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Gigantic Antarctic Lake Suddenly Disappears in Monumental Vanishing Act

Gigantic lake in Antarctica whch abruptly vanished from view during winter 2019

Peter Dockrill

As the world gets warmer, staggering transformations are taking place in some of Earth's coldest locations – events that might go completely unnoticed by humans, were it not for our eyes in the sky. In a new study, satellite observations reveal one such stunning phenomenon: The sudden disappearance of a gigantic lake in Antarctica, which abruptly vanished from view during winter 2019.

This was no small body of water, researchers report, with estimates the lake on Amery Ice Shelf in East Antarctica held some 600–750 million cubic meters (21–26 billion cubic feet) of water: more than all the water in Sydney Harbor, or roughly twice the volume of San Diego Bay.

Of course, that much water doesn't simply disappear into thin air. In this case, scientists say the huge reservoir most likely became too much for the ice layer underneath struggling to support it.

Above: Landsat 8 images of the ice-covered lake (left) and the doline after the waters vanished (right). **(Warner et al., Geophysical Research Letters, 2021)**

"We believe the weight of water accumulated in this deep lake opened a fissure in the ice shelf beneath the lake, a process known as hydrofracture, causing the water to drain away to the ocean below," says glaciologist Roland Warner from the University of Tasmania.

The deluge, which Warner likens to the heavy flow of Niagara Falls – except ultimately into the ocean under the ice shelf – took place over about three days, during which the entire lake was drained, satellite observations suggest.

Those readings didn't just capture what the scene looked like from above; measurements from NASA's ICESat–2 also registered changes in ice shelf elevation resulting from the water displacement.

Understandably, when such a giant pool of water is removed from a floating ice shelf, you would expect the ice shelf underneath to rise up, free of the previous weight pressing it down. Here, the affected region surrounding the lake lifted by up to 36 meters, the researchers say.

While the growing emergence of meltwater lakes and streams across the surface of Antarctica are generally considered evidence of climate change, the researchers say we don't yet know enough about these hydrofracturing events themselves to say whether they too are linked.

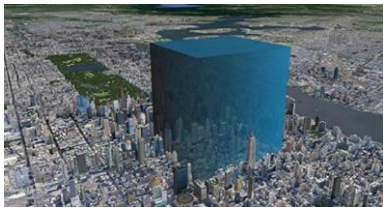
But it's something we need to watch, because when these lakes burst through unstable ice shelves, the volume of that water ingress directly adds to the ocean's water volume. In accumulation, this phenomenon - together with other melting processes - contributes to eventual sea level rise.

"Antarctic surface melting has been projected to double by 2050, raising concerns about the stability of other ice shelves," the researchers write in their study, noting that "processes such as hydrofracture and flexure remain under-studied, and ice-sheet models do not yet include realistic treatment of these processes."

It's time we rectified that, because the flow of meltwater shows no signs of stopping.

After the lake's disappearance in winter 2019, the lake began to fill again as ice melt picked up in summer 2020, peaking at a flow of over 1 million cubic meters of water per day flowing into the icy cavity left behind, called a doline.

It's not fully clear if this newer lake will also vanish through fractures in the ice, or when that might occur, the researchers say, but it's possible that the pooled meltwater in the doline could already be leaking to the ocean.



"It does appear that the fracture reopened briefly during the 2020 summer melt season, so it is certainly a system to watch," Warner says.

Above: Visualization of the 600-750 million cubic meters of water lost to the ocean from the lake. **(Philipp Arndt/Scripps Institution of Oceanography at UC San Diego)**

"This event does raise new questions about how common these deep ice-covered lakes are on ice shelves and how they evolve."

The findings are reported in *Geophysical Research Letters*.

https://bit.ly/3ylATMA

From nerve agent simulant, to pharma ingredient

Scientists in South Korea have shown how to transform a chemical warfare agent simulant into a common drug.

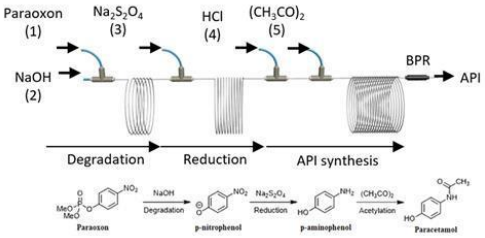
By Matthew Blow

Chemical warfare agents are toxic by design, their aim to injure, disable and kill. Despite international conventions that greatly limit their proliferation, hefty stockpiles pose risks of deliberate or accidental release, and disposal is not a straightforward matter.

