your upper bicep is a cue for what’s about to come next: the injection of a needle that delivers a dose of vaccine.

More than 69% of Americans have received at least one dose of a COVID-19 vaccine, and as a bandage is applied to your arm, you are part of the club. Whether you’re getting your first shot, a second dose or a booster, you’re likely to experience some side effects.

They may be inconvenient, and they certainly can be uncomfortable. But immunologists and virologists say they are to be expected. And you might even welcome them. Here’s why, and what you should expect if you have a date with a needle.

Why do vaccines cause side effects?

To put it simply, the side effects are a biological sign that the vaccine is working.

All three of the COVID-19 vaccines available in the U.S. effectively work in the same way, said Dr. David Pride, an infectious disease specialist at UC San Diego.

The vaccines contain genetic instructions for making copies of the coronavirus’ spike protein. The Pfizer-BioNTech and Moderna vaccines use mRNA to carry the instructions, and the Johnson &

Johnson shot employs a disabled adenovirus, which is harmless.

Once the instructions are delivered, it's up to your cells to do the work. The fake spike proteins can't do any damage since they're not connected to actual coronaviruses. But your body will detect them and think an infection is underway, prompting the immune system to swing into action, Pride said. You can think of it as a training run.

The immune response comes in two parts. First, the innate immune system is alerted to the arrival of possible viruses or other pathogens in your body, said Dr. Peter Hotez, co-director of the Texas Children's Hospital Center for Vaccine Development. Then the adaptive immune system produces antibodies that can respond appropriately to the intruder — and, if necessary in the future, to an actual encounter with the virus.

The side effects you feel are a natural part of your immune system's response to the vaccine's viral payload.

What's normal and what's not?

Side effects to a COVID-19 vaccine are sometimes mild and sometimes more intense, like a bad cold or a case of the flu. Either way, they shouldn't last more than 72 hours.

The most common side effects are low-grade fever, fatigue, muscle pain, headache, chills and nausea, according to the Centers for Disease Control and Prevention. Temporary soreness at the injection site is also common. As Dr. George Rutherford, professor of epidemiology and biostatistics at UC San Francisco, puts it: You just got stuck with a needle; vaccine or not, that's going to hurt.

The clinic or pharmacy where you get your shot will ask you to hang around for 15 or 30 minutes after after your injection, so they can monitor you for rare but serious reactions, such as an acute allergic reaction that requires epinephrine.

Once you get home, you can take over-the-counter pain relievers such as Tylenol or Advil. (Don't take them before your shot because that might dampen the vaccine's effectiveness.) Beyond allowing your body to rest, there's not much else you can do while riding out normal side effects.

In rare cases, people — mostly teenage boys and young men — experience myocarditis (inflammation of the heart muscle) or pericarditis (inflammation of the tissue that surrounds the heart) in the week after getting a shot. Symptoms include chest pain, shortness of breath and feelings of having a fast-beating or pounding heart. If this happens to you, seek medical attention right away. In most cases, the conditions are easily treated with medicine and rest.

A very small number of people — mostly women under age 50 — who get the Johnson & Johnson vaccine may experience a serious side effect called thrombosis with thrombocytopenia syndrome, or TTS. Symptoms include chest pain, leg swelling, shortness of breath, headaches and abdominal pain. If you develop these symptoms after getting a J&J shot, see a doctor right away.

Any other symptoms that typically aren't associated with vaccines should be monitored. If they don't improve, call your doctor or go to the emergency room.

Does a lack of side effects mean the vaccine isn't working?

No. Some "lucky people" experience few or no side effects. But that doesn't mean their immune system isn't properly responding.

Hotez said there is no correlation between the severity of side effects and the strength of the immune response. In other words, having no side effects doesn't mean the vaccine is not working, and having more side effects doesn't mean it's working better.

Antibody tests can show how well your immune system has responded to a vaccine, but Pride said people who are immunocompromised are the only ones who need to worry about that. For most people, there's little chance the vaccine will fail to stimulate an immune response. Talk to your doctor if you're unsure.

Other things to know about vaccines

- ***It is impossible for you to get coronavirus from the vaccines.***
- ***Likewise, if you're feeling sick after getting a vaccine, you don't need to worry about being contagious and spreading COVID-19 to anyone else.***
- ***Booster shots are necessary, Hotez said. Vaccinations are almost always given in a series, and the COVID-19 vaccine is turning out to be no different. For the best immune response, you have to continue training your system, he said.***
- ***You have the option of getting a different type of vaccine for your booster shot than you did for your initial immunization. Whether you stick with the same brand or go with a mix-and-match approach won't make much difference in terms of side effects, Pride said.***
- ***Breakthrough infections in fully vaccinated people can occur, but it's rare for them to result in severe illness, hospitalization or death. You can check if you have an active infection by using an over-the-counter rapid test.***

https://bit.ly/3FkYZLe

Waiting 60 Seconds Before Cutting The Umbilical Cord Can Save Lives, Study Finds

Reducing the risk of death and disability in the first two years of childhood by almost one-fifth

Clare Watson

Waiting just 60 seconds to clamp umbilical cords gives very premature babies a better shot at life, reducing the risk of death and disability in the first two years of childhood by almost one-fifth, new research shows.

The Australian-led study, conducted in 25 hospitals across 7 countries, looked at health outcomes of more than 1,500 premature babies two years after they entered the world. Newborns who had their umbilical cords clamped one minute later – rather than immediately after birth – had better survival rates at two years of age.

"This is so significant as it is such a simple technique, suitable for almost all preterm babies that helps saves lives," obstetrician Jonathan Morris from the University of Sydney said in 2017 about the phenomenon, before the researchers began their latest study, tracking infant health for another two years.

By delaying cord clamping, more blood flows from the placenta to babies, with the extra red blood cells, immune cells, and stem cells thought to help newborns achieve healthy oxygen levels and control infections. This could be vital for the 1 million babies born at 30 weeks' gestation each year worldwide.

Previous research has suggested delaying cord clamping improves babies' chances of survival in the first days of life, with fewer babies dying in hospital. This latest analysis goes one step further, reporting infant health outcomes at two years of age for over 1,600 very premature babies, born 10 weeks early.

It's the largest-ever clinical trial comparing delayed and immediate cord clamping for very premature babies, born before 30 weeks and critically-ill. Maternity staff either held off clamping babies' umbilical cords for 60 seconds, or did so within 10 seconds of birth.

When the researchers combined their data on 1,531 babies with results from one other trial, taking the total to 1,637 infants, they found waiting more than 30 seconds to clamp cords reduced the relative risk of death and disability at two years of age by almost one-fifth.

Digging deeper into the results, this mostly reflects the better odds of survival newborns had if cord clamping was delayed – which reduced the relative risk of death by 30 percent – as there was no clear difference in major disability, such as cerebral palsy, vision loss, deafness, or speech problems at two years of age.

Delayed cord clamping is standard practice for full-term babies. Until recently, the umbilical cords of very premature babies were cut almost immediately after birth so the baby could receive urgent

medical care.

But an Australian study, first reported in 2017, also found fewer infants needed blood transfusions after birth if cord clamping was delayed. "Delaying cord clamping ensures that the physiological changes happening at the time of birth can happen and there are clearly very good outcomes, especially for premature babies," says midwifery expert Caroline Home of the Burnet Institute, who was not involved in the study.

The World Health Organization recommends delayed cord clamping for newborns who don't need immediate breathing support, although the practice is not always applied.

"It can be scary for clinicians to wait before they intervene," health researcher and biostatistician Anna Lene Seidler told *The Sydney Morning Herald* earlier this year.

A quarter of the babies in the delayed clamping group of the Australian trial actually had their umbilical cords cut before the 60-second mark, reflecting clinical concerns. "These babies are so tiny and sick, so for clinicians not to do something straight away requires quite a rethink," said Seidler, who led another recent analysis of 42 clinical trials involving more than 5,770 babies.

That analysis found delayed cord clamping was safe and slightly improved survival, though more evidence was needed to assess alternative strategies such as cord milking.

With the evidence mounting, researchers hope that reversing the decades-old practice of clamping cords of premature babies immediately after birth could improve health outcomes in the long run for thousands of kids.

"Applied consistently worldwide, aiming to wait a minute before cord clamping in very preterm babies who do not require immediate resuscitation, could ensure that an extra 50,000 survive without major disability in the next decade," says University of Sydney biostatistician Kristy Robledo, who led the analysis.

