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Study Suggests We Have This STI to Thank For The Evolution of Grandmothers

The arms race between the human immune system and gonorrhea might have had the useful side effect of promoting healthy brain tissue later in life.

Felicity Nelson

This tiny boost to cognitive health in our twilight years might have played a small role in ensuring grandmas were sharp-minded enough for evolution to keep them around.

While it's fiendishly difficult – and may be impossible – to figure out what evolutionary factors are responsible for living beyond ages where we no longer reproduce, researchers at the University of California, San Diego, are closing in on some possible explanations. In [2015](#), a team of researchers led by molecular medicine professor Ajit Varki discovered that humans have a unique type of immune receptor that protects against [Alzheimer's](#) disease and sets us apart from other primates.

In a [paper](#) published this month, the team found that the spreading of this variant immune receptor in our species wasn't entirely random, but rather the result of intense selection pressure over a relatively brief period.

The research showed that some of our closest relatives – [Neanderthals](#) and [Denisovans](#) – did not have this version of immune receptors coded into their genomes.

Something drove humans to develop this special immune receptor early in our history as a species, the researchers said.

The likely culprits are infectious human-specific pathogens like *Neisseria gonorrhoeae* that try to disguise themselves by dressing in the same sugar coating as human cells, which fools patrolling immune cells into thinking the bacteria are harmless.

[Gonorrhea](#) got very good at tricking the human immune system into

thinking it was just another human cell. But the human immune system found a way to fight back.

The researchers showed that the newly evolved immune receptor could see through the disguise and kill the invading bacteria, while the older variation of the immune receptor could not.

Getting rid of gonorrhea is useful for the survival of the species because this disease can mess with human reproduction.

The new version of the immune receptor is called huCD33. Thanks to the way this version is tweaked into two subtly different structures within our body, it's been the subject of investigations by evolutionary scientists for some time.

Once evolved, this immune receptor was probably co-opted by brain immune cells, called microglia, for a different purpose: protection against aging, the researchers suggest.

The human immune system usually doesn't attack itself on purpose, but it needs to when cells start to decay.

The huCD33 receptor, which seems to have evolved as a response to sneaky bacteria, had the added benefit of being able to recognize decaying brain tissue and thereby protect cognitive function in old age.

Microglia use the huCD33 receptor to clear away damaged brain cells and amyloid plaques associated with Alzheimer's disease.

Whether this might have played a role in clearing the way for evolution to add a few more precious years to our lives for the sake of helping out with raising families is a topic open to debate.

Grandparents provide benefits to the human species as they help to look after kids and pass on important cultural knowledge.

And gonorrhea may be to thank for that.

This paper was published in [Molecular Biology and Evolution](#).

<https://bit.ly/3QIU427>

Incredible Hubble Image Reveals a Bizarre Galaxy 'Mirror'

There's something very odd about this image from the Hubble Space Telescope. If you look closely, you can see two almost-mirror image, orange-colored galaxies, seemingly connected by a long filament.

[Michelle Starr](#)

Fascinatingly, that's not two galaxies at all, but one, named SGAS J143845+145407. It just

appears to be two, thanks to the way the gravity of a massive object (or objects, like a cluster of galaxies) distorts the space that distant light travels through.



[Gravitational lensed mirror images of SGAS J143845+145407. \(ESA/Hubble & NASA, J. Rigby\)](#)

Imagine putting a heavy weight on a trampoline, where the weight represents the galaxy cluster, and the trampoline mat represents space-time.

Now roll some marbles from one side of the trampoline to the other. Their normally 'straight' paths will seem to curve along different paths, not unlike rays of light through distorted space.

Called gravitational lensing, this quirk of gravity can be used to magnify the light of background galaxies that would be too distant to otherwise see in much detail, as illustrated in the diagram below.

Gravitational lenses like this can therefore be an important tool for understanding the distant Universe.

Sometimes that light can become really smeared and distorted, as seen in the recent [deep field image from the James Webb Space Telescope](#). Those odd, wobbly, worm-like objects are lensed

galaxies. When the lensing effect results in four images of a distant object arrayed around the central lensing mass, [that's called an Einstein Cross](#).

SGAS J143845+145407 appears at just the right point behind a small galaxy cluster for gravitational lensing to produce two nearly perfect images of the galaxy, with the added bonus of making them both appear bigger and in greater detail.

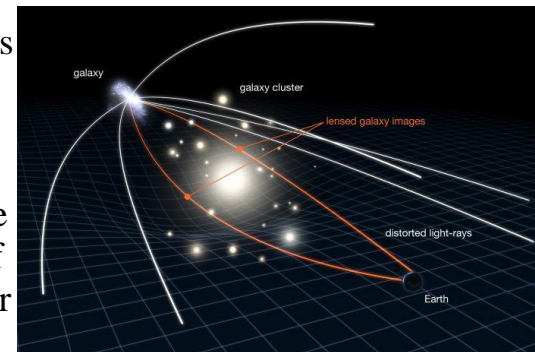


Illustration of gravitational lensing. (NASA, ESA & L. Calçada)

The light from SGAS J143845+145407 traveled around 6.9 billion years to reach us. That's about half the current age of the Universe. The cluster's light traveled about 2.8 billion years.

SGAS J143845+145407 is scientifically interesting because it's a luminous infrared galaxy, glowing relatively brightly due to high star formation activity.

Studying galaxies like it can help scientists understand star formation and how it has changed throughout the Universe's history; for this kind of work, gravitational lenses can be invaluable. Using the gravitational lens, scientists were recently able to [reconstruct the distribution of star formation in SGAS J143845+145407](#), and study the details of the process.

They found that the galaxy is pretty typical of its type, which information will be able to help contextualize and characterize other galaxies.

Webb is expected to reveal even more details, but Hubble revolutionized the study of lensed galaxies. Its observations were the first to resolve details inside lensed galaxies, giving scientists an incredible new window into the early Universe.

The image has been published on the [Hubble website](#).

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Explosive Report Claims a Leading Alzheimer's Theory May Use Fabricated Results

A seminal 2006 study of Alzheimer's disease might contain fabricated results, [an investigation from Science magazine found.](#)

Marianne Guenot, Business Insider

A seminal 2006 study of Alzheimer's disease might contain fabricated results, [an investigation from Science magazine found.](#)

The investigation uncovered evidence suggesting several instances of image manipulation in the work of Sylvain Lesné, a researcher working at the University of Minnesota and an author of the 2006

The paper, [which is cited by more than 2,200 academic papers as a reference](#), launched interest in a specific protein called A β *56 as a promising target for early intervention in Alzheimer's disease.

A β *56 is a beta-amyloid. Beta amyloids are proteins that have [been observed to clump in the brain](#), a phenomenon that is widely believed to be linked to the development of Alzheimer's disease. Several different types of these proteins are potential targets for drugs treating Alzheimer's.

Whistleblower Matthew Schrag, a neuroscientist at Vanderbilt University, first flagged his concerns about the images to the NIH on January 2022. *Science* asked two image analysis experts to review Lesné's published work. They echoed Schrag's concerns.

They identified a total of 20 "suspect papers" authored by Lesné, 10 of which had to do with A β *56, per *Science*.

The publication stopped short of alleging misconduct or fraud, stating that the original images would have to be investigated for manipulation to be confirmed. The most "obvious" effect of this alleged manipulation would be "wasted NIH funding and wasted thinking in the field," Nobel laureate and Stanford University neuroscientist Thomas Südhof told *Science*.

[Several unnamed researchers told the Alzforum](#), an Alzheimer's-

focused outlet, that they tried to reproduce the results but could not. Work like this is often not reported widely, as it is difficult to publish results that invalidate previous work in academic journals.

"Even if misconduct is rare, false ideas inserted into key nodes in our body of scientific knowledge can warp our understanding," Schrag told *Science*.

Nature, the academic journal that published the 2006 paper, is investigating the allegations about the paper, [according to a publisher's note](#). This is the latest blow to the field of beta-amyloid research in Alzheimer's, which has come under scrutiny recently after [scientists raised concerns about the evidence base](#) supporting the idea that [FDA-approved drug Aducanumab](#) can improve cognition in people with Alzheimer's.

Though the allegations into Lesné's work are concerning, they do not compromise the field of research into amyloid proteins and Alzheimer's disease, Alzheimer's Research UK and the Alzheimer's Society said in statements seen by Insider.

"Despite these allegations, we should not allow the work of thousands of Alzheimer's researchers to be undermined – their painstaking efforts are bringing us closer to vital new treatments for the millions of people living with the disease," Sara Imarisio, Head of Research at Alzheimer's Research UK said in a statement seen by Insider. "There are legitimate questions and criticisms of the amyloid hypothesis, but such questions are a perfectly normal and necessary part of science," she said.

A co-author of Lesné's papers, Karen Hsiao Ashe, stands by the role of A β *56 in Alzheimer's, stating that staff scientists in her lab "regularly and reproducibly detect A β *56" in lab mice, [she wrote in a comment on Alzforum's article](#).

Science could not find evidence of image manipulation in Ashe's work that is not co-authored with Lesné. Lesné could not be contacted by *Science* when they reached out for comment.

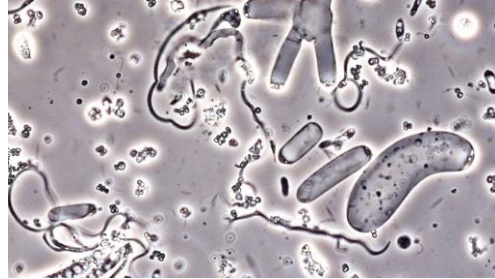
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The Creature That Gave Up Parasitism for ... Wait, What?