'The methods commonly practiced for the neutralisation of organophosphonate agents are incineration and alkaline hydrolysis,' says Se-Jun Yim, a researcher at Pohang University of Science and Technology, who points out secondary contamination issues such as air pollution and the landfill of hazardous waste. Although alternative methods can cleanly capture and neutralise these agents, the batch processes involved are impractical and inefficient, feasible for dealing only with milligram quantities.

Now Yim and his colleagues have developed a new device to break down a toxic nerve agent simulant – dimethyl 4-nitrophenylphosphate, commonly called paraoxon – quickly and efficiently. And instead of waste pollutants, the device has a useable output, namely the active pharmaceutical ingredient paracetamol. 'Allowing us to recycle the generated waste during the process is the main goal of this work – to convert waste to a value-added product,' says Yim.

The Teflon microreactor operates according to a flow process rather than a batch process, allowing paraoxon to be seamlessly fed through the system. Reagents are added while the reaction undergoes three stages: degradation of paraoxon to *p*-nitrophenol, reduction to *p*-aminophenol, and finally the synthesis of paracetamol through acetylation. The reactor is portable, being the size of a suitcase, and can neutralise 700 grams of paraoxon a day.



Scientists have devised a one-flow process for neutralising paraoxon to make paracetamol in a microreactor **Source: © Dong-Pyo Kim/Pohang University of Science and Technology**

'It's a very efficient process, with the highest space–time yield,' remarks Julien Legros, whose group at the University of Rouen Normandy, France, also develops flow reactors to neutralise chemical warfare agents. But he notes that paracetamol as an end product is very specific to paraoxon as a starting ingredient, and paraoxon in turn is of limited relevance to chemical warfare. 'Although paraoxon can be used as a chemical warfare nerve agent simulant, in practice its main use is as a pesticide and it is not intended for military-grade weapons,' he says.

Yim, who used paraoxon in the study due to its relative safety and availability for laboratory use, agrees that it remains a challenge to

produce value-added products from more potent chemical warfare agents. 'Soman, sarin and tabun generate fluoride and cyanide ions upon hydrolysis, and are not suitable for making active pharmaceutical ingredients,' he says.

Legros adds, 'It's still nice that you can make something peaceful and synthesise an active pharmaceutical ingredient from a toxic pesticide.'

References *B M Sharma et al, React. Chem. Eng., 2021, DOI: 10.1039/d1re00147g*

https://bit.ly/2SLVVoH

These ancient weights helped create Europe's first free market more than 3000 years ago

Mesopotamian merchants established a standardized system of weights that spread across Europe

By Andrew Curry

Merchants of the Bronze Age faced the same problem as merchants from London to Lisbon today: how to know you're getting what you pay for in a transaction. It usually takes a ruling authority, like a king, pharaoh, or perhaps the European Union, to establish standard weights, which amount to a unit of value in the age before coins and bills.

These spool-shaped weights from Tiryns, in Bronze Age Greece, weighed about the same as their counterparts in other parts of Europe and the Middle East. **Ialongo et al.**

A new study suggests merchants in Bronze Age Europe were an exception: Through informal networks, Mesopotamian merchants established a standardized system of weights that later spread across Europe, enabling trade across the continent. The advance effectively formed the first known common Eurasian market more than 3000 years ago.

"This is quite a blow to the idea that elites or a central authority is running the show," says Leiden University archaeologist Maikel Kuijpers, who was not involved with the work. "The [researchers] make a really good case."

Standard weights—used by merchants to trade goods of equivalent value—were invented in Egypt or Mesopotamia 5000 years ago. By 3000 years ago, they had spread across Europe, where some graves included pouches or boxes containing bone balance beams, tweezers for picking up scraps of gold or silver, and stone weights.

For more than 100 years, historians have assumed that weight standards were handed down from on high, first created by a king or religious authority to collect taxes or tribute, then later adopted by merchants. The first artifacts to clearly be weights, for example, were found in the highly stratified civilizations of ancient Mesopotamia and Egypt. But Bronze Age Europe boasted few such states when weights proliferated.

To find out whether standardization without centralization was possible, Georg August University of Göttingen archaeologists Lorenz Rahmstorf and Nicola Ialongo spent nearly 10 years visiting museum collections and weighing stones and other objects they thought might have been used for commerce. They analyzed weights from previously excavated sites spanning nearly 3000 years in Europe, Anatolia, and Mesopotamia.

To their surprise, more than 2000 such objects crafted over the course of 2000 years and an area spanning nearly 5000 kilometers weighed nearly the same amount—between 8 and 10.5 grams from Great Britain to Mesopotamia. Over the time spans involved, the consistency was remarkable, they report today in the *Proceedings of the National Academy of Sciences*. "It is like we were still using the Roman systems of measurement [today], with just some minor variations," Ialongo says.