"In other words, for every 20 very preterm babies who get delayed instead of immediate clamping, one more will survive without major disability."

More data on the timing of cord clamping from a spectrum of clinical settings would help to solidify the findings, give clinicians more confidence, rule out potential harms, and track health outcomes further into childhood. "Intensive staff training in the new protocols will also be vital as it can be daunting to delay treatment in very early and sick babies, but the evidence suggests this results in the best outcomes for these children," says Morris. The research was published in *The Lancet Child and Adolescent Health*.

https://bit.ly/3yC2iey

When a Gene Illness Discovery Means Breaking Bad News

When scientists discover genes linked to dangerous illnesses in their samples, how should they convey that news to the study participants? The geneticist Cristen Willer had to tackle that challenge.

https://bit.ly/3E9ASOu

Fossil find reveals giant prehistoric 'thunder birds' were riddled with bone disease

Until around 45,000 years ago, Australia was home to Genyornis newtoni, a fearsomely huge bird weighing roughly 230kg – almost six times as much as an emu – and standing 2 metres tall.

Phoebe McInerney[*] Lee Arnold[**] Trevor H. Worthy[***]

This giant, from a unique group of Australian flightless birds called the dromornithids or "thunder birds", was among the largest birds that have ever lived. And then, along with many of Australia's other "megafaunal" species, it disappeared, for reasons that still remain debated.

Fossils of *Genyornis* are mainly found at the famous South

Australian fossil site of Lake Callabonna, which was first studied in 1893. This exceptional site preserves hundreds of megafaunal fossils, in the same location and in many cases the same exact body position in which they died after becoming stuck in the muddy lake bed.

New research, published in the journal Papers in Palaeontology, shows that getting stuck in the mud was not the birds' only concern. Bone infections also seem to have been common in this population – highlighting the challenges these birds were facing as their species began to die out.

The sickness

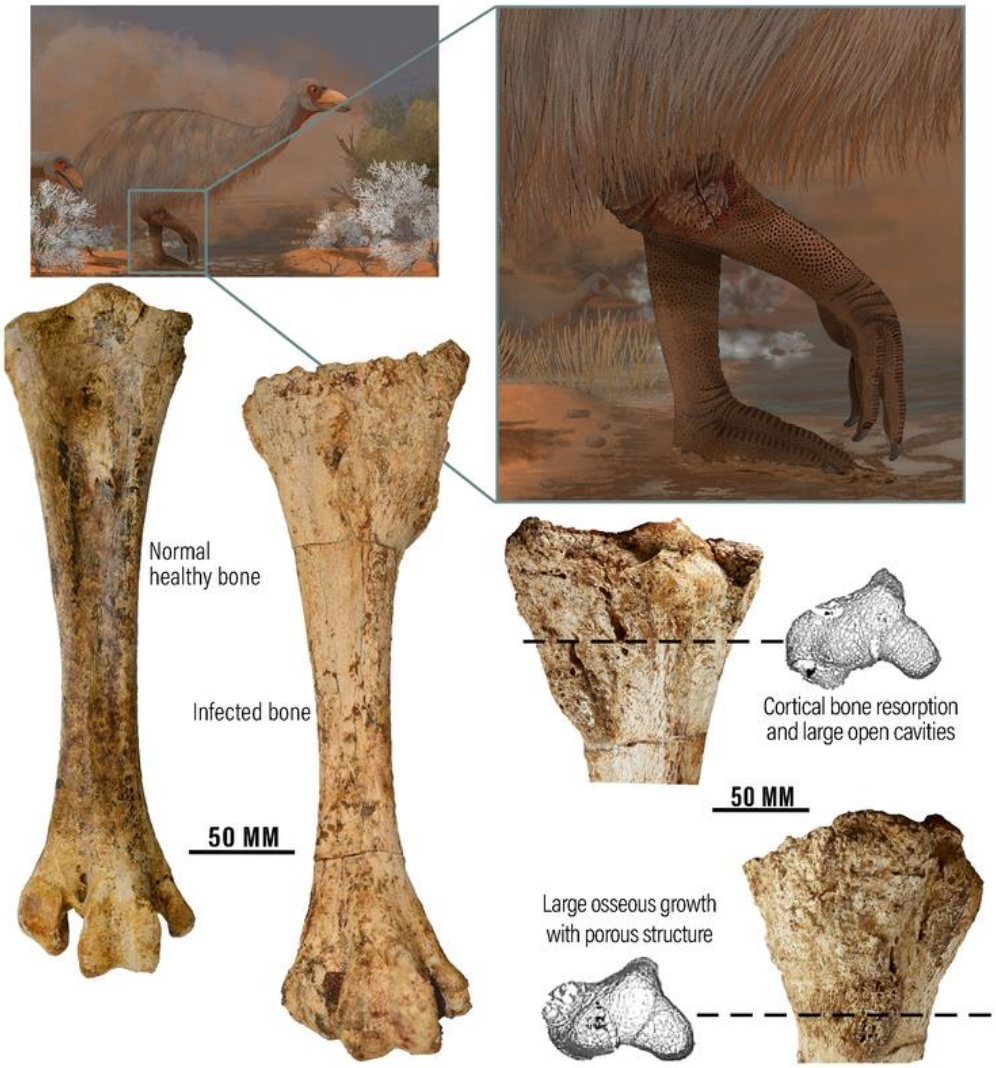
As we worked on the fossils in the Flinders University's palaeontology lab, we noticed several of the bones just didn't look quite right. They showed unusual distortions, cavities, and a "frothy" surface texture – all clear signs of abnormal bone infections.

We next looked inside the affected bones with the help of CT scans, which confirmed they had suffered abnormal development, distortion and destruction of their internal structure. Investigation into the type of illness that could cause such pathologies led to their diagnosis as osteomyelitis.

Osteomyelitis is a chronic bacterial infection of bone tissue, which can be caused either by trauma that lets microbes directly enter bone tissue, or via transmission from infected soft tissues nearby. It can cause serious damage.

Of the 34 partial skeletons of *Genyornis*, four showed signs of bone infections. But the real number is likely higher, because we couldn't assess all bones from all 34 individuals.

With the chest, leg and foot regions afflicted, individuals would have suffered pain and restricted mobility. As a result, finding enough water and food around the muddy lake beds of Lake Callabonna would have become an arduous task.



Infection on the leg of Genyornis newtoni and a life reconstruction of the injured bird. **Credit: PL McInerney**

Disease and drought

These birds seem to have suffered an unusually high rate of bone disease, compared with today's birds. This suggests the disease was not random, but instead was associated with a particular

environmental cause – but what?

One way to help answer this question is to date the fossils accurately, and then to compare their plight with what we know was happening to the environment at Lake Callabonna at the time.

Calculating the age of these intriguing fossils is not necessarily straightforward because, like many of Australia's extinct megafauna, they are too old for the classic radiocarbon dating method to work.

So we used an alternative dating technique called single-grain optically stimulated luminescence, which reveals when sand grains in the surrounding lake sediments were deposited. This provides a useful estimate of when the birds became mired in the mud.

As this dating technique applies to sediments rather than bones, it can also be used to reveal the lake history. In particular, it can distinguish between times when the lake was full of water and was accumulating mud on the lake floor, and times when it was much drier and was accumulating wind-blown sands.

Our study revealed that the beleaguered *Genyornis* population met its demise getting stuck in sediments laid down between 54,200 and 50,400 years ago. Sediments dated from Lake Callabonna and nearby lake systems reveal that a protracted drought phase began around 50,000-46,000 years ago. After this time, the permanent and extensive water body was transformed into the dry lake bed seen today.

This suggests the birds' fate was sealed once the lake began to dry up. The population became trapped in the freshly exposed lake floor muds as they searched for ever-diminishing water supplies.

A role in their extinction?

The rare preservation of *Genyornis* fossils at Lake Callabonna offers an extraordinary opportunity to investigate the impact of environmental change on this now-extinct population.

When resources are limited, as they would have been during these severe droughts, birds can initiate a stress response that helps them survive until the next time of plenty. But in the long term, this stress response directs resources away from the immune system, ultimately increasing the birds' susceptibility to infection and disease. Thus, it is perhaps no surprise the *Genyornis* bones bear the hallmarks of severe disease.

There is no conclusive evidence that *Genyornis* survived for long beyond this time. The drying-out of the lakes they called home may have ultimately sealed their extinction fate.