It's a mystery wrapped in a riddle wrapped in a kidney.

By [Ed Yong](#)

In the late 19th century, when scientists first discovered the single-celled creature called *Nephromyces*, they thought it was a parasitic fungus. They were wrong.



Mary Beth Saffo, Ph.D / PNAS

Instead, it's ... well ... how to even describe it? It's a reformed parasite. It's a creature of extremes, surviving in a world of acids and dining on, of all things, kidney stones. And perhaps strangest of all, it's an organism that cannot survive as an individual. A single ant will do badly away from its colony; a single *Nephromyces* wouldn't even get that far.

Nephromyces can be found all along the eastern coast of the U.S., living inside the bodies of translucent, blobby animals called sea squirts. Some sea squirts look like beautiful glass vases; those that house *Nephromyces*, known as sea grapes, look more like cysts that have grown bunny ears.

Inside each sea grape is a large organ called a renal sac, so named because scientists originally thought it was a kidney. But as the biologist Mary Beth Saffo [once told *The New York Times*](#), "If it was a kidney, it was a pretty odd one." For a start, it has no opening. It accumulates the chemicals you'd expect in a kidney—uric and oxalic acids, crystallized into what are essentially kidney stones—but instead of excreting these as a normal kidney would, it stores them for the sea grape's entire life. As a result, the sac is extremely acidic. Despite that, it's *teeming* with life. Cut into one, and "cells

just pour out," Saffo told me.

Scientists first noticed these cells in the 1870s. They came in a variety of shapes—blobs and threads, barrels and baskets. Researchers deduced that these were all different life stages of the same organism, which was named *Nephromyces* after the Greek for "kidney fungus." But [as Saffo showed in 2010](#), after sequencing its DNA, *Nephromyces* isn't a fungus at all. It's part of a group of nefarious single-celled organisms called the "apicomplexans," whose members cause diseases such as malaria and toxoplasmosis. There are about 6,000 species of apicomplexans, and they're almost all parasites.

Almost all. If *Nephromyces* is a parasite, it's a pretty odd one. It doesn't seem to harm its sea-grape host in any way. And though sea grapes aren't born with *Nephromyces*, they *always* acquire these cells from the surrounding seawater. Every adult sea grape that's ever been examined harbors hordes of *Nephromyces* in its renal sac—a 100 percent infection rate that actual parasites almost never achieve, because their hosts tend to fight back. The sea grape doesn't, which suggests that *Nephromyces*'s presence is benign, if not beneficial. It just sits there in the renal sac, minding its own business, eating kidney stones. It's a black sheep in a family of vampires—a creature that has abandoned its relatives' penchant for exploitation.

Such transitions are incredibly rare. Parasites usually become so dependent on their hosts that they lose genes, traits, and body parts that they need for a free-living life. That's why [many organisms take up parasitism](#) but very few ever give it up. How did *Nephromyces* manage?

One possibility: It has help. In 1990, Saffo showed that *Nephromyces* [is chronically infected with bacteria](#)—microbes that live inside it, just as it lives inside the sea grape. Many creatures [are home to internal bacteria](#), which might provide them with nutrients

that are missing from their diet, or help them to digest food they otherwise could not. Perhaps, then, the bacteria in *Nephromyces* perform similar services. Do they help it to digest the kidney stones around it? Do they provide it with nutrients that its parasitic ancestors lost the ability to make?

Like others before him, Chris Lane, from the University of Rhode Island, assumed that sea grapes contain just one species of *Nephromyces*, which contains just one species of bacteria, perhaps a pretty odd one. [But when he looked closer](#), he found that they're pretty odd *ones*. By sequencing the DNA inside the renal sacs, his students found evidence of a much larger community—many kinds of bacteria, and dozens of *Nephromyces* species.

The team showed that the bacteria in the community belong to three major groups, which Lane thinks of as “flavors.” Each species of *Nephromyces* contains one and only one of these flavors (perhaps because there simply isn't space for more). The bacteria are crucial; they help *Nephromyces* produce essential vitamins and amino acids that it otherwise couldn't get. But here's the catch: No single flavor of bacteria can provide *all* of the nutrients that *Nephromyces* requires.

It needs all three flavors together—but no species can ever have more than one. For that reason, no *Nephromyces* species can thrive on its own. They *must* exist as a community—a multispecies swarm that survives by trading the nutrients that their respective bacteria provide. Lane describes it as a “hippie commune.” In the wild, he showed, sea grapes tend to harbor somewhere between three and eight *Nephromyces* species.

Is this what it takes for a parasite to give up parasitism? Because, to be honest, “it's a bit of a mess,” Lane told me. Remember that each sea grape picks up *Nephromyces* from the surrounding seawater. And *Nephromyces* does well only if different species with the right bacterial flavors somehow manage to co-infect a single sea grape.

“This is just a dumb situation,” Lane said. “It seems like it shouldn't exist, but here we are.”

“The first time you see something like this, you say to yourself: *Go home, evolution, you're drunk*,” John McCutcheon, an evolutionary biologist at Arizona State University, told me. “But evolution doesn't go out looking for simple solutions to things: It cobbles together solutions from what's there.” The results can be bafflingly complicated.

McCutcheon and his colleagues [have studied an insect](#) with bacteria living inside its cells and bacteria living inside those—bugs in bugs in bugs. They found an entire dynasty of bacteria that are splitting into new species [inside the bodies of cicadas](#). “Now we all know to look for crazy combinations,” he said.

With *Nephromyces*, many mysteries still remain. Are the blobs, barrels, and other shapes actually different life stages, as scientists once thought, or different species, as Lane now suspects? How do the different *Nephromyces* species ensure that enough of them get inside the same sea grape? How did the different species even evolve? (Many organisms diversify into a variety of species when each gets to exploit a different corner of the environment—but where are the niches in a sea grape's renal sac?) Are there *Nephromyces* species that cheat—that contain no bacteria of their own and instead mooch off the nutrients produced by the rest of the commune? (“We think so,” Lane told me.) Also, how does the sea grape benefit from the community inside its body, if at all? Lab-raised sea grapes that lack *Nephromyces* “do just fine,” Lane said. And though many animals depend on internal microbes for nutrients, sea grapes “live in almost laughably nutrient-rich ocean waters,” Saffo told me.

“We're not even close to getting to the bottom of this,” she said. “The details get filled in and get even more confusing.”

<https://bit.ly/3oFEpib>

New Zealand's 'tobacco endgame' law will be a world first for health – here's what the modelling shows us

With the first reading of a new bill in parliament today, Aotearoa New Zealand's plan to be smokefree by 2025 takes another tangible step forward.

[Tony Blakely](#)¹ [Andrew Waa](#)² [Driss Ait Ouakrim](#)³

The [Smokefree Environments and Regulated Products \(Smoked Tobacco\) Amendment Bill](#) will now go to the Health Select Committee for submissions and review, and (presumably) return to the House in late 2022 to be passed into law.

Assuming the final legislation looks similar to what is being proposed, it will mean Aotearoa New Zealand leapfrogs all other countries to be at the vanguard of tobacco control, with policy settings aimed at getting smoking prevalence beneath 5% of the adult population within years (not decades).

The bill provides for three key strategies:

- drastically reducing nicotine content in tobacco so it is no longer addictive (known as “denicotinisation” or “very low nicotine cigarettes” (VLNC))
- a 90% to 95% reduction in the number of shops that can sell tobacco
- making it illegal to sell tobacco to people born in 2009 or later (thus creating a “smokefree generation”).

If implemented effectively this is anticipated to have a profound impact on smoking.

Reducing Māori health inequity

If successful, this would be a monumental achievement for generations of tobacco-control advocates and researchers. The concept of a “tobacco endgame” will move beyond aspiration and into reality.

We've got to this point after decades of Māori leadership, research

and advocacy, with the proposed legislation having its roots in the aim of reducing health inequities between Māori and non-Māori. This kaupapa (principle or policy) has driven the process and is supported by Māori communities.

Much more will be written in coming months about this groundbreaking legislation.

Here we focus on the [modelling we were commissioned to provide](#) by the New Zealand government in 2021-2022 on the potential health and cost impacts of the [Smokefree Aotearoa 2025 Action Plan](#).

Projected effects of the combined endgame interventions on smoking prevalence to be introduced in 2023. Likely delays in implementation will shift the curves to the right commensurately. Author provided

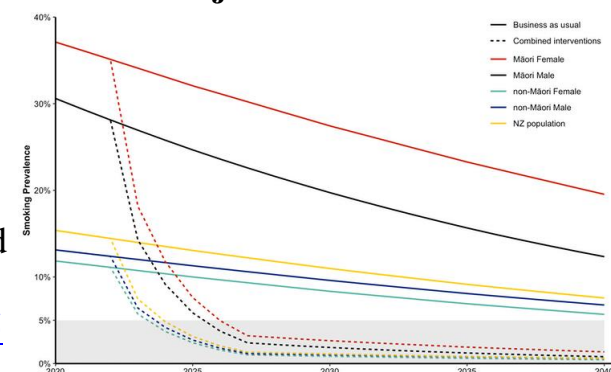
Our findings underpinned the [regulatory impact statement](#) that set out the options to regulate tobacco products as part of the action plan, which Cabinet considered in early 2022.

Large reductions in mortality rates

In our work at Otago University's [BODE3 program](#) and the University of Melbourne's Scalable Health Intervention Evaluation ([SHINE](#)) we model many potential public health interventions, from dietary counselling and reducing salt in bread to the evaluation of screening programmes and drug treatments.