In Mesopotamia, that unit was referred to as a shekel. "Weight

systems in Europe were only slightly different from weight systems in Anatolia, which were only slightly different than in Mesopotamia," Ialongo says.

The researchers suggest that in all these areas it was merchants who kept the weights standard, because it was in their interest to do so. Each time traders met, the archaeologists write, they would bring out their own scales and weights and compare them—or introduce them to new traders. With enough time and contacts, a standard system emerged—laying the groundwork for the equivalent of an integrated market from Great Britain to Babylon. "The weight units were regulated by the market," Ialongo says.

To test its model, the team came up with a unique experiment. Using replica Bronze Age bone balance scales, co-author and Göttingen archaeologist Raphael Hermann carved 100 weights out of stone. Each new weight was modeled randomly from the weights already produced: Weight two was based on weight one, but weight three could be modeled on either weight one or weight two, weight 10 could be modeled from any of the previous nine, and so on.

Human error, combined with the slight imprecision of the ancient balance, led to deviations up to 25 grams from the original 153-gram weight. But the drift tended to stay within 5%, still within a range that would have been acceptable in an ancient marketplace, Rahmstorf says. In a system where all the weights were copied from a central standard under palace supervision, the deviations would have been much smaller.

When the researchers plotted their own weights on a graph, the pattern matched the distribution of the ancient samples they had found.

The research helps explain how far-flung Bronze Age societies traded across long distances, says Johannes Gutenberg University of Mainz archaeologist Christopher Pare, who was not involved in the research. "Complex systems are perpetuated by convention and exchange, rather than a central authority. … It's fascinating."

In a related study, published last month in the *Journal of Archaeological Science*, Ialongo and colleagues found nearly 3000 bronze fragments from the same time period in hoards in central Germany and Italy that were all multiples of the same 10-gram weight. That suggests people in both regions were using hacked-up bronze in standard amounts as an early form of currency, Ialongo says.

However, Pare and others caution that it's tricky to apply modern economic concepts to the distant past. Pare notes that when 19th century archaeologists applied their concepts of how societies were organized to the question of weights, they concluded a king must be in charge. The idea of the market standardizing itself "fits a little too well into our modern neoliberal discourse," he says. "Should we really be using these terms to talk about societies which are so foreign to us?"

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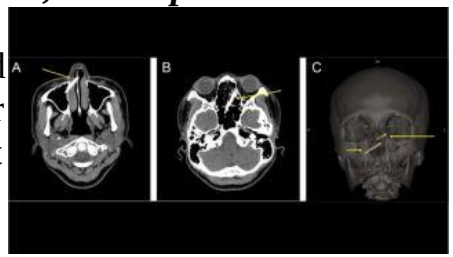
Woman unknowingly had chopsticks embedded in her sinuses for a week

The utensils became lodged in her sinuses after she was "attacked by her sister at the dinner table," the report said.

By Rachael Rettner - Senior Writer

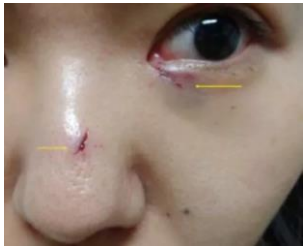


A woman in Taiwan unknowingly had two chopstick fragments lodged in her sinuses for a week after a violent fight with her sister, according to a new report.

A CT scan showing chopstick fragments penetrating the woman's sinuses (A,B). A 3D reconstruction of the woman's skull showing the positions of the chopstick pieces (C). (Image credit: Reprinted with permission of Elsevier (2021).)

The 29-year-old woman went to the emergency room after she was

"attacked by her sister with plastic-wood chopsticks while at the dinner table," according to the report, published June 24 in The Journal of Emergency Medicine. The woman said she had experienced a mild nosebleed and swelling in her left eye after the attack. Doctors saw that she had two small cuts under her eye and on her nose. But an X-ray did not show anything unusual.



After the woman was attacked by her sister, doctors observed two small wounds under her eye and on her nose. **(Image credit: Reprinted with permission of Elsevier (2021).)**

However, one week later, the woman began to suspect that her injury was more serious than it appeared. She noticed that "some parts of the chopsticks used in the attack were missing," according to the report authors, from Hualien Tzu Chi Hospital in Taiwan. And when she looked in the mirror, she thought she could see a gray object in her nose.

A doctor then examined the inside of her nose and saw pieces of chopstick penetrating her nasal septum, or the wall dividing the two nasal passages, the report said. A CT scan showed two chopstick pieces in her sinuses, with one embedded more deeply than the other.