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https://bit.ly/3yLUGGE

Scientists Create "Time Machine" Made of Human Cells To Reverse Pancreatic Cancer Progression

"Time machine" has shown a way to reverse the course of cancer before it spreads throughout the pancreas

What makes pancreatic cancer so deadly is its covert and quick spread. Now, a "time machine" built by Purdue University engineers has shown a way to reverse the course of cancer before it spreads throughout the pancreas.

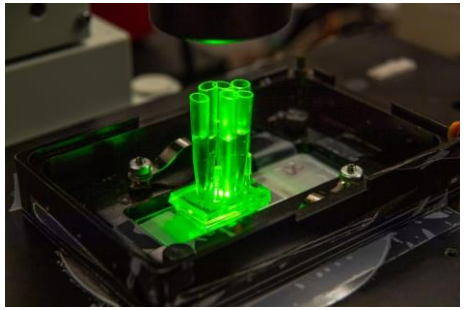
"These findings open up the possibility of designing a new gene therapy or drug because now we can convert cancerous cells back into their normal state," said Bumsoo Han, a Purdue professor of mechanical engineering and program leader of the Purdue Center for Cancer Research. Han has a courtesy appointment in biomedical

engineering.

The time machine that Han's lab built is a lifelike reproduction of a pancreatic structure called the acinus, which produces and secretes digestive enzymes into the small intestine. Pancreatic cancer tends to develop from chronic inflammation that happens when a mutation has caused these digestive enzymes to digest the pancreas itself.

If there were a way to go back in time to reprogram the cancerous acinar cells that produce those enzymes, then it might be possible to completely reset the pancreas.

For the past decade, Stephen Konieczny, professor emeritus in Purdue's Department of Biological Sciences, has studied a potential reset button: a gene called PTF1a.



Purdue researchers used this experimental setup to reprogram pancreatic cancer cells back into their normal state. **Credit: Purdue University photo/John Underwood**

"The PTF1a gene is absolutely critical for normal pancreas development. If you lack the PTF1a gene, you don't develop a pancreas," Konieczny said. "So, our whole idea was, if we turn the PTF1a gene back on in a pancreatic cancer cell, what happens? Will we revert the cancer phenotype? Indeed, that's exactly what happens."

Konieczny collaborated with Han's lab to take these findings in molecular biology studies to the next level by testing them in a realistic model of the acinus – the time machine. The published study is featured on the cover of a recent issue of *Lab on a Chip*, a journal by the Royal Society of Chemistry.

Researchers typically investigate possible pancreatic cancer treatment approaches in animal models, but it can take months for pancreatic cancer to develop in an animal. Having a way to study cancer development and treatment concepts in a microenvironment that is just as realistic would save time and give researchers more control over the model.

The model that Purdue researchers developed overcomes a major challenge in accurately capturing the anatomical complexity of the acinus, a circular cavity lined with cells.

"From an engineering perspective, creating this kind of three-dimensional cavity is not trivial. So, figuring out a way to build this cavity is an innovation in itself," Han said.

Han's lab already had experience building a realistic model of another pancreatic structure, the duct, where cancer grows after emerging from the acinus. The researchers took this knowledge and developed a new technique that builds both the duct and acinus in a two-step "viscous fingering" process.

Here's how it works: The model, a postage stamp-size glass platform on top of a microscope slide, has two interconnected chambers. Loading a collagen solution into one chamber fills the finger-like shape of a pancreatic duct, which bulges and then expands to create the cavity structure of the acinus in the second chamber.

Dropping cancerous human cells into the acinar chamber made the model even more realistic. Konieczny's lab engineered the PTF1a gene of a pancreatic cancer cell line to turn on in the presence of doxycycline, a compound commonly used in antibiotics. Once the gene was activated, the cells started constructing the rest of the acinus in Han's model, indicating that they were no longer cancerous and had been reprogrammed.

"In this model, not only do the cancerous cells become reprogrammed, but for the first time, we're able to show the normal three-dimensional architecture of the acinus, which looks very similar to the same structures we see in a healthy pancreas,"

Konieczny said.

Han's lab is currently conducting experiments exploring a possible gene therapy based on these findings.

Reference: " Engineering of a functional pancreatic acinus with reprogrammed cancer cells by induced PTF1a expression" by Stephanie M. Venis, Hye-ran Moon, Yi Yang, Sagar M. Utturkar, Stephen F. Konieczny and Bumsoo Han, 9 August 2021, Lab on a Chip.
DOI: 10.1039/D1LC00350J
This study was partially supported by grants from the National Institutes of Health, the Walther Embedding Program in Physical Sciences in Oncology, and the Purdue Center for Cancer Research, which is one of only seven National Cancer Institute Basic Laboratory Cancer Centers in the nation.

https://bit.ly/3J2UZkO

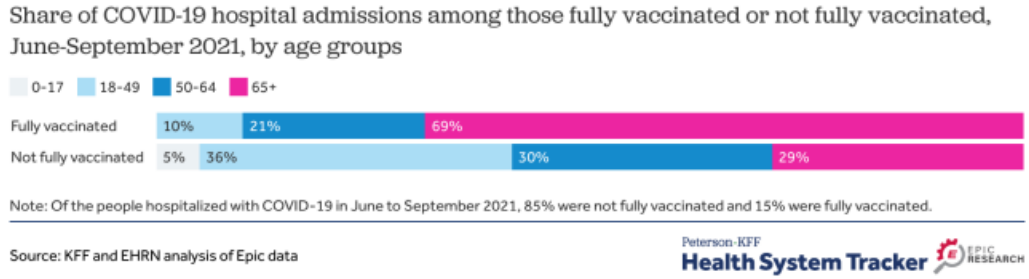
"Breakthrough" COVID-19 Hospitalizations Among Fully Vaccinated Patients Occur Most Often among Older Adults and Involve People with Chronic Health Conditions

Compared to Hospitalizations of Unvaccinated Patients, Breakthrough Cases Involve Shorter Stays on Average and Appear More Likely to Be Hospitalized Primarily for Non-COVID Ailments

"Breakthrough" hospitalizations involving COVID-19 among people who are fully vaccinated against the disease most often affected older adults and people with other chronic health conditions, finds a new analysis of hospital data from June through September by KFF and Epic Research.

More than two-thirds (69%) of breakthrough COVID-19 hospitalizations occurred among people ages 65 and older, who are more likely than younger age groups to have gotten vaccinated. A fifth (21%) of breakthrough hospitalizations occurred among people ages 50-64, while 10% occurred among younger adults.

COVID-19 hospitalizations among people who were not fully vaccinated skew much younger, with about 3 in 10 (30%) involving patients ages 50-64 and 4 in 10 (41%) involving patients under age 50.



The analysis examines data from June to September from Epic's Cosmos research platform, which includes data for more than 120,000 hospitalizations with a COVID diagnosis during the four-month period. Patients are considered "fully vaccinated" if they received a dose of Johnson & Johnson vaccine or two doses of the Pfizer or Moderna vaccine at least two weeks before they were hospitalized, regardless of whether they were eligible for or received a booster shot.

Compared to those who are unvaccinated, a small share (15%) of hospital admissions for COVID-19 during the four-month period involve people who were fully vaccinated against the disease.

Other findings include:

**** Larger shares of fully vaccinated adults hospitalized with breakthrough COVID-19 had selected chronic conditions including, hypertension, diabetes, heart failure, or chronic obstructive pulmonary disease) compared to those hospitalized with COVID-19 who were not fully vaccinated.***

**** Fully vaccinated people who are hospitalized with breakthrough COVID-19 are less likely than those who are not fully vaccinated to have COVID-related complications such as viral pneumonia or respiratory failure, or to receive a ventilator or dexamethasone treatment. This suggests that fully vaccinated patients with COVID-19 diagnoses may be somewhat more likely to be in the hospital primarily for reasons other than COVID-19.***

**** Fully vaccinated people with breakthrough infections had shorter hospital stays compared to others in their age group who were not fully vaccinated. For example, among those at least 65 years old with COVID-19, the median stay was 5.6 days for those who were fully vaccinated compared to 6.7 days for those who were unvaccinated or partly vaccinated.***

The analysis is available on the Peterson-KFF Health System Tracker, an online information hub dedicated to monitoring and assessing the performance of the U.S. health system

https://bit.ly/3J5ADra

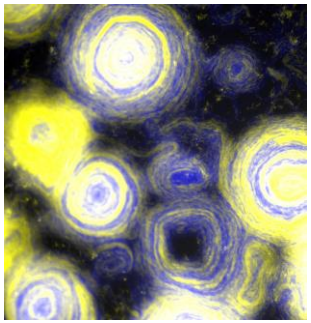
Mutant Bacteria Accidentally Recreated One of Van Gogh's Most Iconic Paintings

The line between art and science is sometimes a swirly one.