We tally the likely health gains from these interventions, and how much they might reduce inequities in health. When we do this for the government's tobacco endgame strategy, the forecasts are breathtaking.

Projected declines in smoking



Consider reductions in health inequities between Māori and non-Māori. First, we forecasted what Māori and non-Māori mortality rates will be in 2040 (and beyond) given trends we have seen in recent decades (business as usual in the graph above).

Second, we estimated how much smoking (and vaping) rates would change into the future for the combined endgame policy (denicotinisation, retail reduction, smokefree generation regulations, augmented by some media promotion of the policy).

Third, allowing for time lags, we modelled future disease rates (for example, lung and heart disease) and then the overall impact on mortality rates.

We then compared the gap between Māori and non-Māori mortality or death rates in 2040 if there were no major policy changes, and under the combined tobacco endgame strategy. For those aged 45 and over, the gap was reduced by a staggering 22.9% for Māori females compared to non-Māori females, and a still very large 9.6% for males.

Longer, healthier lives

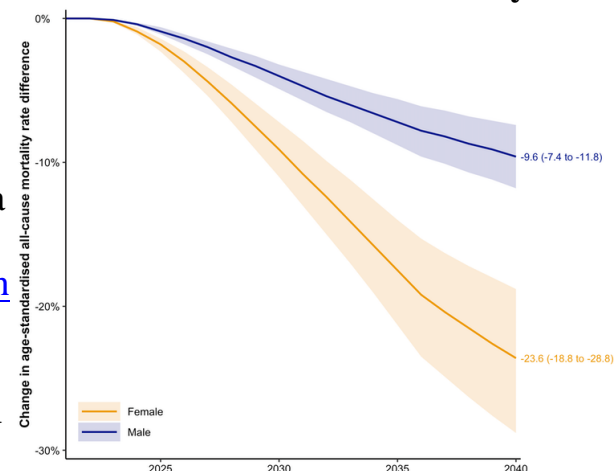
In all our previous research, we have never seen a single health intervention with the potential to reduce health inequities this much. Why is a tobacco endgame so powerful at reducing Māori and non-Māori health inequities? Because smoking is so bad for health, smoking rates are particularly high among Māori, and Māori also have higher smoking-related disease rates.

Therefore, Māori see more health gains from the dramatic falls in tobacco smoking that will result from the policy. (Non-Māori also see large gains – just not as much per capita as Māori.)

What about overall health gains? Our modelling suggests that, over the remaining lifespan of the New Zealand population alive in 2020, the tobacco endgame strategy will result in an extra 600,000 “health-adjusted life years” lived (a measure of the impact of those interventions on life expectancy, adjusted for quality of life).

Projected decline in gap between Māori and non-Māori mortality rates

To put this in perspective, this amount of health gain – accruing just to those people quitting smoking earlier or not taking it up, a minority of the population – is equivalent to the [health gains that would result](#) from a policy taxing sugar, fat and salt in all foods and removing the GST on healthy food.



Projected effects of the combined endgame interventions on the percentage change in the mortality rate difference between Māori and non-Māori aged 45 and up. Author provided

Major health system savings

Not only is this endgame policy increasing the health of the nation, it is also reducing future health expenditure.

We estimated NZ\$1.3 billion of health system costs would be avoided in the next 20 years. These savings can be used for other things, such as mental health and dementia care.

And while the government will lose tax revenue from drastically reduced tobacco sales, the overall health of the population increases, meaning more people are in work for longer. We estimated an income gain to the New Zealand population of \$1.4 billion in the next 20 years, which means more tax revenue as well.

All modelling of the future is uncertain. But even allowing for that uncertainty, the health gains, the health inequity reductions, the savings in health expenditure, and the increased income productivity of New Zealanders that will result from this tobacco

endgame strategy will be large.

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New hypothesis emerges to explain mysterious hepatitis cases in kids

Two viruses and a genetic predisposition linked to the puzzling condition in preliminary data.

[Beth Mole](#)

Researchers in the United Kingdom have come up with the most detailed, complex hypothesis yet to explain the burst of mysterious cases of liver inflammation—aka hepatitis—in young children, which has troubled medical experts worldwide for several months.

The cases first came to light in April, when doctors noted an unusual cluster of hepatitis cases in young children in Scotland. The illnesses were not linked to any known cause of hepatitis, such as hepatitis (A to E) viruses, making them unexplained. Though unexplained cases of pediatric hepatitis arise from time to time, [a report that month](#) noted [13 cases in Scotland](#) in two months when the country would typically see fewer than four in a year.

Since then, the World Health Organization has [tallied more than 1,000 probable cases from 35 countries](#). Of those cases, 46 required

liver transplants, and 22 died. The Centers for Disease Control and Prevention identified [355 cases](#) in the US. As of June 22, [20 US cases required liver transplants, and 11 died](#).

Hypotheses to explain the cases have been wide-ranging. Some have suggested—particularly adamantly—that the cases may be aftereffects of an infection with the pandemic coronavirus, SARS-CoV-2. The CDC, meanwhile, published data that found there hasn't been an increase in pediatric hepatitis cases or liver transplants over pre-pandemic baseline levels, which suggested the unusual clusters may not represent a new phenomenon.

Combination of factors

But a common feature among the cases has been an infection with an adenovirus. The extremely common childhood viruses have shown up in many cases. As such, many hypotheses have involved adenoviruses, but this, too, is puzzling, because adenoviruses are *not* known to cause hepatitis in previously healthy children.

In two new reports, UK researchers offer a fresh hypothesis that may be the clearest but most complex explanation. Their data suggests that the cases may arise from a co-infection of two different viruses—one of which could be an adenovirus and the other a hitchhiking virus—in children who also happen to have a specific genetic predisposition to hepatitis.

In [one of the new studies](#), looking at nine early cases in Scotland, researchers found that all nine children were infected with adeno-associated virus 2 (AAV2). This is a small, non-enveloped DNA virus in the *Dependoparvovirus* genus. It can only replicate in the presence of another virus, often an adenovirus but also some herpesviruses. As such, it tends to travel with adenovirus infections, which spiked in Scotland when the puzzling hepatitis cases arose.

Advertisement

Most striking, while all nine of the hepatitis cluster cases were positive for AAV2, the virus was completely absent in three

separate control groups. It was found in zero of 13 age-matched healthy control children; zero of 12 children who had an adenovirus infection but normal liver function; and zero of 33 children hospitalized with hepatitis for other reasons.

This finding was backed up in a [separate study](#) led by researchers in London, which looked at 26 unexplained hepatitis cases with 136 controls. It also found AAV2 in many of the hepatitis cases but in very few of the control cases.

Predisposition

The study of the nine cases in Scotland went a step further by examining the children's genetics. The researchers noted that eight of the nine children (89 percent) had a gene variant for a human leukocyte antigen called [HLA-DRB1*04:01](#). But this gene variant is only found in about 16 percent of Scottish blood donors, well below the frequency found in the hepatitis cases. Moreover, HLA-DRB1*04:01 is already known to be linked to [autoimmune hepatitis](#) and some [rheumatoid arthritis cases](#).

Generally, human leukocyte antigen (HLA), also known as major histocompatibility complex or (MHC), are proteins outside of immune cells that present antigen—such as viral or bacterial peptides—to T cells. This presentation trains the T cells on how to respond to potential threats, triggering immune responses to invading germs or tolerance to specific antigens. Thus, HLA proteins play a critical role in influencing immune responses.

The Scottish study suggests that all three factors combine to explain the hepatitis cases: An adenovirus infection and a tag-along AAV2 infection, one of which triggers an aberrant immune response in children with a genetic predisposition. It's unclear how all the factors combine exactly, but, based on the nine cases, all three factors are necessary. This could explain why the hepatitis cases are so rare, linked to adenovirus infections, and appeared to cluster after pandemic restrictions were lifted, when many susceptible

children became infected with common viruses, including adenoviruses.

Of course, this is just a hypothesis for now—and one mainly based on only nine cases in a study that has yet to be peer-reviewed. Researchers will have to do far more work to determine if this hypothesis explains the cases, including looking at larger cohorts of children and molecular research to understand the potential mechanism.

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OSIRIS-REx Would Have Sunk Deep into Asteroid Benu if it Tried to Land

Would have sunk into the asteroid Benu had the spacecraft not fired its thrusters immediately after collecting samples

A pair of studies published in [Science](#) and [Science Advances](#) have helped identify that NASA's OSIRIS-REx (Origins, Spectral Interpretation, Resource Identification, Security-Regolith Explorer) spacecraft would have sunk into the asteroid Benu had the spacecraft not fired its thrusters immediately after collecting samples from the surface of the small planetary body in October 2020. The respective studies examined the loosely packed exterior of Benu, comparing its surface to stepping into a pit of plastic balls that people of all ages enjoy. The paper in *Science* was led by Dr. David Lauretta, Principal Investigator of OSIRIS-REx and a Regents Professor at the University of Arizona, and the paper in *Science Advances* was led by Dr. David Walsh, a member of the OSIRIS-REx team from the Southwest Research Institute in Boulder, Colorado.

“If Benu was completely packed, that would imply nearly solid rock, but we found a lot of void space in the surface,” said Walsh.

Launched on September 8, 2016, OSIRIS-REx is the [first U.S. mission](#) to collect a sample from an asteroid. After conducting an Earth flyby a year later, OSIRIS-REx arrived at Benu in December

2018 and successfully collected its sample in October 2020. The spacecraft then began its trip back home in May 2021 and its sample capsule is slated to return to Earth in September 2023.