The chopstick fragments after they were removed from the woman's sinuses. **(Image credit: Reprinted with permission of Elsevier (2021).)**

The route that the chopsticks followed to enter the woman's skull was the same as the route doctors use when performing surgery on the ethmoid sinuses — which are located between the corner of your eye and the bridge of your nose — to treat sinus infections.

The woman needed surgery to remove the fragments, which were about 1.4 inches (3.5 centimeters) and 2 inches (5 cm) long, respectively, according to the report. She experienced no surgical complications.

Emergency room doctors should be aware that foreign bodies entering the skull the way these chopsticks did "could present only as tiny laceration wounds and may be asymptomatic," the authors wrote. If doctors suspect a foreign body lodged near the nose, it's important they perform an examination of the ears, nose and throat, as well as a CT scan to identify it as soon as possible, they said.

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Tuskegee Relatives Promote COVID-19 Vaccines in Ad Campaign

New ad campaign launched with relatives of men who unwittingly became part of the infamous experiment wants to change minds.

Lindsey Tanner, AP Medical Writer

Tuskegee is the one-word answer some people give as a reason they're avoiding COVID-19 vaccines. A new ad campaign launched Wednesday with relatives of men who unwittingly became part of the infamous experiment wants to change minds.

Omar Neal, 63, a former mayor of the Alabama town, said he was hesitant at first about the shots. Neal is a nephew of Freddie Lee Tyson, a family man who was among several hundred Black men who decades ago became involved without their consent in the federally backed syphilis study.

Neal said he agreed to appear in the national campaign after doing research to gain confidence in the vaccines. "I want to save lives," Neal told The Associated Press. "I didn't want people to use Tuskegee and what transpired there as a reason for not taking the vaccine."

In 1932 and over 40 years, Black men in Tuskegee, Alabama, were subjected to experimentation without their knowledge. Most of the 600 men had syphilis — including Tyson, who got infected before birth — but they were left untreated so researchers could study the natural history of the disease.

Tyson died from unrelated causes in 1988, 16 years after the study

ended. But many others died from a disease that can be cured with penicillin.

Neal and other Tyson relatives are among half a dozen Tuskegee descendants involved in the ads, which focus on vaccine hesitancy among Black Americans. They say vaccination is needed to help communities of color and curb a disease that has disproportionately affected Black Americans. "Don't deny ourselves the opportunity the men were denied," Tyson's 76-year-old daughter, Lillie Tyson Head, said in one of the ads.

"It's really up to us to take ownership of our health and this story," Carmen Head Thornton, the granddaughter Tyson called his "'Çarmen girl," said in another ad.

Vaccines are highly effective against COVID-19. Yet U.S. vaccination rates are lower than government goals, with 46% fully vaccinated while 54%, have received one dose. People of color have lagged behind white Americans in getting the shots.

Authorities are concerned about the slowing pace of new vaccinations amid persistent pockets of resistance. Limited access is an issue for some Black people, but so is mistrust of the medical system.

Thornton, a director at the American Academy of Child & Adolescent Psychiatry, was a young girl when she learned what had happened to her grandfather. The two were extremely close; she recalls catching her first fish with him and watching mesmerized while he stitched quilts by hand. She pledged to devote her life to fighting health inequities and injustice, and sees COVID-19 vaccines as a way to address disparities the pandemic laid bare.

The campaign includes a minidocumentary and shorter 60-second versions made for TV and online use. They are part of the Ad Council's ongoing multimillion-dollar education campaign aiming to encourage confidence in the shots, paid for by donations from media corporations.

https://bit.ly/368Ad1h

Earliest known strain of plague could have come from a beaver bite

The disease may be 2000 years older than we thought.

By Ben Turner - Staff Writer

Scientists have found the earliest known strain of plague in the remains of a 5000-year-old hunter gatherer.

The "astonishing" discovery pushes back the first appearance of the plague bacterium (*Yersina Pestis*) by more than 2,000 years, study senior author Ben Krause-Kyora, a biochemist and archaeologist at the University of Kiel in Germany said in a statement. This date is probably close to when the bacteria first evolved, he added.

The plague-carrying hunter-gatherer, dubbed "RV 2039", was a 20- to 30-year-old man and one of four people whose remains were excavated from a burial site near the Baltic Sea in Latvia. An analysis of samples from the man's teeth and bones revealed that he was likely the only one among those buried with the disease. Researchers reconstructed the bacteria's genome using genome sequencing, and believe the bacteria was likely a part of a lineage that emerged roughly 7,000 years ago, not long after *Yersina Pestis* split from a predecessor, *Yersina pseudotuberculosis.*

The analysis also revealed that most of the deadly disease's key genes were already in place, even at this early stage of its history. "What's so surprising is that we see already in this early strain more or less the complete genetic set of *Y. pestis*, and only a few genes are lacking. But even a small shift in genetic settings can have a dramatic influence on virulence," Krause-Kyora said.