Carly Cassella

Researchers studying a social bacterium that moves and feeds in coordinated swarms have unintentionally recreated something that looks a lot like a familiar masterpiece.

When a certain gene is overexpressed in a bacterium known as *Myxococcus xanthus*, the individual organisms self-organize into tiny circular swarms within hours.



Above: A mixture of two strains of myxobacteria, one that overexpresses TraAB (yellow) and another that is non-adhesive and non-reversing (blue) at x10 magnification. **(D. Wall/University of Wyoming)**

Once the resulting swarms are artificially colored, the scene looks remarkably similar to Van Gogh's *The Starry Night*.

"Our work highlights how a social bacterium, known for rich sources of therapeutic natural products and as crop biocontrol agents, serves as a powerful model for studying emergent behaviors that also exhibit artistic beauty," says microbiologist Daniel Wall from the University of Wyoming.

Bacteria have a reputation for being selfish, but *M. xanthus* is described as a social bacterium because it needs to find and recognize relatives to survive.

The Starry Night. **(Vincent van Gogh/Wikimedia Commons/Public Domain)**

After it has formed big, familial clumps, this rod-shaped bacterium is much better at attacking its prey to feed. Each cell produces digestive enzymes that facilitate predatory feeding.

Researchers have been fascinated by this social behavior for years now, but we still don't have a comprehensive and broadly accepted model for their complex movements.

In 2017, Wall and his colleagues announced the discovery of a single genetic 'switch' responsible for turning this grouping behavior on and off.

The switch specifically controls for a protein sequence, known as TraA, which provides a surface receptor for the bacterium to recognize and attach to the partner receptor, TraB, on its kin.

Once it has glued itself to a family member via these two receptors (TraAB), the bacterium can then exchange nutrients and proteins with the rest of the group. When the swarm encounters food, lab research shows the organisms can actually pool their enzymes and metabolites together via these connections to give the most powerful punch to their prey.

But all of that changes when the team induced mutant bacteria to overexpress TraAB connections. This connection is what allows the cells to stick together in the first place, but when there's too much of this 'social glue', the swarm can't break apart as easily to change its shape or direction.

"In normal wild-type cells, they go back and forth, back and forth, like a commuter train," explains bioengineer Oleg Igoshin at Rice University. "The head becomes the tail and the tail becomes the

head. And they do it every 8 minutes or so."

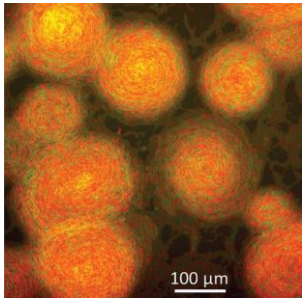
An overexpression of TraAB, however, seems to stop the swarm from switching its head to its tail and vice versa.

This is what computational models suggested would happen, but the authors still couldn't figure out why. As far as they knew, the TraAB connection wasn't directly involved in the regulation of the swarm's movements, only its stickiness.

Ultimately, the team suspected the sticky quality of TraB was indirectly stopping the swarm of cells from changing direction.

"Our idea was maybe there is some sort of contact-dependent signal between cells that suppresses the reversals," explains Igoshin.

"The cells are in dense groups and are in contact with others all the time, but those contacts are transient. But if TraAB overexpression really makes you sticky, your neighbor will remain your neighbor for longer, and that could trigger the signal that suppresses the reversals."



Above: Two myxobacteria strains that overexpress different types of TraA receptors (red and green) that adhere to themselves but not each other. **(D. Wall/University of Wyoming)**

Running this scenario in computational models, the authors were able to verify their hunch. With only changes to the TraAB connection, the usual head-to-tail swarms suddenly became rotating swirls of cells, as large as a millimeter or more.

Further experiments in the lab then confirmed this also happened to the bacteria in real life. Specifically, the swirls can occur when a strain overexpresses stickiness, but also when a strain is genetically modified to be directly 'non-reversing'.

The result is not only a better understanding of how millions of cells coordinate their movements, it's also a mesmerizing picture of the microbial world. The study was published in *mSystems*.

https://bit.ly/3JeuGsn

The Common Mouth Microbe That Keeps Popping Up in Tumors

Lab studies link the oral bacteria Fusobacterium nucleatum to cancers from the gut to the head and neck. Could targeting the microbe tackle tumors?

David Adam

Might brushing your teeth protect against cancer? The suggestion looks like it belongs in the pages of an unreliable tabloid, but scientific evidence for the link is strong and growing.

Take head and neck cancer, which kills some 450,000 people worldwide every year. It's associated with smoking and drinking alcohol, which is one reason why the most common form of the disease, oral squamous cell carcinoma (OSCC), tends to cluster in under resourced areas. But plenty of people diagnosed with OSCC say they never drank nor smoked, so researchers have been looking for other possible causes.

One likely candidate is gum disease. A series of studies have identified periodontitis, a bacterial infection that eats away soft tissue and eventually bone around teeth, as a risk factor for OSCC. That might be because the disease changes the behavior of usually benign bugs that live in the mouth.

A study published late last year, for example, showed that mice infected with oral bacteria developed significantly larger and more numerous tumors compared to those not infected.

"The moment they sense that there's some problem in the mouth, or that there is a decrease in the immune system, they respond and attack, because they're looking for food," says Jorge Frias-Lopez, a microbiologist at the University of Florida's school of dentistry who studies the link between the oral microbiome and cancer.

And of the 700 or so bacterial species typically found in our mouths, scientists studying OSCC have zeroed in on a spindle-shaped

suspect called *Fusobacterium nucleatum*. It's early days, but researchers think *F. nucleatum* could explain why gum disease is linked to the development of oral tumors.

"All of the signs are leaning towards that this bacterium is in some way involved," says Daniel Slade, a biochemist at Virginia Tech who studies the role bacteria play in cancer. "But it's still an open question and needs more research on whether it can initiate cancer, or whether or not it's accelerating cancer."

Bacteria and tumors

Infectious microbes are reckoned to contribute to some 20 percent of human tumors. Viruses tend to soak up most of the blame, from the common human papillomaviruses that lead to cervical cancer to hepatitis B and C, which raise the risk of liver cancer.

"The concept that bacteria are important in cancer is new," says Yvonne Hernandez-Kapila, a periodontologist at the University of California, San Francisco. When a link between colon cancer and the bacteria *H. pylori* was discovered in the 1990s, it triggered a search for other pathogenic and perhaps carcinogenic types, she says. "Then large population studies began to see some associations between bacteria, especially oral bacteria, and some cancers."

Such association studies make up the bulk of the evidence that currently links OSCC to *F. nucleatum*. Starting in 1998, research on people with cancer has shown time and again that levels of the bacteria and bacterial gene expression are higher in OSCC tumors than in normal tissue.

"*Fusobacterium nucleatum* is actually present in many people," says Hernandez-Kapila. "However, the relative numbers increase in cancer patients. We've shown that in oral and head and neck cancer patients."

Association studies can only identify a correlation between the bacteria and disease, and famously, correlation does not equal causation. Without longitudinal studies to examine whether people with higher numbers of the bacteria go on to develop higher rates of cancer, scientists struggle to determine whether the bugs perhaps cause and worsen the disease, or if they are simply found alongside tumors. "There might be a role for *Fusobacterium* in promoting cancers, but I think it's a kind of chicken and egg question," says Miguel Reis Ferreira, a clinical oncologist at Guys and St Thomas NHS Foundation Trust in London.

But scientists like Slade and Hernandez-Kapila believe that *F. nucleatum* does contribute to cancer. That's because studies have made connections between elevated *F. nucleatum* numbers and cancer in other parts of the body where the bacteria isn't normally found. Scientists think *F. nucleatum* gets from the mouth to the colon, breast, and other places where it is linked to cancer when it enters the bloodstream through bleeding gums.

In 2012, well before the association between the bacteria and OSCC was spotted, *F. nucleatum* was found to be prevalent in human colorectal carcinoma (CRC). "The link is really strong in gastrointestinal cancer," says Frias-Lopez. Reis Ferreira agrees that the evidence that the bacteria plays a role in CRC is relatively strong, both because of the number of studies that have made the connection and for a very simple physiological reason: "The bacteria shouldn't be there."