Before OSIRIS-REx arrived at Bennu, the mission team observed the asteroid using Earth- and space-based telescopes expecting to find a surface that resembled a smooth, sandy beach. Instead, upon the spacecraft's arrival at Bennu in December 2018, the team was surprised to find a surface littered with boulders and [particle plumes](#) erupting from Bennu's surface.

When OSIRIS-REx ultimately collected its samples in October 2020 using its Touch-and-Go Sample Acquisition Mechanism (TAGSAM), the Sample Acquisition Verification Camera (SamCam) of the OSIRIS-REx Camera Suite (OCAMS) photographed stunning images of the sample site looking downward over TAGSAM every 1.2 seconds with an image resolution of approximately 1 mm/pixel. These frame-by-frame images from SamCam show substantial disturbances at the sample site caused by contact from TAGSAM.

"What we saw was a huge wall of debris radiating out from the sample site," Laurretta said. "We were like, 'Holy cow!'" The mission team was even more startled when they saw the 8-meter (26-foot) wide crater that TAGSAM left despite how gently the spacecraft had touched the surface to collect the sample. This result was in stark contrast to lab tests carried out before the mission.

"Every time we tested the sample pickup procedure in the lab, we barely made a divot," Laurretta said. A few months after sample collection, the team sent OSIRIS-REx back to take more images of Bennu's surface "to see how big of a mess we made," Laurretta said. By analyzing the volume of debris in sample site images before and after collection, along with studying acceleration data during spacecraft touch down, the mission scientists were able to deduce that Bennu experienced the same level of resistance that a person

would feel while [pressing the plunger](#) (starting at 1:35 of the video) on a French press coffee carafe. Imagine creating an 8-meter (26-foot) wide hole just by making coffee.

These studies are intriguing since it challenges previous notions about the makeup of asteroids, which could aid in the design of future missions as well as developing methods to protect Earth from asteroid impacts. If asteroids are as loosely packed as Bennu, it's possible they could simply break up in the Earth's atmosphere, posing a different type of hazard than more solid asteroids. Instead of a massive asteroid crashing into Earth, you might have hundreds of smaller pieces literally raining down from the sky and cause damage to a wider area.

In the meantime, the OSIRIS-REx sample capsule is slated to arrive back on Earth in September 2023, and these samples might help us gain a better understanding of the makeup and composition of asteroids within our solar system.

Is Bennu just one of many loosely packed asteroids roaming the solar system, or is it the exception? What will these samples teach us about asteroids and the history of the solar system? Only time will tell, and this is why we science!

<https://bit.ly/3vqxAVm>

Falling Space Junk has a 10% Chance of Killing Someone in the Next Decade

New study claims a 6-10% chance someone will die from debris falling from space over the next ten years

The statistics of how people die offer a gruesome but informative way to understand both how humans perceive threats and how they react to fear. For example, you are more likely to be crushed by a falling vending machine (~13 people killed per year) than be eaten by a shark (~10 per year). However, there is one currently statistically unlikely cause of death that has a real risk of increasing dramatically in likelihood over the coming decades – falling space

debris. According to a new study, there's a 6-10% chance that someone will die from debris falling from space over the next ten years.

This probably isn't surprising to anyone involved in the space industry. The debris problem has been growing for decades at this point, as rockets and satellites leave little pieces of themselves floating uncontrolled around Earth. We here at UT report on incidents involving it consistently (such as [yesterday](#)), though thankfully, we haven't had to report any deaths from it so far.

That might prove that we're just lucky. Some debris has undoubtedly hit unpopulated areas in the near past, and a part of a Long March 5B rocket hit a town in Ivory Coast on the west coast of Africa. Luckily, while there was some building damage, no one was hurt.

It's only a matter of time before someone is, though. Space is getting increasingly crowded, with private companies sending up [thousands of satellites](#) to provide services like broadband internet and near-real-time surface imaging. But at [what cost](#)?

Currently, there is no regulatory requirement on how to dispose of the non-reusable rocket stages that provide the launch capabilities to the myriad companies and nations that want to get to orbit. Some of these components can weigh literal tons and might not entirely break up as they effectively aero brake through the atmosphere.

What's more – one particular part of the world that is more susceptible to these risks – the “global south.” While most of these nations, which reside below the equator, do not have space-faring capabilities of their own, an unfortunate reality of physics makes them more likely to be affected by it. Rockets' paths to get themselves into orbit typically put their unrecoverable bits into a position to fall somewhere below the equator.

In a new paper published in Nature Astronomy, researchers from the University of British Columbia have pointed out all these

disparities and risks. By their calculation, space debris has a 6-10% chance of killing at least one person in the next ten years. Most likely, that person will not be from the nation that created the piece of debris.

That sounds like a recipe for international acrimony, yet no polity has yet come forward to develop a framework for handling the regulation of these potentially hazardous pieces of technology. As the UBC team points out, there are systems and technologies in place that can stop this potential loss of life – we just have to be willing to accept the increased cost.

The most obvious of these would be to require controlled reentry from any rocket fairing. With controlled reentry, the dangerous bits of debris can be landed safely over one of the giant bodies of water that populate our planet. Given SpaceX's success in landing its own booster stages back on a platform, the technology is obviously there to do this. But rocket companies won't implement such a scheme unless required to by regulatory bodies.

Lots of methods have been proposed to clean up space junk. Here's UT's review of them.

Unfortunately, unless something is done soon to curb the likelihood of this event, someone will eventually die from falling space debris. In a worst-case scenario, hundreds could die from a single piece of debris – if it happens to hit an airplane, for example. Hopefully, governments will take a proactive approach to curb that likelihood well before it gets to that point. This new paper points them on the right path, at least.

<https://bit.ly/3zKDWSk>

Dietary Supplement Cuts Risk of Hereditary Cancer by 60%, Scientists Find

Can reduce the risk of some of those cancers by more than 60 percent, simply by adding more resistant starch to their diets

[Fiona MacDonald](#)

A trial spanning more than 20 years and almost 1,000 participants worldwide has found an important result – people with a condition that gives them a higher chance of developing [certain cancers](#) can reduce the risk of some of those cancers by more than 60 percent, simply by adding more resistant starch to their diets.

In fact, the results were so compelling when it came to cutting the risk of upper gastrointestinal (GI) cancers specifically that the researchers are now looking to replicate them to ensure they're not missing anything. Upper GI cancers include esophageal, gastric, and pancreatic cancers.

"We found that resistant starch reduces a range of cancers by over 60 percent. The effect was most obvious in the upper part of the gut," [says lead researcher and nutritionist John Mathers](#) from Newcastle University in the UK.

"The results are exciting, but the magnitude of the protective effect in the upper GI tract was unexpected, so further research is required to replicate these findings," [adds one of the researchers, Tim Bishop](#), a genetic epidemiologist from the University of Leeds.

Resistant starch is a type of starch that passes through the small intestine and then ferments in the large intestine, where it feeds beneficial gut bacteria. It can be bought as a fiber-like supplement, and is naturally in a range of foods, including slightly green bananas, oats, cooked and cooled pasta and rice, peas, and beans.

The double-blind trial was carried out between 1999 and 2005 and involved a group of 918 people with a condition known as [Lynch syndrome](#). Lynch syndrome is one of the most common genetic predispositions to [cancer](#) that we know of, with around one in 300 people estimated to carry an associated gene.

Those who've inherited Lynch syndrome genes have a significantly increased risk of developing colorectal cancer, as well as gastric, endometrial, ovarian, pancreatic, prostate, urinary tract, kidney, bile duct, small bowel, and brain cancers.

To figure out how they could reduce this risk, participants were randomly assigned to one of two groups, with 463 unknowingly given a daily 30 gram dose of resistant starch in powdered form for two years – roughly the equivalent of eating a not-quite-ripe banana daily.

Another 455 people with Lynch syndrome took a daily placebo that looked like powdered starch but didn't contain active ingredients.

The two groups were then followed up 10 years later. The results of this follow-up are what the researchers have just published.

In the [follow-up period](#), there had only been 5 new cases of upper gastrointestinal (GI) cancers among the 463 people who'd taken the resistant starch. This is in comparison with 21 cases of upper GI cancer among the 455 people in the placebo group – a pretty remarkable reduction.

"This is important as cancers of the upper GI tract are difficult to diagnose and often are not caught early on," [says Mathers](#).

However, there was one area where the resistant starch didn't make much difference – in the rate of bowel cancers.

Further work is needed to figure out exactly what's going on here, but the team has some ideas.

"We think that resistant starch may reduce cancer development by changing the bacterial metabolism of bile acids and to reduce those types of bile acids that can damage our DNA and eventually cause cancer," [says Mathers](#).

"However, this needs further research."

To be clear, this trial was carried out on people already genetically predisposed to developing cancer and doesn't necessarily apply to the broader public. But there could be a lot to learn by better understanding how resistive starch can help protect against cancer.

The original trial was called the CAPP2 study, and the team are now carrying out a follow-up called CaPP3, involving more than 1,800 people with Lynch syndrome.

While it may sound concerning that the rate of colorectal cancers didn't seem affected by the resistive starch, don't worry, the study had good news on that front, too.

The original trial also looked at whether taking aspirin daily could reduce cancer risk. Back in 2020, the [team published](#) results showing that aspirin reduced the risk of large bowel cancers in Lynch syndrome patients by 50 percent.

"Patients with Lynch syndrome are high risk as they are more likely to develop cancers, so finding that aspirin can reduce the risk of large bowel cancers and resistant starch other cancers by half is vitally important," [says Newcastle University geneticist Sir John Burns](#) who ran the trial with Mathers.