Modern plague variants contain one important thing that the newly-discovered ancient strain lacked — a gene enabling fleas to carry the disease. This adaptation hugely increased the rate at which the plague bacteria could infect human hosts, entering the body and travelling to the lymph nodes where it would rapidly replicate. The

host would then form painful, pus-filled buboes — from which the bubonic plague gets its name — on their skin.

But the switch to fleas as a means of transmission required the disease to kill its host: an old host's death encourages fleas to move to a new host and pass on the disease. The researchers speculate that this new gene was responsible for driving the plague to become deadlier.

Because this early strain of Y. pestis was not yet flea-borne, the scientists think that the bacteria originally entered the hunter-gatherer's body through a rodent bite, possibly from a beaver, a common carrier of the plague predecessor *Y. pseudotuberculosis* and the species with the most remains recorded at the site. Once there, the course of the disease was fairly slow, with bacteria slowly accumulating in high quantities in the man's bloodstream until he died.

The three pandemics the bacteria would go on to cause are among the deadliest biological events in human history. The first pandemic, the Justinian Plague (which occurred roughly between A.D. 542 and 750), may have caused the Mediterranean population to decline by 40% by the end of the sixth century. The second, and most infamous, pandemic caused by the disease was the 14th century European Black Death, which killed approximately 25 million people — between 33 to 50% of Europe's population. A third, lesser known, pandemic began in 1855 in China's Yunnan province and killed more than 12 million people in India and China alone.

The people buried around RV 2039 were not infected and he was carefully placed in his grave, two indications that he didn't carry the later, highly-contagious version of the disease. But because of its presence in his blood, scientists still think the plague bacteria could have killed him.

The idea that this ancient bacteria replicated slowly and was passed from rodent to human is bolstered by the fact that scientists have found other ancient skeletons infected with *Y. pestis* at other sites, where people lived very different lifestyles. "Isolated cases of transmission from animals to people could explain the different social environments where these ancient diseased humans are discovered. We see it in societies that are herders in the steppe, hunter-gatherers who are fishing, and in farmer communities — totally different social settings but always spontaneous occurrences of *Y. pestis* cases," Krause-Kyora said.

The picture of the early plague as a slow-acting, less virulent disease raises serious challenges to theories about the development of civilization in Europe and Asia.

One of these theories is that the plague was the cause of large declines in Western European populations towards the end of the Neolithic Age. In 2019, a tomb in modern-day Sweden containing 78 hastily buried bodies was dated to roughly the same period as RV 2039, and one set of bones and teeth, belonging to a woman also contained plague bacteria fragments, Live Science previously reported.

In fact, remains containing traces of plague bacteria have been found in sites all across Eurasia, and dated to coincide with the rapid decline in Neolithic populations between five and six thousand years ago.

Another theory is that the plague evolved in European "mega settlements" containing 10,000 to 20,000 people which existed between 6,100 and 5,400 years ago. But the new research suggests *Y. Pestis* could have split from *Y. pseudotuberculosis* as far back as 7400 years ago, a time when European populations had yet to grow beyond collections of sparse settlements.

The mystery of this population collapse, and whether it was caused by an early form of plague, has yet to be fully unravelled. The researchers believe that their work could open further investigation into the history of plague, offering valuable insights not just on the

evolution of the disease, but on early human history and genomics.

"Different pathogens and the human genome have always evolved together. We know Y. pestis most likely killed half of the European population in a short time frame, so it should have a big impact on the human genome," Krause-Kyora said. "But even before that, we see major turnover in our immune genes at the end of the Neolithic Age, and it could be that we were seeing a significant change in the pathogen landscape at that time as well."

Their findings were published June 29 in the journal Cell Reports.

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How Protected Are You With Just One Dose of a COVID-19 Vaccine? Here Are Some Stats

More than 179 million Americans and more than 44 million Britons have received their first dose of a two-shot COVID-19 vaccine.

Catherine Schuster-Bruce, Business Insider

The US has authorized vaccines from Moderna and Pfizer-BioNTech, while the UK has authorized Pfizer's shot as well as one made by AstraZeneca and Oxford University. Both countries have authorized Johnson & Johnson's vaccine, which is a single dose.