Compared with other cancers, there has since been much more research, including in animals and cell culture, into how the bacteria might raise the risk of CRC. "Mechanistic studies of these bacteria and colon cancer have been happening for years now and a few things have emerged," says Robert Holt, a genomic scientist and immunogeneticist at the BC Cancer Research Center in Vancouver.

One important mechanism seems to be how *F. nucleatum* can attach itself to a sugar molecule called Gal-GalNAc that is overexpressed on the surface of many cancer cells.

Slade says, "the bacteria are not necessarily picking specific cancers. But if cancers are expressing the sugar on the surface, then they are able to bind to it." In addition to colon tumors, studies show that *F. nucleatum* can also bind breast cancer cells in this way. Once bound, the bacteria could aid cancer progression in several ways. *F. nucleatum* could act as a bridgehead to allow other bacteria to colonize tumors, helping to generate biofilms within which microorganisms aggregate and interact.

"There are lots of other different bacteria, especially in the oral environment. So, does it have a partner that we're missing, or is it able to do this alone?" Slade adds. "I think that's an area that is really going to explode in the near future."

There is evidence that proteins expressed by *F. nucleatum* interfere with cell signaling processes, which can influence the progression of tumors. Some studies show the bacteria rev up cancer cell proliferation and can decrease levels of DNA repair proteins.

Inflammation could play a role as well. *F. nucleatum* is known to trigger a powerful inflammatory immune response. And chronic inflammation is associated with both the onset and progression of cancer at various sites in the body. A study published in September of this year that analyzed human colorectal tumors suggested that *F. nucleatum* might even help cancer spread from site to site through the body.

Possible treatments

Uncertainty around causality shouldn't delay work on possible clinical implications of a link from the bacteria and tumors in the mouth or elsewhere, says Holt. "It doesn't hurt at this stage to be exploring approaches to intervene," he says. "It's unlikely that removing these bacteria would be negative. There is nothing good that this bug appears to be doing. But it does do a lot of bad."

Antibiotics are the most obvious way to attack bacteria, but are a nonstarter for cancer, Slade says. "One problem with that is it is very difficult to create a specific antibiotic," he says. "With broad spectrum antibiotics you will be wiping out potentially good parts of our microbiome." Research suggests that patients with a healthy microbiome respond better to cancer chemotherapy. "It could be that while you are trying to eliminate this bacterium to prevent or treat cancer, you also remove a good subset of bacteria that actually allows cancer treatment to perform better," Slade says.

Hernandez-Kapila is looking instead to combat *F. nucleatum* with nisin, an antimicrobial peptide produced by the bacteria *Lactococcus lactis*. Nisin is a tested and approved preservative in the food industry, and encouraging reports of its anticancer effects in animal studies have led to some cancer patients taking it. Hernandez-Kapila is now trying to raise funds to carry out a proper clinical trial.

Another promising strategy is to develop a vaccine, Holt says. His group is trying to identify possible antigen targets for a vaccine against *F. nucleatum* that could be given to cancer patients who have responded well to treatment but who have a high genetic risk of tumor reoccurrence. "That would be a good scenario where we could potentially see good vaccine efficacy," he says. "It would truly be experimental at this stage, but there are some good reasons to be pursuing this."

If *F. nucleatum* does promote the development of tumors across the body, from the gut to the head and neck, then such a vaccine could address many cancer types. "The pharma industry has adapted well to now considering very precise indications and more personalized indications. So in a sense we would be taking things in the other direction, of potentially a broader application," Holt says.

Hernandez-Kapila says a growing number of scientists are realizing that solid results from lab and animal studies suggest that targeting pathogenic bacteria like *F. nucleatum* could offer a new route to

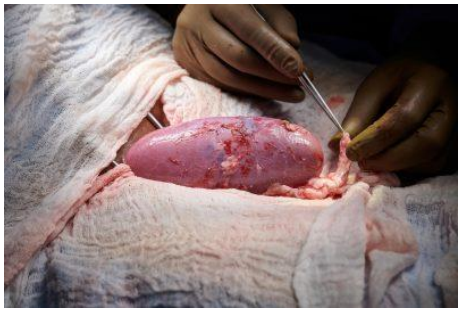
addressing cancer in the head and neck and elsewhere. “I’ve spent my whole career on this, and people used to tell me it was nonsense,” she says. “But radiation and chemo are very difficult for patients to take, and if you can find something like a probiotic that is very selective and doesn’t cause as many off-target effects, then that would be very helpful.”

https://bit.ly/3ecxjMF

Successful Xenotransplantation Surgery: Genetically Engineered Pig Kidney Transplanted to Human Body

Second successful investigational xenotransplantation procedure using a genetically engineered pig kidney

Less than two months after the first breakthrough surgery, NYU Langone Health has performed its second successful investigational xenotransplantation procedure using a genetically engineered pig kidney. This second surgery is a sign of continued progress toward a potential alternative supply of life-saving organs.



The porcine kidney appears healthy and following perfusion and the ureter is prepared to allow for urine production. **Credit: Joe Carrotta / NYU Langone Health**

Leading the second surgical procedure was Robert Montgomery, MD, DPhil, the H. Leon Pachter, MD, Professor and chair of the Department of Surgery at NYU Langone and director of the NYU Langone Transplant Institute. He transplanted a pig kidney lacking the alpha-gal gene to a recently deceased donor maintained on a ventilator. LiveOnNY, the nonprofit organization that facilitates organ and tissue donation in the greater New York City area, assisted in identifying a generous whole-body donor to help move this landmark research forward.

“We have been able to replicate the results from the first transformative procedure to demonstrate the continued promise that these genetically engineered organs could be a renewable source of organs to the many people around the world awaiting a life-saving gift,” says Montgomery. “There is much more work to do before we begin living human trials, but our preliminary findings give us hope.”

The procedure, part of an ongoing study, was performed on Monday, November 22, 2021, at an NYU Langone research laboratory in Manhattan. The kidney was procured from a GalSafe™ pig engineered by Revivicor, Inc., a subsidiary of United Therapeutics Corporation. The gene that encodes the glycan known as alpha-gal—which is responsible for a rapid antibody-mediated rejection of porcine organs by humans—was “knocked out” in the donor pig. The pig’s thymus gland, responsible for “educating” the immune system, was fused with the kidney before transplantation.

The kidney was attached to the blood vessels in the upper leg, outside the abdomen, and covered with a protective shield for observation and kidney tissue sampling over a 54-hour period of study. Urine production and creatinine levels—key indicators of a properly functioning kidney—were normal and equivalent to what is seen in a human kidney transplant. Throughout the procedure and subsequent observation period, there were no signs of rejection.

“We continue to make progress with the single-gene knockout xenotransplantation,” says Montgomery. “With additional study and replication, this could be the path forward to saving many thousands of lives each year.”

There are currently more than 90,000 people in the United States awaiting a life-saving kidney transplant, according to the United Network for Organ Sharing.

GalSafe™ is a trademark of Revivicor, Inc.

https://nyti.ms/3e81h4I

New Studies Raise Hopes That Vaccines Prevent Severe Disease From Omicron

In the lab, immune cells put up a strong fight against Omicron, suggesting that vaccines will be able to prevent the worst outcomes of the virus variant.

By Carl Zimmer and Sheryl Stolberg

A flurry of new laboratory studies indicate that vaccines, and especially booster shots, may offer protection against the worst outcomes from the fast-spreading Omicron coronavirus variant. The highly mutated virus, however, will still cause many breakthrough infections in vaccinated people and in those who have been infected with older versions of the virus, according to the research.

At a World Health Organization meeting on Wednesday, scientists reported on several studies suggesting that T cells in vaccinated people can put up a strong defense against the variant, which could help prevent severe disease, hospitalization and death.

Also on Wednesday, Dr. Anthony S. Fauci, President Biden's top medical adviser for the coronavirus response, shared preliminary data from his institute's analysis of the Moderna vaccine. While two shots produced a negligible antibody response against Omicron in the laboratory, the protection shot up after a third dose, he said.

Other researchers at the W.H.O. meeting presented similar results, showing that booster shots of either Moderna or Pfizer-BioNTech mRNA vaccines lifted antibodies back to levels believed high enough to offer strong protection against infection.