"Based on our trial, NICE [the UK's National Institute for Health and Care Excellence] now recommend Aspirin for people at high genetic risk of cancer, the benefits are clear – aspirin and resistant starch work."

The research has been published in [Cancer Prevention Research](#).
<https://bit.ly/3zHpgKW>

Why Did Europeans Evolve Into Becoming Lactose Tolerant?

Famine and disease from millennia ago likely spurred the rapid evolution of the trait on the continent

[Brian Handwerk](#) Science Correspondent

Just 5,000 years ago, even though it was a part of their diet, virtually no adult humans could properly digest milk. But in the blink of an evolutionary eye northern Europeans began inheriting a genetic mutation that enabled them to do so. The trait became common in just a few thousand years, and today it's found in [up to 95 percent of the population](#). By piecing together Neolithic pottery fragments and ancient human genomes, scientists may have solved the riddle of how European lactose tolerance evolved.

In a [study published today](#) in *Nature*, researchers compared

archaeological evidence for 9,000 years of European milk use with genetics, and found an unusually rapid, evolution of lactose tolerance among Europeans well after they first started consuming the beverage. The authors suggest that something more extreme than regular milk consumption drove the genetic change. Exceptional stressors like famines and pathogens may have exacerbated milk's typically mild gastrointestinal effects on the lactose intolerant, creating deadly bouts of diarrhea and dehydration while making the ability to digest milk extra valuable.

"It rewrites the textbooks on why drinking milk was an advantage," says lead author Richard Evershed, director of the [Biogeochemistry Research Center](#) at the University of Bristol. "In order to evolve a genetic mutation so quickly, something has to kill off the people that don't carry it."

The wide-ranging study, led by Evershed and colleagues from the University of Bristol and University College London, included contributions from experts in 20 other countries.

Almost all babies around the world are born with the ability to digest lactose—after all, it's found in breast milk. But about two-thirds of adults can no longer digest the natural milk sugar because the production of a milk-digesting enzyme called lactase switches off after they've finished weaning. That's why the majority of the world's adult population is lactase non-persistent, otherwise known as lactose intolerant.

The other third of the world's adult population has evolved lactose tolerance, meaning they keep producing lactase, and that's particularly true among groups like those of northern European descent.

Shevan Wilkin, a biological anthropologist at the Max Planck Institute for the Science of Human History, says that until perhaps five years ago, the lactose tolerance story seemed simple. Once groups of humans began herding animals and drinking their milk

the health benefits of milk favored those who could digest it, while digestive ailments worked against the success of the intolerant, so the genetic mutation that helped humans digest milk eventually spread through those populations.

“Then we realized some crazy trends,” says Wilkin, who wasn’t involved in the study. “When you look at the ancient genomes no one has lactose tolerance until recently, the past few thousand years.” For a genetic trait to become widespread that quickly, there should be a very important reason why people who have it survive and reproduce, while others die off.

“We also realized that huge populations throughout the steppe, people in modern day Kazakhstan, Russia, Mongolia, people who are drinking a ton of milk, aren’t lactase persistent at all.” If the simple benefits of drinking a lot of milk produced and propagated a mutation for lactase persistence, the steppe dwellers surely should have evolved the trait just as Europeans did.

What’s more, studies of ancient human DNA have shown that the genetic mutation that enabled European lactase persistence doesn’t look like something that conferred a marginal nutritional advantage. In European genomes it is the [single trait most favored by positive natural selection](#) over the past 10,000 years.

The authors used several different lines of inquiry to delve into the murky past of European milk.

Richard Evershed and colleagues mapped human milk use during the past 9,000 years, creating an enormous database from 6,899 animal fat residues derived from 13,181 fragments of pottery from 554 archaeological sites around Europe. Over the past three decades scientists, with experts like Evershed at the fore, have developed methods to analyze ancient pottery and reveal evidence of what it contained.

As luck, or science, would have it, milk fat is absorbed into ancient pottery and preserved at a remarkable level. Studying carbon

isotope compositions of the two major fatty acids that exist and persist in degraded animal fats in pots reveals that milk leaves a distinctive signature because it’s made in a different way than carcass fat in ruminant animals.

Evershed found plentiful evidence that humans were drinking milk widely, across Europe, from around 9,000 years ago.

Co-author Mark Thomas, an evolutionary geneticist at University College London, launched his own mapping project, this one charting where and when the genetic variant appeared that enabled lactase persistence in Europeans. Combing through DNA sequences of more than 1,700 prehistoric humans he found that its first appearance was not till some 5,000 years ago, or some 4,000 years after regular milk consumption began. The mutation has become commonplace in the short time since then but its late appearance means humans were drinking milk for thousands of years before they could digest it.

Thomas and colleagues compared Evershed’s datasets for historic European milk use with the genetic evidence for the rise of lactose tolerance. They found no relationship between changes in milk use over time and the rise of humans’ ability to tolerate lactose.

That’s puzzling, because for humans who don’t digest lactose the sugary milk component can cause intestinal problems ranging from flatulence to diarrhea. For this reason, the lactose intolerant don’t drink much milk—or at least that’s what many had erroneously assumed.

In fact, the work of co-author George Davey Smith shows that they do drink milk, according to his study of the UK Biobank’s data, which includes over 500,000 living individuals. His analysis found virtually no difference between the milk consumption of persistent and non persistent adults. He also found that most non persistent milk drinkers reported no long-term health impacts, nothing that would shorten their lifespans or reduce their ability to reproduce.

“So how could people possibly have been dairy farmers when they were lactase non-persistent?” Evershed asks. “Because they can happily consume milk and get health benefits from it.”

George Davey Smith’s finding created another question for researchers; if lactose intolerant individuals can drink milk with no major ill effects, what drove the dramatic genetic shift that caused so many Europeans to quickly develop lactose tolerance?

Some factor or factors must have fast-forwarded the evolution of lactose tolerance, likely by making it critically important and even a matter of life and death. “That’s where we started imagining scenarios where this would be the case,” Evershed explains.

Mark Thomas theorizes that famine may have played a major role. Typically, most non-tolerant adults won’t fare too poorly after drinking milk, he notes. “You get flatulence, diarrhea, it’s not nice, it might be unpleasant and embarrassing, but nobody ever died of lactose intolerance.”

“But if you have diarrhea when you are severely malnourished then you’ve got real problems,” he continues. “That’s a major cause of death in the world even today.” If foods like grain run out during a famine, non-persistent humans may resort to consuming a lot more dairy, exactly when they shouldn’t, which could have the biggest detrimental impact on their health.

Davey Smith, the director of the MRC Integrative Epidemiology Unit at the University of Bristol has a different idea with a similar concept; he theorizes that pathogens played a major role. Though his work shows drinking milk isn’t hurting the health of non-tolerant adults today, it’s potentially a major problem among those suffering from gut disturbances, dehydration and other ailments. During times when humans were living close together, amongst domestic animals and lacking proper hygiene, disease likely became widespread and may have severely weakened many individuals for whom lactose driven diarrhea and dehydration

proved fatal. On the other hand, those who could drink and digest milk had a resource to help them pull through.

The team put these ideas to a test using models which suggested that the gene variant for lactase persistence did increase in populations when they were impacted by famine or pathogens.

The environmental stressors that drove lactose tolerance could have worked in tandem, and they might have been very different during each of the five different times it is known to have evolved in Europe, the Middle East and Africa. “In Europe it could be about settlements and famine, while in Africa for example, it could be much more about droughts and higher disease loads,” Thomas says.

The group’s methods might also be employed to find out what happened where humans never did develop the ability to digest milk when common sense suggests they might have.

“Because across the [Eurasian] steppe people who are not lactase persistent are drinking a ton of milk,” says Shevan Wilkin. “What was happening where that didn’t evolve, when it did evolve in Europe?”

Wilkin adds that scientists have been floating various ideas to explain the mysteries of milk digestion, including how lactose tolerance evolved so late and so quickly, and why heavy milk consumers like the steppe dwellers remain lactose intolerant. Now, she says, a framework exists that can further investigate those questions. “It’s such an impressive undertaking. And through that they’ve come up with some ideas that make a lot of sense.”

<https://bit.ly/3cQJCB1>

Gulf Coast tests confirm deadly tropical soil bacterium now endemic to US

The bacterium causes melioidosis, which is hard to diagnose and resistant to some drugs.

[Beth Mole](#)

For years, health officials in the US noted sporadic, mysterious

cases of a foreign bacterial infection, called melioidosis. The infection—which is difficult to diagnose, tricky to treat, and often deadly—was thought to only strike travelers or those who came in contact with contaminated imported goods or animals. Yet, now and then, an American would inexplicably fall ill—no recent travel, no clear links.

Now, health officials have a definitive explanation. And it confirms a dreaded, long-held suspicion: The deadly bacterium is foreign no more. Rather, it's a permanent US resident entrenched in American soil.

Three samples taken from soil and puddle water in the Gulf Coast region of southern Mississippi tested positive for the bacterium, [officials from the Centers for Disease Control and Prevention announced Wednesday](#). The sampling was part of an investigation into two mysterious cases in the area that occurred in 2020 and 2022. The positive test results mark the first time that investigators have caught the deadly germ in US environmental samples, though they've been looking for it for years.

It's unclear how long the bacterium has resided in the US or how widespread its distribution has become. But CDC modeling suggests the environmental conditions of the Gulf Coast states are conducive to the bacterium's growth. The agency has called for extensive environmental sampling.