The UK is delaying the second dose of the vaccines for up to 12 weeks for most people to prioritize giving people their first shot because of an initial shortage of vaccines. In the US, the Centers for Disease Control and Prevention has recommended giving second doses of Pfizer's vaccine 21 days after the first, and 28 days after the first for Moderna, with an interval of up to six weeks in "unavoidable" situations.

The data for how well the vaccines work after one dose isn't clear cut – it depends on what you're measuring, and when you're measuring it. Stephen Evans, a professor of medical statistics at the London School of Hygiene & Tropical Medicine and a former drug-safety committee member at the European Medicines Agency, helped Insider break down the data.

Evans said the Food and Drug Administration presentation of the data from late-stage trials of each vaccine was generally the best data available. This is how much protection one shot of each vaccine gives you, based on that data.

Pfizer-BioNTech: at least 80 percent

Pfizer's shot was 52.4 percent effective at protecting against COVID-19 with symptoms between the first and second dose, according to the FDA documents. But the 52.4 percent figure includes the 11 days before protection kicks in after the first dose, so the real percentage could well be higher.

The true value lies between 29.5 percent and 84.5 percent, according to the FDA documents. There was a wide range because not many people caught COVID-19 in the trial during this time period.

Pfizer's shot was 100 percent effective at protecting against hospitalization and death. This was based on a small number though – only four people got severe COVID-19 in the trial after receiving placebo rather than the vaccine.

Evans said there was "pretty clear evidence" that you get at least 80 percent protection – and "probably" better than 90 percent – for Pfizer's vaccine against COVID-19 with symptoms after a single dose. He said you couldn't be absolutely sure what happens after 21 days because it hadn't been fully tested.

Evans said this was based on his overall reading of the trial data used by the FDA in their briefing document before authorization.

Moderna: at least 80 percent

Moderna's vaccine was 69.5 percent effective at preventing COVID-19 with symptoms between the first and second dose, with a true value between 43.5 percent and 84.5 percent. There was a fairly wide range because the number of people that caught

COVID-19 in the trial during this time period was low.

The 69.5 percent figure includes the 13 days before protection starts, so the real percentage could be higher.

There were a small number of people in Moderna's trial – about 7 percent – that didn't get their second dose for unknown reasons. In this group, the shot was 50.8 percent effective at preventing COVID-19 with symptoms for up to 14 days after the first dose and 92.1 percent effective after 14 days.

It is unclear how well one shot of the vaccine protects against hospitalization and death because not many people got severe COVID-19 – two in the vaccine group and four in placebo.

Evans said that you get at least 80 percent protection – and probably better than 90 percent – for Moderna's vaccine against COVID-19 with symptoms after a single dose for 28 days. After 28 days it was unclear because it hadn't been tested. Again, this was based on his overall reading of the FDA data, he said.

AstraZeneca: more than 70 percent

Evans said it was harder to ascertain a figure for AstraZeneca's vaccine because late-stage trials used differing study designs, and a large US study was ongoing. The FDA also has not yet presented the data for the shot in the same way it has done for other vaccines.

A single dose of AstraZeneca's shot was 76 percent effective at protecting against COVID-19 with symptoms for at least 90 days, according to late-stage-trial data published in The Lancet on February 19. The study authors also reported that one dose provided 100 percent protection against hospitalization, but the numbers were small.

Based on his reading of existing studies, Evans said the single-dose efficacy for AstraZeneca's vaccine was probably at least 70 percent against COVID-19 with symptoms for the first 90 days. After this time period, it's unclear, he said.

Johnson & Johnson: 66 percent

J&J looked at protection against moderate to severe COVID-19 in trials, rather than symptomatic COVID-19, like Pfizer, Moderna, and AstraZeneca.

Protection kicked in at 14 days and was 66.1 percent effective at 28 days. The vaccine's efficacy varied depending on the country it was used in – it was 72 percent effective in the US but 64 percent and 68 percent effective in South Africa and Brazil, respectively. These countries both have coronavirus variants circulating that could partially evade antibodies.

What percentage efficacy means

Percentage efficacy for vaccines refers to the proportion of people that get full protection after a vaccine. With 80 percent efficacy, 80 percent of people have full protection, and 20 percent don't.

For those who get full protection the first time around, the second shot improves the quality of the immune response and its durability.

For the people who don't get full protection with the first shot, some will get full protection after the second dose. Some people won't ever get full protection from a vaccine because their immune system doesn't respond at all.