Though the research is based on preliminary observations of cells in the laboratory, it is nevertheless a welcome departure from a torrent of worrying new data about Omicron. Over the past week, it has become increasingly clear that Omicron can deftly evade antibodies, part of the body's first line of defense, which probably explains why infections with the variant have exploded in many countries.

But antibodies are not the only important players in a person's immune response to the virus. T cells have their own role.

"The good news is that T cell responses are largely maintained to Omicron," said Wendy Burgers of the University of Cape Town during a presentation of new research she and her colleagues have carried out in recent days.

Omicron infections are happening more frequently in two groups of people who carry antibodies: those who have received shots, as well as those who aren't vaccinated but have recovered from an earlier infection with the coronavirus.

This week, scientists in South Africa reported that two doses of the Pfizer vaccine were 33 percent effective against an Omicron infection, down from about 80 percent during what Dr. Fauci called "the pre-Omicron era." The study found that two doses of the Pfizer vaccine offered 70 percent protection against severe hospitalization and death, down from about 95 percent before Omicron was detected.

At Wednesday's W.H.O. meeting, one scientist after another presented similar laboratory findings showing that vaccine-induced antibodies performed much worse against Omicron than against other variants.

But boosters seem to provide enough extra antibodies to lessen these infections. Dr. Fauci described experiments at the National Institutes of Health, in which scientists took blood serum from people who had two doses of the Moderna vaccine as well as from others who had a third dose. The researchers then mixed the serum with viruses engineered to carry Omicron's surface proteins.

These "pseudoviruses" evaded many antibodies from people who had received two doses of Moderna, but the boosters produced such high levels of antibodies that the viruses were blocked from invading cells.

"So the message remains clear: If you are unvaccinated get

vaccinated, and particularly in the arena of Omicron, if you are fully vaccinated, get your booster shot," Dr. Fauci said.

Dr. Fauci's admonition comes as Biden administration officials are bracing for a potential wave of Omicron infections that could overwhelm the health care system. The Centers for Disease Control and Prevention warned recently that the percentage of coronavirus cases in the United States caused by the Omicron variant had increased sharply and might portend a significant surge in infections as soon as next month. The Delta variant remains by far the dominant version across the United States.

In anticipation of that wave, the administration is trying to encourage all Americans who are eligible — those 16 and older who received their second vaccine dose at least six months ago — to get their booster shots. About 27 percent of fully vaccinated Americans have also had booster shots, according to the C.D.C.

Many countries are rushing boosters to their populations, but Omicron is spreading so fast it may well outstrip even the best efforts.

"The projected transmission rates, if borne out, do not give us much time for interventions," Phil Krause, a former vaccine regulator at the Food and Drug Administration, said at the W.H.O. meeting.

That prospect has led many scientists to hope that T cells will serve as an effective backup when antibodies fail. If these immune cells can fight Omicron, they may prevent many infections from turning into severe disease.

After a cell is infected with the coronavirus, T cells can learn to recognize fragments of viral proteins that end up on the cell's outer surface. The T cells then kill the infected cell, or alert the immune system to launch a stronger attack against the virus.

Dr. Alessandro Sette, an immunologist at the La Jolla Institute for Immunology, and Andrew Redd of the National Institutes of Health reported that despite Omicron's many mutations, most of the protein fragments recognized by T cells are identical to those of other variants.

Those findings suggest that T cells trained by vaccines or previous infections will respond aggressively to Omicron, rather than standing by. "It appears the T cell response is largely preserved," Dr. Sette said.

Dr. Burgers and her colleagues tested that possibility by collecting T cells from 16 people vaccinated with two doses of the Pfizer-BioNTech vaccine and exposing those T cells to protein fragments from the Omicron variant. The scientists found that the response of the T cells to the variant was about 70 percent as powerful as their response to the original form of the virus.

A number of scientists at the meeting cautioned that these data come from studying cells in a laboratory, known as in vitro experiments. It will take a few more weeks of examining infections in people before it becomes clear how well T cells prevent severe disease.

"We don't know yet what these in vitro findings actually mean for disease severity," said Nora Gerhards, a virologist at Wageningen University in the Netherlands. "And that's what it's all about. Because in the end we want to prevent a collapse of the health care systems in our countries."

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Vaccine trial finds a glitch with children in one age range

Company is adding a third dose to the trial after finding a low immune response.

John Timmer

On Friday, Pfizer and BioNTech announced that their latest vaccine trial was showing some odd results in children within a specific age range. Children in the 2- to 5-year age group didn't produce as strong of an antibody response to the vaccine as older and younger

children did. As a result, the trial is being modified to include a third dose of vaccine for participants in this age group.

The trial was designed to enroll as many as 4,500 children to test the safety and efficacy of the companies' messenger RNA vaccine. It included an early test of how well the vaccine was tolerated in different age groups. Based on these results, the companies went ahead with a two-tiered strategy: children from 5 to 11 years of age got two doses of 10 µg; younger children (down to six months in this trial) received two doses of 3 µg.

The trial is ongoing, and both the participants and doctors involved remain blinded to the status of the participants. But blood samples were obtained from some participants one month after the second dose and analyzed by a separate group of researchers who were not blinded as to the vaccine/placebo status of the participants. The analysis they performed showed an unexpected pattern.

The baseline for a successful response was set as being equivalent to the results in those ages 16 to 25 years, where high efficacy was already demonstrated. An equivalent level of response was seen in those from 6 months to 2 years of age. But for those older than 2 years and younger than 5 years old, the dosing didn't generate as robust of a response.

The companies' current plan, already accepted by the US Food and Drug Administration and the European Medicines Agency, is to wait at least two months after the second dose, and then give a third 3 µg shot in order to boost the immune response further.

The companies emphasized that the trial demonstrated that the 3 µg dose is safe in all age groups it has been used on. And the company hasn't provided details on how the immune response was measured. It's likely that it was done by checking for antibody levels, which have strongly correlated with protection, but are only part of the immune system's response. Also lacking are details on the magnitude of the difference between the response in trial participants and that in older populations. So, it's possible that there is a significant immune response, but it's simply not as strong.

In any case, simply extending the trial will mean that data won't be available to submit to the FDA until later next year. Which, for a lot of anxious parents, will undoubtedly be frustrating.

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Why Invasive Plants Pushing Out Native Flora Is Pushing Us Closer to a 'New Pangaea'

According to the first global analysis of plant diversity, the world's flora is growing increasingly uniform, even on isolated islands like Australia.

Carly Cassella

For decades now, scientists have been warning the world we are headed for a new geological epoch, called the 'Homogecene', when unique life forms become overshadowed by more adaptable species that can live alongside humans.

The new research on flowering plants reveals the extent to which that may already be happening to some flora.

"These effects are now evident even in the most remote corners of the world," says ecologist Mark van Kleunen from the University of Konstanz in Germany.

"Unless more effective protective measures are taken to counter the ongoing spread and naturalization of alien plants in the future, they will continue to destroy the uniqueness of our ecosystems—making the world a less diverse place."

This destruction of ecosystems is largely thanks to us. Humans have collapsed the distance between ecoregions worldwide, and some scientists are concerned that the loss of natural barriers could one day create a 'New Pangaea'.

Instead of solid land connecting all the major continents and their flora and fauna, the bridge this time will be us. On our backs already flow numerous super-invaders, ready to take over new

territory and displace native species.

Their domination has begun.

Blackberries, for example, growing feral in Australia, impact at least 47 threatened species through reduction of habitat and by providing shelter to other introduced predators and competing species. They cost hundreds of millions of dollars in damage and containment attempts.

Stronger biosecurity measures for human trade and transport could help protect the native vegetation that's left on our planet for hotspots like Australia and other Pacific islands.

Isolated nations like these are home to many unique endemic species, and yet because these life forms have evolved to suit a very specific ecological niche, they are least likely to adapt to a rapidly changing world.

Drawing on floral data from 658 regions around the world, including 189,762 flowering-plant species, researchers have now broadly compared how native flowers are coping compared to invasive flowers.

Over time, their findings suggest geographically distant plants have become less distinct from one another due to the introduction of invasive species.

Ultimately, the authors found alien plants are more likely to become naturalized in a distant environment when the climate, and especially the temperature, is similar to their last home.

Rainfall, on the other hand, didn't seem to influence plant uniformity nearly as much. This suggests many invasive plants are weeds, thriving on agricultural lands and along rivers.

"The more similar two regions are in terms of climate, the more likely it is that a plant from one region will succeed in establishing itself as a naturalized species in the other region, once geographic barriers have been crossed," explains ecologist Qiang Yang, also from the University of Konstanz.