While the finding explains the puzzling cases, the most important thing now is for health officials to get the word out. This is no longer a traveler's disease. In [a health advisory released yesterday](#), the CDC emphasized that its notice "serves to alert clinicians and public health officials throughout the country to consider melioidosis in patients whose clinical presentation is compatible with signs and symptoms of the disease, regardless of travel history to international disease-endemic regions, as melioidosis is now considered to be locally endemic in areas of the Gulf Coast region

of Mississippi."

The bacterium at hand is *Burkholderia pseudomallei*, which lives in the soil and water of tropical and subtropical regions and causes rare but dangerous sporadic infections. The areas with the highest endemicity are in Southeast Asia and northern Australia, but it has also popped up in areas of Southwest Asia, Africa, the Pacific, and the Americas, such as Peru, Brazil, Haiti, and some US territories, including Puerto Rico.

B. pseudomallei causes melioidosis by transmitting in various ways, all involving direct contact with contaminated soil and water. People can be infected if they ingest contaminated soil, water, or food; if they breathe in contaminated dust or water droplets; or if contaminated soil or water comes in contact with a break in the skin. The people more at risk of melioidosis than others are those with specific conditions, such as diabetes, heavy alcohol use, chronic lung disease, chronic kidney disease, and conditions that weaken immune responses. One bit of good news is that the infection rarely transmits from person to person.

The ensuing symptoms of [melioidosis](#) can depend on which route *B. pseudomallei* takes into the body. If it enters through a skin wound, it could cause pain, swelling, and an abscess. If it gets into the blood, it can cause joint pain, abdominal discomfort, and disorientation. If it enters through the lung, it can cause coughing and chest pain. And if it goes systemic, it can cause weight loss, a brain infection, and seizures. Overall, the symptoms can appear nebulous and can easily be mistaken for other conditions. It has been described as "[the great mimicker](#)" because of how frequently and easily it is mistaken for other serious infections, such as tuberculosis.

Its indistinct nature contributes to its deadliness. *B. pseudomallei* is naturally resistant to many commonly used antibiotics. Any delays to an accurate diagnosis can allow the bacterium to cause more

severe disease. According to the CDC, melioidosis is fatal in 90 percent of people who are not properly treated. When people are treated with the correct antibiotics, the fatality rate falls below 40 percent. And if patients have access to intensive care and the right drugs, the fatality rate drops to around 20 percent.

For all these reasons, the US government considers *B. pseudomallei* a [bioterrorism threat](#), listing it as a [Tier 1 Select Agent](#) along with anthrax bacteria (*Bacillus cereus* Biovar *anthracis*) and Ebola virus.

US cases

According to the CDC, the US averages about 12 melioidosis cases per year, most of which have been travel-related. But there have been notable and puzzling exceptions over the years.

Last year, melioidosis made headlines when [four people](#) in four states became infected with the same strain of *B. pseudomallei*. The first unexplained case, which was fatal, occurred in an adult in Kansas in March. Then, another adult in Minnesota survived, and a [4-year-old in Texas](#) was left with brain damage. Last, a child in Georgia was identified as a case through a post-mortem exam.

In October, investigators announced [a break in the puzzling outbreak](#): the strain of *B. pseudomallei* causing the infections was found in an aromatherapy room spray, made in India, which contained "gemstones." Specifically, it was the [Better Homes & Gardens Lavender & Chamomile Essential Oil Infused Aromatherapy Room Spray with Gemstones](#), which Walmart sold.

Though investigators suspected an imported product from the start, the cluster drew attention to other puzzling cases in the US—cases that had raised concern that *B. pseudomallei* was lurking in US soil. In 2015, for instance, researchers at the CDC surveyed the 34 human melioidosis cases in the US between 2008 and 2013, finding that cases appeared to be increasing each year in that period. [The study](#), published in the CDC's Morbidity and Mortality Weekly Report, concluded that *B. pseudomallei* may be an emerging

infectious disease in the US.

"Of note, three cases of melioidosis occurred in US residents with no travel history either outside of the United States or to regions where melioidosis is endemic, possibly indicating unrecognized sources of exposure in the United States," the researchers wrote. "Therefore, being aware that this infection can be seen in persons without an obvious history of travel to locations where *B. pseudomallei* is endemic is important."

The cautionary note came up again in [a case report published in 2020](#), also written by CDC researchers and published in the journal Emerging Infectious Diseases. The report documented a puzzling case of melioidosis from 2018 in a 63-year-old man from Atascosa County, Texas—which is in the Gulf Coast region. The man had no relevant travel history, only reporting a trip to Mexico taken 30 years before his illness.

Connecting the dots

The CDC researchers quickly drew a line between that case and one in 2004, which occurred in a Texas resident from the same county. Like in 2018, the 2004 case was not explained by any recent travel. At the time, investigators had hypothesized that the 2004 infection stemmed from an exposure the person had 62 years prior while serving in Southeast Asia during World War II.

Genetic testing concluded that the *B. pseudomallei* strains in the 2004 and 2018 Texas cases were similar and were most closely related to other strains found in the Americas, not Southeast Asia.

The connection helped spur investigators to dig deep into the 2018 case. They took 56 environmental samples from the man's home, a small, rural ranch with no running water. The sampling effort included the two large water storage tanks (a 500-gallon and a 1,600-gallon) that the man used on the property and cleaned once or twice a month by climbing inside to scrub the walls. The onset of his melioidosis case began two days after a cleaning.

Ultimately, all of the environmental samples came back negative. Still, the CDC researchers held onto the possibility that *B. pseudomallei* was around Texas, lurking somewhere in the environment. The two cases, connected by location and bacterial genetics, "suggests *B. pseudomallei* might be present in the environment in this area," they concluded. But, they went on, "Only when *B. pseudomallei* is isolated from the environment can it be definitively stated that *B. pseudomallei* is endemic to the continental United States."

They ended by repeating the cautionary note to clinicians: "Increased awareness among healthcare workers and diagnostic laboratory personnel for melioidosis as a disease potentially endemic to the southwestern United States is critical to improve case outcomes and prevent laboratory exposures."

Two years after that report was published, the definitive evidence of endemicity came in the way of two similarly linked cases several hundred miles away in Mississippi. The two cases occurred in July 2020 and May 2022 among two people who lived close to each other in southern Mississippi. Neither had recent travel history outside the country, and they were both infected with the same strain of *B. pseudomallei*. Both people were hospitalized with sepsis due to pneumonia but recovered upon receiving proper antibiotic treatment.

Health officials in Mississippi collected environmental samples from both people's homes. Three soil and water samples from the home of the 2020 case tested positive at the CDC for *B. pseudomallei*. The finding indicates that "bacteria from the environment was the likely source of infection for both individuals and has been present in the area since at least 2020," the CDC concluded.

CDC recommendations

In addition to raising awareness of this threat to clinicians, the CDC

advises those living in the Gulf Coast region, where *B. pseudomallei* is now considered endemic. First, given the small number of melioidosis cases in the US and elsewhere, the CDC still believes the risk to the general population is "very low."

But, for those who have health conditions that may put them at higher risk—such as diabetes, chronic kidney disease, chronic lung disease, or excessive alcohol use—the CDC recommends the following extra precautions:

** Avoid contact with soil or muddy water, particularly after heavy rains, and protect open wounds with waterproof dressings.*

** Wear waterproof boots when gardening, doing yard work, or doing agricultural work, which can prevent infection through the feet and lower legs—particularly after flooding or storms.*

** Wear gloves to protect your hands when working directly with soil.*

<https://bit.ly/3zi8w4g>

'Never seen anything like it': Impeccably preserved Jurassic fish fossils found on UK farm

One 3D fossil resembled a singing animatronic fish toy.

By [Jennifer Nalewicki](#)

A farm in England was the unlikely source of a Jurassic jackpot: a treasure trove of 183 million-year-old fossils. On the outskirts of Gloucestershire in the Cotswolds, beneath soil that is currently trampled under the hooves of grazing cattle, researchers recently uncovered the fossilized remains of fish, giant marine reptiles called ichthyosaurs, squids, insects and other ancient animals dating to the early part of the Jurassic period (201.3 million to 145 million years ago).

Of the more than 180 fossils logged during the dig, one of the standout specimens was a three-dimensionally preserved fish head that belonged to *Pachycormus*, an extinct genus of ray-finned fishes. The fossil, which researchers found embedded in a hardened

limestone nodule poking out of the clay, was exceptionally well preserved and contained soft tissues, including scales and an eye. The 3D nature of the pose of the specimen's head and body was such that the researchers couldn't compare it to any other previous find.



The 3D fossil of a Jurassic fish known as a Pachycormus was one of more than 180 fossils found on a farm in the UK. (Image credit: Courtesy Sally and Neville Hollingworth)

"The closest analogue we could think of was Big Mouth Billy Bass," said Neville Hollingworth, a field geologist with the University of Birmingham who discovered the site with his wife, Sally, a fossil preparator and the dig's coordinator. "The eyeball and socket were well preserved. Usually, with fossils, they're lying flat. But in this case, it was preserved in more than one dimension, and it looks like the fish is leaping out of the rock," Hollingworth told Live Science. "I've never seen anything like it before," Sally Hollingworth added. "You could see the scales, skin, spine — even its eyeball is still there."

The sight astounded the Hollingworths so much that they contacted ThinkSee3D, a company that creates digital 3D models of fossils, to create an [\(opens in new tab\)interactive 3D image](#) [\(opens in new tab\)](#) of the fish to help bring it to life and to allow researchers to study it more closely.

Most of the fossils the Hollingworths and a team of scientists and specialists unearthed were located behind the farm's cowshed. (The farm is home to a herd of English longhorn — a British breed of beef cattle with long, curved horns — many of which kept a close eye on the excavation.)