The latest real-world data: One shot significantly reduces infections and transmission

- A UK study found Pfizer or AstraZeneca's vaccine cut COVID-19 infections with symptoms by 72 percent after one dose, and protection probably held up for 10 weeks. Protection from Pfizer's vaccine rose to 90 percent after two doses. The study hasn't been peer-reviewed.
- A US study of essential workers found that a single dose of Pfizer of Moderna's COVID-19 vaccines were 80 percent effective against all coronavirus infections from 14 days.
- A Scottish study found that a single dose of Pfizer's vaccine was 91 percent effective against hospitalization at 28 to 34 days following vaccination. One dose of AstraZeneca's

vaccine was 88 percent effective against hospital admissions after the same time period.

- A UK study found that a single dose of either Pfizer or AstraZeneca's vaccine cut spread of symptomatic COVID-19 within a household by up to 50 percent.
- A South Korean study found one dose of Pfizer's vaccine was 89.7 percent effective at preventing COVID-19 in South Koreans aged over 60, at least two weeks after vaccination. AstraZeneca's vaccine was 86 percent effective at preventing COVID-19 after one dose. The severity of illness that the shots protected against was unclear – generally they're more effective at preventing COVID-19 infections that caused hospitalization or death.
- An English study found that a single dose of either Pfizer or AstraZeneca's vaccine was about 80 percent effective at preventing hospitalization in people over 70-years-old. Protection lasted for at least 6 weeks, including against the Alpha variant first identified in the UK.
- An Israel study showed that Pfizer's vaccine was 54 percent effective against symptomatic COVID-19, from 13 days to 24 days after vaccination, a figure comparable to the late stage trial data presented to the FDA.
- A UK study estimated that a single dose of either Pfizer or AstraZeneca's vaccine was between 56 percent and 62 percent effective at preventing COVID-19 infection caused by the Alpha variant in people over 75 years-old, four to seven weeks after the first dose. The severity of illness that the shots protected against was unclear, but probably included asymptomatic infections.
- A UK study estimated that one dose of Pfizer vaccine was 79.3 percent effective at reducing the risk of hospitalization from COVID-19 in people aged over 80. A single shot of AstraZeneca's was 80.4 percent effective, the researchers said.

Newest data suggests second shot provides better protection against variants

Real-word data from the UK posted May 23 by Public Health England showed that Pfizer's and AstraZeneca's COVID-19 vaccines worked better against the variants when two doses were given rather than just one. Both vaccines were 30 percent effective against COVID-19 with symptoms caused by the Delta variant, first identified in India, three weeks after the first dose.

This was boosted to between 60 percent and 88 percent effectiveness two weeks after the second dose. The two vaccines were 50 percent effective against COVID-19 with symptoms against the variant first found in the UK, Alpha, three weeks after the first dose. This increased to between 66 percent and 93 percent two weeks after the second dose.

Dr. Anthony Fauci, President Joe Biden's chief medical advisor, said on June 8 that getting two doses of COVID-19 vaccines would stop the Delta variant from spreading across the US.

In the UK, Professor Deborah Dunn-Walters, chair of the British Society for Immunology COVID-19 Taskforce, said in a statement on June 4 that two doses of Pfizer's vaccine were "critical for protection" against emerging strains of the virus.

https://go.nature.com/2Tseu1C

Vaccine made of live malaria parasites shows early success

Strategy uses a combination of parasites and medicines to generate immunity while avoiding symptoms.

Heidi Ledford

An experimental malaria vaccine that contains live parasites protected nearly all recipients from infection in a small clinical trial. Participants in the study, published on 30 June in *Nature*[1], were given a shot containing live *Plasmodium falciparum* parasites,

along with drugs to kill any parasites that reached the liver or bloodstream, where they can cause malaria symptoms. Participants were then intentionally infected with malaria three months later to test the vaccine's efficacy.

The vaccination protected 87.5% of participants who were infected after three months with the same strain of parasite that was used in the inoculation, and 77.8% of those who were infected with a different strain. This is a significant improvement on earlier efforts to use live parasites in a malaria vaccine, which did not perform as well against different strains.

The study also yielded important information about how immunity against malaria can be achieved, says Pedro Alonso, director of the World Health Organization's Global Malaria Programme in Geneva, Switzerland. "It contributes considerably to the science of vaccines," he says. "I cannot overstate how important this is, because the field of malaria vaccines has been a neglected one for a long time."

Proteins and parasites

Several malaria vaccines are in development. The most advanced — RTS,S — has been given to more than 650,000 children as part of a pilot programme in three African countries to assess its safety and efficacy, as well as the logistics of rolling it out.

Another vaccine, called R21, was recently shown to be up to 77% effective in a trial of 450 young children, and a larger study is under way.

Both of these use the same malaria protein, called circumsporozoite protein, to trigger immune responses. That protein decorates the outside of the sporozoite form of the parasite, the stage in its life cycle when it first enters the human body from the salivary glands of infected mosquitoes.