"In a sense, plants from a region with short climatic distance to their new habitat are climatically pre-adapted."

Those regions of the world that share the same current or past political administrations also have relatively uniform flora.

This is likely because human trade and transport are much more common between states in a nation, nations in a union, or historic colonial networks.

At one point, for instance, the British global empire had set up 126 botanical gardens around the world, all of which exchanged plant species.

Similarly, European colonizers brought many alien species to Australia, which is probably why this region of the world is such a hotspot for homogenization.

Today, invasive alien plants in Australia number in the thousands, and each year about 20 new species are added to the list, displacing even more native plants and altering natural habitats.

The ecological, evolutionary, and socioeconomic consequences of all this change remain unclear. Still, given how important biodiversity appears for local ecosystems, the arrival of a 'New Pangaea' could be very destructive.

Previous studies suggest the last time a supercontinent existed on Earth, it increased the cosmopolitanism of global fauna and led to mass extinctions, causing homogenous 'disaster faunas' to take over. There's no reason why it couldn't happen again.

The current analysis is a rough estimate of how much homogeneity has already occurred among flowering plants, but far more research is needed to determine how uniform the entire biosphere has become and why.

Only then will we know what needs to be done to save it.

The study was published in *Nature Communications*.

https://bit.ly/3eajTAU

When humans are gone, what animals might evolve to have our smarts and skills?

Is this a "Planet of the Apes" situation?

By Joanna Thompson

Humans are pretty unique among life on Earth. As far as we know, we're the only living species to evolve a higher intelligence, wear clothes, cook our food, invent smartphones and then get locked out of them when we forget our passwords.

But what if humans suddenly went extinct? What other animals might evolve to have the smarts and skills to create large, complex societies like we have?

With modern gene-sequencing technology and our understanding of evolution, "we're pretty good at making short term predictions," Martha Reiskind, a molecular ecologist at North Carolina State University, told Live Science. For example, we can predict that if humans were to suddenly go extinct tomorrow, climate change would continue to drive many species toward drought resiliency in order to survive. Cold-specialized species will continue to struggle as well, meaning that, sadly, polar bears and penguins are unlikely to thrive in the millennia after humans are gone.

"A big thing will be the concept of convergence," Dougal Dixon, a geologist, science writer and author of the speculative book "After Man: A Zoology of the Future" (St. Martin's Press, 1998), told Live Science. Convergence is an evolutionary process by which two unrelated organisms end up developing similar traits in order to succeed in a particular environment or fill a particular niche.

The classic example, Dixon said, is the fish shape. With their sleek, torpedo-like bodies and stabilizing fins, fish are optimized for life in water. However, dolphins have evolved a very similar body plan — and unlike fish, they are warm-blooded, air-breathing mammals with a totally different evolutionary background.

One feature that makes humans uniquely good at building and spatial reasoning is our dexterous hands, according to research from the University of Manchester. In order to fill the same ecological role as humans — that is, building cities and heavily modifying our environment — another species would need to develop a similar capacity to manipulate objects. In other words, they would need opposable thumbs — or at least thumb equivalents.

Other primates, like chimpanzees (*Pan troglodytes*) and bonobos (*Pan paniscus*), our closest living relatives, already have opposable thumbs that they use to make tools in the wild. It's possible that if humans go extinct, these hominids might replace us hominins, à la "Planet of the Apes." There is precedent for that kind of overlap — after all, our species managed to outlast the intelligent Neanderthals during the most recent ice age 40,000 years ago, according to a 2021 study published in the journal Nature. That said, it would probably take hundreds of thousands or even millions of years of evolution for other apes to develop the ability to create and use sophisticated, human-like tools. To add context to this scenario, the common ancestor of modern humans and chimpanzees lived about 7 million years ago, Live Science previously reported.

But any disaster potent enough to wipe out humans is also likely to wipe out chimps, which leaves another tool-using candidate to fill humans' niche: birds.

When non-avian dinosaurs went extinct 66 million years ago, mammals rose to fill many of their vacant niches. If humans were to disappear, it's possible that birds, the only surviving dinosaurs, could fill our roles as the smartest and handiest land animals. Despite stereotypes to the contrary, birds are very brainy: Some birds, such as crows and ravens, have intellects that rival even chimps, according to research published in 2020 in the journal Science. And some birds can use their dexterous feet and beaks to fashion wire into hooks, according to a famous 2002 study

published in Science. Meanwhile, trained African grey parrots (*Psittacus erithacus*) can learn upward of 100 words and do simple math, including understanding the concept of zero, Live Science previously reported.

Birds can flock together in large groups, and some, such as sociable weavers (*Philetairus socius*), even build communal nesting sites. Some sociable weaver nests remain occupied by birds for decades, according to research published in the journal Frontiers in Ecology and Evolution. However, these arboreal dwellings wouldn't look much like human metropolises.

But there is another group of animals that is extremely adept at manipulating objects with their limbs — all eight of them.

"Intelligence is modifying your behavior as a result of influence from your environment," Jennifer Mather, a cephalopod intelligence researcher at the University of Lethbridge in Alberta, Canada, told Live Science. By that measure, octopuses are probably the smartest non-human animals on Earth. They can learn to distinguish between real and virtual objects, according to 2020 research published in The Biological Bulletin, and they can even engineer their environment by removing unwanted algae from their dens and barricading the entrance with shells, according to a study in the journal Communicative and Integrative Biology. They're even known to live in communities, of sorts, as shown by the discovery of "Octlantis" off Australia.

However, octopuses would be hard-pressed to adapt to life on land. Vertebrates have iron in their blood cells, which binds to oxygen very efficiently. In contrast, octopuses and their relatives have copper-based blood cells. These molecules still bind to oxygen, but less readily, and as a result octopuses are confined to oxygen-saturated waters as opposed to thin air. "They've taken an inefficient metabolism as far as they can go," Mather said.

Because of this, Mather thinks that octopuses and other cephalopods are unlikely to make the transition to land and take over humanity's mantle as the smartest and most ecologically impactful land animal. Her money is on social insects, like ants and termites. "I think that the insects are tougher than us," Mather said. "Unfortunately, they're tougher than cephalopods as well."

Here's why: Insects are incredibly adaptable to different types of environments. They have been around for 480 million years, according to the Natural History Museum in London. In that time, they've evolved to fill almost every niche imaginable, from flying to burrowing to swimming and even building elaborate city-like towers. The organization of ant and termite colonies probably resembles human civilization more than any other non-human species on Earth. Ants are known to farm fungi, according to research published in 2017 the journal Proceedings of the Royal Society B, and termites can communicate over long distances inside their colonies using vibrations, according to a 2021 study in the journal Scientific Reports. If humans go extinct, it's possible that these insect colonies might take over the world — assuming they survive climate change.

Of course, all of this is speculation; it's virtually impossible to truly predict how evolution will unfold on a geologic time scale. "As you go further and further out, your precision is less clear, because there's all these other wonderful things that cause variation," Reiskind said. Those factors include random mutations, sudden extinction events and population bottlenecks, in which a species pulls itself back from the brink of extinction but loses much of its genetic diversity. And it's even more difficult to predict whether another species will develop human-level intelligence or the desire to build cities. Mather thinks that it could happen, but not without millions of years of the right selective pressure. Dixon, however, is less optimistic. "I don't think nature will make that mistake twice," he said.

https://bit.ly/3FiN5Sf

Winter Is Coming Paradox: Researchers Uncover the Surprising Cause of the Little Ice Age

Cold era, lasting from early 15th to mid-19th centuries, triggered by unusually warm conditions.

New research from the University of Massachusetts Amherst provides a novel answer to one of the persistent questions in historical climatology, environmental history, and the earth sciences: what caused the Little Ice Age? The answer, we now know, is a paradox: warming.