"It was a bit unnerving digging when you're being watched by a

herd of longhorn," Sally Hollingworth told Live Science.

At one time, this region of the United Kingdom was completely submerged by a shallow, tropical sea, and the sediments there likely helped preserve the fossils; Neville Hollingworth described the Jurassic beds as slightly horizontal, with layers of soft clays under a shell of harder limestone beds.

"When the fish died, they sank to the bottom of the seabed," said fossil marine reptile specialist Dean Lomax, a visiting scientist at the University of Manchester in the U.K. and a member of the excavation group. "As with other fossils, the minerals from the surrounding seabed continually replaced the original structure of the bones and teeth. In this case, the site shows that there was very little to no scavenging, so they must've been rapidly buried by the sediment. As soon as they hit the seabed, they were covered over and protected immediately."

During the four-day dig earlier this month, the eight-person team used a digger to excavate 262 feet (80 meters) across the farm's grassy banks, "pulling back layers to reveal a small slice of geological time," Neville Hollingworth said. A number of diverse specimens dated to the Toarcian age (a stage of the Jurassic that occurred between 183 million and 174 million years ago) and included belemnites (extinct squid-like cephalopods), ammonites (extinct shelled cephalopods), bivalves and snails, in addition to fish and other marine animals.

"It's important that we can compare these fossils with other Toarcian age fossil sites, not only in the U.K. but also across Europe and potentially sites in America," Lomax said. He pointed to Strawberry Bank Lagerstätte, an early Jurassic site in southern England, as one such example. The group plans to continue studying the specimens and is working toward publishing the findings. Meanwhile, a selection of the fossils will be placed on display at the Museum in the Park in Stroud.

<https://bit.ly/3oGpFiX>

Lava Tubes on the Moon Maintain Comfortable Room Temperatures Inside

Searching for a comfortable place to set up a research station on the Moon? Look no further than the interior parts of lunar pits and caves.

While lack of air will be an issue, new research indicates these underground sanctuaries have steady temperatures that hover around 17 Celsius, or 63 Fahrenheit, even though the Moon's surface heats up to about 127 C (260 F) during the day and cool to minus 173 C (minus 280 F) at night.

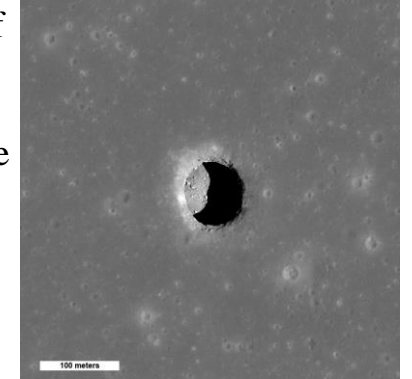
Lunar pits, or lava tubes were discovered in 2009 by the Lunar Reconnaissance Orbiter and Japan's Kaguya spacecraft. These are deep holes on the moon that could open into vast underground tunnels. They likely could serve as a safe shielding from cosmic rays, solar radiation and micrometeorites for future human lunar explorers. But now we know they could provide thermally stable sites for lunar exploration.

These long, winding lava tubes are like structures we have on Earth. They are created when the top of a stream of molten rock solidifies and the lava inside drains away, leaving a hollow tube of rock. For years before their existence was confirmed, scientists thought there were hints that the Moon had lava tubes based on observations of long, winding depressions carved into the lunar surface by the flow of lava, called sinuous rilles.

So far, about 200 lunar pits have been found and at least 16 of these are probably collapsed lava tubes, with the potential for 'livable' space, said Tyler Horvath, a UCLA doctoral student in planetary science, who led the new research. Two of the most prominent pits have visible overhangs that clearly lead to some sort of cave or void, and there is strong evidence that another's overhang may also lead to a large cave.

Horvath processed images from the Diviner Lunar Radiometer Experiment — a thermal camera and one of six instruments on LRO — to find out if the temperature within the pits diverged from those on the surface. Diviner is designed to measure surface temperatures on the Moon, and Horvath's team had to focus in on extremely small areas to get their data.

They focused on a pit found in the Sea of Tranquility (Mare Tranquillitatis). This image, below, was taken as the Sun was almost straight overhead, illuminating the region. By comparing this image with previous images that have different lighting, scientists can estimate the depth of the pit. They believe it to be over 100 meters.



This is a spectacular high-Sun view of the Mare Tranquillitatis pit crater, revealing the overhang and deep, dark pit. This image from LRO's Narrow Angle Camera is 400 meters (1,312 feet) wide, north is up. Credits: NASA/Goddard/Arizona State University

The researchers used computer modeling to analyze the thermal properties of the rock and lunar dust and to chart the pit's temperatures over a period of time. Their research, [recently published in the journal Geophysical Research Letters](#), revealed that temperatures within the permanently shadowed reaches of the pit fluctuate only slightly throughout the lunar day, remaining at around 17 C (63 F). If a cave extends from the bottom of the pit, as images taken by the Lunar Reconnaissance Orbiter Camera suggest, it too would have this relatively comfortable temperature. The researchers think the overhang is responsible for the steady temperature, limiting how hot things get during the day and preventing heat from radiating away at night.

However, if this particular pit was to be used as a habitat or

research station, there would likely be a heat problem just inside the pit. The sunbaked part of the pit floor not protected by the overhang hits daytime temperatures close to 150 C (300 F), which is even hotter than the Moon's surface.

"Because the Tranquillitatis pit is the closest to the lunar equator, the illuminated floor at noon is probably the hottest place on the entire moon," said Horvath.

Since a day on the Moon lasts nearly 15 Earth days, the lunar surface is constantly bombarded by sunlight and is frequently hot enough to boil water. Conversely, the equally long lunar nights (also 15 Earth days long) reach incredibly cold temperatures. Any habitat or base would mean inventing heating and cooling equipment that can operate under these conditions, as well as ways to produce enough energy to power it nonstop. This could prove to be an insurmountable barrier to lunar exploration or habitation.

However, the researchers say that building bases in the shadowed parts of these pits allows scientists to focus on other challenges, like growing food, providing oxygen for astronauts, gathering resources for experiments and expanding the base.

"Humans evolved living in caves, and to caves we might return when we live on the moon," said UCLA professor of planetary science David Paige, who leads the Diviner Lunar Radiometer Experiment and participated in the research.

<https://bit.ly/3zigWbD>

Primordial Soup: Scientists Discover New "Origins of Life" Chemical Reactions

The reaction generates the building blocks of proteins and DNA: amino acids and nucleic acids.

Four billion years ago, the Earth looked very different than it does today. It was devoid of life and covered by a vast ocean. Over the course of millions of years, life emerged in that primordial soup. For a long time, researchers have theorized how molecules came

together to spark this transition. Now, scientists at Scripps Research have discovered a new set of chemical reactions that use ammonia, cyanide, and carbon dioxide—all thought to be common on the early Earth—to generate amino acids and nucleic acids, the building blocks of proteins and DNA.

"We've come up with a new paradigm to explain this shift from prebiotic to biotic chemistry," says Ramanarayanan Krishnamurthy, PhD, and an associate professor of chemistry at Scripps Research. "We think the kind of reactions we've described are probably what could have happened on early Earth." Krishnamurthy is the lead author of the new paper that was published in the journal *Nature Chemistry* on July 28, 2022.

In addition to giving scientists insight into the chemistry of the early Earth, the newly discovered chemical reactions are also useful in certain manufacturing processes. For example, in the generation of custom-labeled biomolecules from inexpensive starting materials. [Earlier this year](#), Krishnamurthy's team showed how cyanide can enable the chemical reactions that turn prebiotic molecules and water into basic organic compounds required for life. This one worked at room temperature and in a wide pH range, unlike previously proposed reactions. The scientists wondered whether, under the same conditions, there was a way to generate amino acids, which are more complex molecules that compose proteins in all known living cells.

In cells today, amino acids are generated from precursors called α -keto acids using both nitrogen and specialized proteins called enzymes. Scientists have discovered evidence that α -keto acids likely existed early in Earth's history. However, many researchers have hypothesized that before the advent of cellular life, amino acids must have been generated from completely different precursors, aldehydes, rather than α -keto acids, since enzymes to carry out the conversion did not yet exist. But that idea has led to

debate about how and when the switch occurred from aldehydes to α -keto acids as the key ingredient for making amino acids.

After their success in using cyanide to drive other chemical reactions, Krishnamurthy's group suspected that cyanide, even without enzymes, might also help turn α -keto acids into amino acids. Because they knew nitrogen would be required in some form, they added ammonia—a form of nitrogen that would have been present on the early Earth. Then, through trial and error, they discovered a third key ingredient: carbon dioxide. With this mixture, they quickly started seeing amino acids form.

“We were expecting it to be quite difficult to figure this out, and it turned out to be even simpler than we had imagined,” says Krishnamurthy. “If you mix only the keto acid, cyanide, and ammonia, it just sits there. As soon as you add carbon dioxide, even trace amounts, the reaction picks up speed.”

Because the new reaction is relatively similar to what occurs inside cells today—except for being driven by cyanide instead of a protein—it seems more likely to be the source of early life, rather than drastically different reactions, the scientists say. The research also helps bring together two sides of a long-standing debate about the importance of carbon dioxide to early life, concluding that carbon dioxide was key, but only in combination with other molecules.

In the process of studying their chemical soup, Krishnamurthy and his colleagues discovered that a byproduct of the same reaction is orotate, a precursor to nucleotides that make up DNA and RNA. This indicates that the same primordial soup, under the right conditions, could have given rise to a large number of the molecules that are required for the key elements of life.