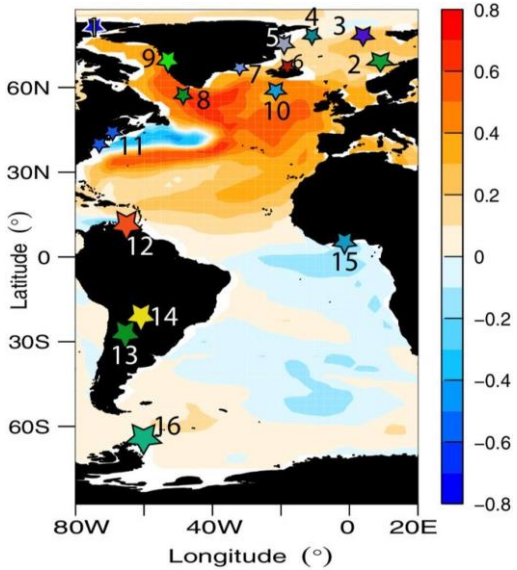
The Little Ice Age was one of the coldest periods of the past 10,000 years, a period of cooling that was particularly pronounced in the North Atlantic region. This cold spell, whose precise timeline scholars debate, but which seems to have set in around 600 years ago, was responsible for crop failures, famines, and pandemics throughout Europe, resulting in misery and death for millions. To date, the mechanisms that led to this harsh climate state have remained inconclusive. However, a new paper published recently in *Science Advances* gives an up-to-date picture of the events that brought about the Little Ice Age. Surprisingly, the cooling appears to have been triggered by an unusually warm episode.

When lead author Francois Lapointe, postdoctoral researcher and lecturer in geosciences at UMass Amherst and Raymond Bradley, distinguished professor in geosciences at UMass Amherst began carefully examining their 3,000-year reconstruction of North Atlantic sea surface temperatures, results of which were published in the *Proceedings of the National Academy of Sciences* in 2020, they noticed something surprising: a sudden change from very warm conditions in the late 1300s to unprecedented cold conditions in the early 1400s, only 20 years later.

Using many detailed marine records, Lapointe and Bradley discovered that there was an abnormally strong northward transfer of warm water in the late 1300s which peaked around 1380. As a result, the waters south of Greenland and the Nordic Seas became much warmer than usual. “No one has recognized this before,” notes Lapointe.

Normally, there is always a transfer of warm water from the tropics to the arctic. It’s a well-known process called the Atlantic Meridional Overturning Circulation (AMOC), which is like a planetary conveyor belt. Typically, warm water from the tropics flows north along the coast of Northern Europe, and when it reaches higher latitudes and meets colder arctic waters, it loses heat and becomes denser, causing the water to sink at the bottom of the ocean. This deep-water formation then flows south along the coast of North America and continues on to circulate around the world.

But in the late 1300s, AMOC strengthened significantly, which meant that far more warm water than usual was moving north, which in turn cause rapid Arctic ice loss. Over the course of a few decades in the late 1300s and 1400s, vast amounts of ice were flushed out into the North Atlantic, which not only cooled the North Atlantic waters, but also diluted their saltiness, ultimately causing AMOC to collapse. It is this collapse that then triggered a substantial cooling.



Multimodel mean correlation map between the low-frequency AMOC at 26°N and SST (12). Stars numbered 1 to 15 denote location of sites. **Credit: Image from Lapointe et. al., https://doi.org/10.1126/sciadv.abi8230**

Fast-forward to our own time: between the 1960s and 1980s, we

have also seen a rapid strengthening of AMOC, which has been linked with persistently high pressure in the atmosphere over Greenland. Lapointe and Bradley think the same atmospheric situation occurred just prior to the Little Ice Age—but what could have set off that persistent high-pressure event in the 1380s?

The answer, Lapointe discovered, is to be found in trees. Once the researchers compared their findings to a new record of solar activity revealed by radiocarbon isotopes preserved in tree rings, they discovered that unusually high solar activity was recorded in the late 1300s. Such solar activity tends to lead to high atmospheric pressure over Greenland.

At the same time, fewer volcanic eruptions were happening on earth, which means that there was less ash in the air. A "cleaner" atmosphere meant that the planet was more responsive to changes in solar output. "Hence the effect of high solar activity on the atmospheric circulation in the North-Atlantic was particularly strong," said Lapointe.

Lapointe and Bradley have been wondering whether such an abrupt cooling event could happen again in our age of global climate change. They note that there is now much less arctic sea ice due to global warming, so an event like that in the early 1400s, involving sea ice transport, is unlikely. "However, we do have to keep an eye on the build-up of freshwater in the Beaufort Sea (north of Alaska) which has increased by 40% in the past two decades. Its export to the subpolar North Atlantic could have a strong impact on oceanic circulation," said Lapointe. "Also, persistent periods of high pressure over Greenland in summer have been much more frequent over the past decade and are linked with record-breaking ice melt. Climate models do not capture these events reliably and so we may be underestimating future ice loss from the ice sheet, with more freshwater entering the North Atlantic, potentially leading to a weakening or collapse of the AMOC." The authors conclude that there is an urgent need to address these uncertainties.

Reference: "Little Ice Age abruptly triggered by intrusion of Atlantic waters into the Nordic Seas" by Francois Lapointe and Raymond S. Bradley, 15 December 2021, Science Advances. DOI: 10.1126/sciadv.abi8230

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https://bit.ly/325cFMo

Neanderthals Changed Ecosystems 125,000 Years Ago – Were Not "Primal Hippies"

Hunter-gatherers caused ecosystems to change 125,000 years ago.

By Tim Senden, Leiden University

These are the findings of an interdisciplinary study by archaeologists from Leiden University in collaboration with other researchers. Neanderthals used fire to keep the landscape open and thus had a big impact on their local environment. The study was published in the journal *Science Advances* on December 15, 2021.

"Among other things, we found the remains of hundreds of slaughtered animals, surrounded by numerous stone tools and a huge amount of charcoal remains." — Wil Roebroeks

"Archaeologists have long been asking questions about the character and temporal depth of human intervention in our planet's ecosystems. We are increasingly seeing very early, generally weak signs of this," says Wil Roebroeks, Archaeology professor at Leiden University.

Excavation of a 125,000-year-old archaeological site at Neumark-Nord 2 near Halle, Germany, summer 2007. **Credit: Leiden University**

These signs proved much stronger in research at a lignite quarry near Halle in Germany. Archaeological research has been carried out at this quarry, Neumark-Nord, in the last few decades, and alongside a huge amount of data about the early environment, abundant traces of Neanderthal activities have been found. "Among

other things, we found the remains of hundreds of slaughtered animals, surrounded by numerous stone tools and a huge amount of charcoal remains."

Open for 2,000 years

The traces were found in what 125,000 years ago was a forest area where not only prey such as horses, deer, and cattle, but also elephants, lions, and hyenas lived. This mixed deciduous forest stretched from the Netherlands to Poland. In several places in the area were lakes, and on the edges of some of these, traces of Neanderthals have been found, Roebroeks explains. At the point in time when these Neanderthals turned up there, the closed forest made way for large open spaces, in part due to fires.

Flint artifacts excavated in the shore area of the small lake. **Credit: Leiden University**

"The question is, of course, whether it became open because of the arrival of hominins, or whether hominins came because it was open? However, we have found sufficient evidence to conclude that hunter-gatherers kept the area open for at least 2,000 years." Comparative research conducted by Leiden palaeobotanist Professor Corrie Bakels has shown that at similar lakes in the area, where the same animals roamed, but where there are no traces of Neanderthals, the dense forest vegetation remained largely intact.

"Hunter-gatherers weren't simply "primal hippies" who roamed the landscape picking fruit here and hunting animals there." — Wil Roebroeks

Until now it was generally thought that it was only when humans took up agriculture about 10,000 years ago that they began to shape their environment, for instance by cutting down trees to create fields. But many archaeologists believe it started much sooner, on a smaller scale, and according to Roebroeks, Neumark-Nord is the earliest example of such intervention. The new research findings are not only important for archaeology, says Roebroeks, but also for disciplines involved in nature restoration, for instance. "It also adds something to the behavioral spectrum of early hunter-gatherers. They weren't simply "primal hippies" who roamed the landscape picking fruit here and hunting animals there. They helped shape their landscape."

Oospores of stoneworts (algae), roughly 1 mm in size, and charred seeds. **Credit: Leiden University**

Major impact of fire

A previous study by Roebroeks and his research team showed that knowledge about fire was already being passed down by hominins at least 400,000 years ago. "We shouldn't be surprised if in future research we find traces that indicate that hominins had a major impact on their environment much earlier, on a local scale at least."

Reference: "Landscape modification by Last Interglacial Neanderthals" by Wil Roebroeks, Katharine MacDonald, Fulco Scherjon, Corrie Bakels, Lutz Kindler, Anastasia Nikulina, Eduard Pop and Sabine Gaudzinski-Windheuser, 15 December 2021, Science Advances. *DOI: 10.1126/sciadv.abj5567*