“What we want to do next is continue probing what kind of chemistry can emerge from this mixture,” says Krishnamurthy.

“Can amino acids start forming small proteins? Could one of those

proteins come back and begin to act as an enzyme to make more of these amino acids?”

Reference: “Prebiotic synthesis of α -amino acids and orotate from α -ketoacids potentiates transition to extant metabolic pathways” by Sunil Pulletikurti, Mahipal Yadav, Greg Springsteen and Ramanarayanan Krishnamurthy, 28 July 2022, Nature Chemistry.

DOI: [10.1038/s41557-022-00999-w](https://doi.org/10.1038/s41557-022-00999-w)

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<https://bit.ly/3PMnEh3>

Balloon fleet senses earthquakes from stratosphere

A new study in AGU's Geophysical Research Letters reports on the first detection of a large, distant earthquake in a network of balloon-bound pressure sensors in the stratosphere.

The technique could one day be applied on Venus, whose hot, dense and corrosive atmosphere limits our ability to sense Venus-quakes from the planet's surface. The balloons could also be used on Earth in hard-to-reach places.

Monitoring [seismic activity](#) on other planets is critical for learning about their interior structures, but unlike on Earth, planetary scientists can't rely on a global network of ground-based sensors. Instead, they turn to the atmosphere.

When an earthquake hits, the vibrating ground sends infrasound high into the atmosphere, where the balloons and their instruments are waiting. The balloons float through the stratosphere for several months after launch, passively following high-altitude atmospheric patterns. At about 11 meters in diameter and 30 kilograms (66 pounds), the balloons can support up to four instruments.

Seismology is relatively new in the stratosphere; the balloons are mostly used for atmospheric science. Previous research has confirmed that these [balloon](#)-based sensors can pick up small, local quakes, but until now, a multi-balloon network had not yet detected [large earthquakes](#) at a great distance.

On December 14, 2021, a magnitude 7.3 earthquake hit Indonesia's Flores Sea. Within 10 minutes, four of IASE's Strateole-2 balloons within a 3,000 kilometer (1860-mile) radius detected the resulting infrasound, at altitudes as high as 20 kilometers (12 miles). From those [sensor data](#), Garcia's research team was able to accurately back-calculate the earthquake's magnitude and several other key parameters about both the quake and planetary structure. They were even able to track the dispersion of the seismic wave across the surface with their network.

"We are very, very happy because it was not only a single balloon that detected the earthquake, it was sensed on multiple balloons," says Raphael Garcia, lead author on the new study and a [planetary scientist](#) at the Institut Supérieur de l'Aéronautique et de l'Espace of the University of Toulouse.

The study is an important proof-of-concept for applying this seismic monitoring technique on Venus. While the balloons have only been tested in Earth's atmosphere, Garcia and his colleagues believe they will work in Venus' carbon dioxide-rich atmosphere too.

Vivacious Venus

In 2021, scientists studying Venus began referring to the next ten years as "the decade of Venus," as three missions to the planet have been accepted for the early 2030s. Venus, Earth's "sister planet," intrigues [planetary scientists](#) with its unknown internal structure and poorly understood long term interactions between tectonics and atmosphere that ended up with such an inhabitable world compared to the nearby Earth. "The story for our interest in Venus is that we know nothing of its interior," Garcia says. "We don't know how it's made inside, and on Earth, seismology is one of the best tools to figure that out."

As part of the decade of Venus, several teams are working on balloon-based seismic monitoring, but the new study is the first to

successfully capture large, natural quakes with multiple balloons, Garcia says.

"The search for detecting a big quake on stratospheric balloons, it's a bit competitive," he says. "But it's a nice competition, because in the end, we're working to demonstrate the same concept." Still, he is pleased their team nabbed this accomplishment. The proposal for balloon-based seismic monitoring on Venus, called Phantom, will be submitted to the New Frontiers NASA missions in collaboration with JPL-NASA and North Carolina State University.

The network's success also highlights the potential for balloon-based [seismic monitoring](#) to complement areas that are difficult to monitor with a ground-based network, such as the sea floor. The [balloons](#) could also be deployed as a rapid-response tool for monitoring aftershocks.

More information: Raphael F. Garcia et al, *Infrasound from large earthquakes recorded on a network of balloons in the stratosphere*, *Geophysical Research Letters* (2022). [DOI: 10.1029/2022GL098844](https://doi.org/10.1029/2022GL098844)

<https://bit.ly/3JxEjTz>

Scientists May Have Found a Key Shift Between The Brains of Humans And Neanderthals

Scientists experimenting on mice have found evidence that key parts of the modern human brain take more time to develop than those of our long extinct cousin, the Neanderthal.

[Michelle Starr](#)

Like the hare and the tortoise, slow and steady is the winner here. The extra time is caused by protein differences that also appear to reduce chromosome errors, ultimately resulting in a healthier, more robust population.

The study's results imply that this step in the development of our neocortex (the wrinkled outer layer responsible for higher order thinking) plays a role in protecting us from disease, a feature [Neanderthals](#) appear to be missing.

In recent years, advances in genetics have allowed scientists to sequence DNA extracted from [ancient remains](#), revealing detailed information about how the Neanderthal genome compares and contrasts with our own.

We know, for example, of around 100 amino acids – the compounds that make up proteins – that changed when modern humans diverged from the branch that gave rise to Neanderthals and another close cousin, the [Denisovans](#).

Amino acid substitution can have significant effects, but it was unclear what functions these substitutions changed between humans and Neanderthals.

Six of the identified substitutions exist in proteins already known to play a role in the distribution of chromosomes during cell division. So a team of researchers led by geneticist Felipe Mora-Bermúdez of the Max Planck Institute of Molecular Cell Biology and Genetics in Germany conducted experiments to see if they could determine the role these amino acid changes might play in neocortex development. The natural subject was laboratory mice, which happen to share with Neanderthals (and apes) those same six amino acids within the relevant proteins. Using [CRISPR](#) Cas-9, the researchers substituted those amino acids for those found in modern humans.

They also took the research in the opposite direction. They grew [organoids of human brains](#) from embryonic [stem cells](#) – lumps of brain tissue that are not alive or sentient – and replaced the modern human amino acids with the Neanderthal/mouse/ape variants.

The results were striking and fascinating.

"We found that three modern human amino acids in two of the proteins cause a longer metaphase, a phase where chromosomes are prepared for cell division," [Mora-Bermúdez explained](#), "and this results in fewer errors when the chromosomes are distributed to the daughter cells of the neural stem cells, just like in modern humans."

In addition, the metaphase in the Neanderthalized human organoids

was shorter, resulting in twice the number of chromosome separation errors compared to the control organoids. This suggests that three modern human amino acid substitutions are responsible for fewer chromosome distribution errors compared to Neanderthals. Since errors in the number of chromosomes, known as [polysomies](#), can result in serious disorders, as well as cancers such as leukemia and carcinoma, the results suggest that the change was to the benefit of modern humans. They also suggest that brain function in Neanderthals may have been impacted by chromosomal disorders at a higher rate than we see in modern humans.

"The present data imply that the probability of any such detrimental effects of chromosomal mis-segregation may be lower in modern humans than in Neanderthals, Denisovans, and apes," [the researchers wrote in their paper](#).

"Further work is needed to address the importance of these effects for traits characteristic of modern humans."

The research has been published in [Science](#).

<https://bit.ly/3OMii48>

US regulators will certify first small nuclear reactor design

NuScale will get the final approval nearly six years after starting the process.

[John Timmer](#)

On Friday, the Nuclear Regulatory Commission (NRC) [announced](#) that it would be issuing a certification to a new nuclear reactor design, making it just the seventh that has been approved for use in the US. But in some ways, it's a first: the design, from a company called NuScale, is a small modular reactor that can be constructed at a central facility and then moved to the site where it will be operated.

The move was expected after the design [received an okay](#) during its final safety evaluation in 2020.

Small modular reactors have been promoted as avoiding many of the problems that have made large nuclear plants exceedingly expensive to build. They're small enough that they can be assembled on a factory floor and then shipped to the site where they will operate, eliminating many of the challenges of custom, on-site construction. In addition, they're structured in a way to allow passive safety, where no operator actions are necessary to shut the reactor down if problems occur.



Enlarge / NuScale's reactor-in-a-can. NuScale

Many of the small modular designs involve different technology from traditional reactors, such as the use of molten uranium salts as the reactor fuel. NuScale has a much more traditional design, with fuel and control rods and energy transported through boiling water. Its operator-free safety features include setting the entire reactor in a large pool of water, control rods that are inserted into the reactor by gravity in the case of a power cut, and convection-driven cooling from an external water source.

NuScale started the certification process in 2016. According to the NRC, that process required the company to submit technical information that allows the Commission to evaluate it as follows:

Applications must closely analyze the design's appropriate response to accidents or natural events. Applications must also lay out the inspections, tests, analyses and acceptance criteria that will verify the construction of key design features. In addition, the NRC also requires design certification applicants to assess how the designs protect the reactor and spent fuel pool from the effects of a large commercial aircraft impact.

Once complete, the certification is published in the Federal Register,

allowing the design to be used in the US. Friday's announcement says that the NRC is all set to take the publication step.

The NRC will still have to weigh in on the sites where any of these reactors are deployed. Currently, one such site is in the works: a project called the Carbon Free Power Project, which will be situated at Idaho National Lab. That's expected to be operational in 2030 but has been facing some [financial uncertainty](#). Utilities that might use the power produced there have grown hesitant to commit money to the project.