For decades, researchers have tried to find ways to use whole sporozoites as vaccines: many vaccines against viruses, for example, have used weakened whole viruses, and the approach offers the immune system many targets, rather than just one protein. But for malaria, success has been limited. One study using sporozoites that had been attenuated using radiation found 20% efficacy when vaccine recipients were challenged with a different strain of the parasite[2].

Some scientists have reasoned that a live parasite might yield stronger immune responses because it can replicate inside the body, producing more foreign proteins in the process. Sara Healy and Patrick Duffy, both working on malaria vaccines at the US National Institute of Allergy and Infectious Diseases in Bethesda, Maryland, have been looking for ways to boost the efficiency of this approach.

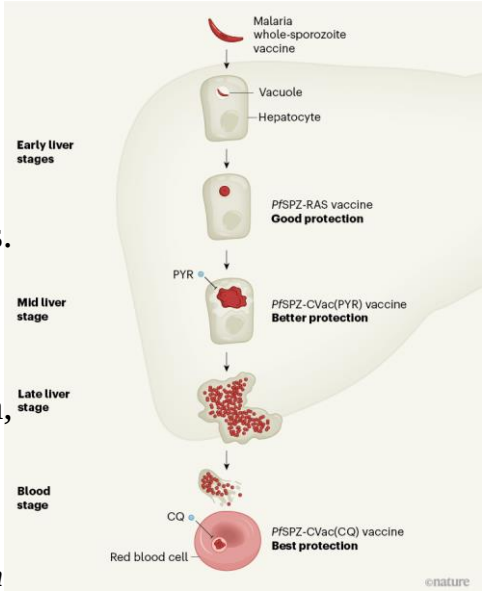

Figure 1 | Vaccine strategies. ***Attention is turning towards malaria-vaccination approaches that use whole Plasmodium falciparum (Pf) parasites, which are the disease-causing agent. After the parasite enters the bloodstream in a form called a sporozoite, it reaches the liver, where it resides inside a vacuole in cells called hepatocytes. The parasite develops in the liver and then returns to the bloodstream to infect red blood cells and cause illness. Previous work[8,9] indicates that vaccination using a weakened form of the parasite (PfSPZ-RAS), which cannot develop in the liver, offers some protection against malaria. Mwakingwe-Omari et al.[3] report a clinical trial of vaccines containing whole, live parasites given with drug treatments to kill the parasite at a particular developmental stage. The PfSPZ-CVac(PYR) vaccine, which harnesses the drug pyrimethamine (PYR), offered improved protection against malaria compared with that reported[8,9] for PfSPZ-RAS vaccines. The best vaccine results were for the PfSPZ-CVac(CQ) vaccine, which requires the drug chloroquine (CQ).***

In the latest trial, which enrolled 42 people, the researchers tested injected participants with live sporozoites. But they also treated the vaccine recipients with medicines to kill the parasite if it reached the liver or blood, where it would normally go on to infect blood cells and cause disease symptoms. This approach worked well, not only against the same strain of parasite as was used in the vaccination, but also against another strain, found in South America. A field study testing the vaccine in adults is under way in Mali.

Challenging scale-up

The results of the trial are promising, but producing whole-sporozoite vaccines on the scale needed to combat malaria would present a challenge. The sporozoites must be harvested from mosquito salivary glands and then stored at extremely low temperatures, complicating their distribution in resource-poor areas. No vaccine has ever been mass-produced using mosquitoes before. Alonso remembers someone asking him about the possibility years ago: "It is a crazy idea," he replied at the time.

But Sanaria, a biotechnology company in Rockville, Maryland, has been working to make sporozoite vaccines more practical. The company, which collaborated on the vaccine study, can now make large quantities of sporozoites and is working on ways to do so without mosquitoes. "It's amazing what has been accomplished in terms of the science and technologies that have been developed along the way," says Alonso.

Sanaria is also working with researchers to use gene-editing techniques such as CRISPR–Cas9 to genetically weaken the parasite, so that it can be injected live without the need for additional medicines. An edited sporozoite might replicate a few times and then die, before it has a chance to cause symptoms.

Ultimately, several vaccine options will probably be needed in the fight against malaria, says Duffy. "There's probably not going to be a single vaccine solution," he says.

Malaria control measures, including mosquito nets and preventive medicines, have saved more than 7 million lives and prevented 1.5 billion malaria cases in the past decade, Alonso says. But these measures are being challenged by insecticide resistance, drug resistance and population growth. Each year, 400,000 people die from malaria — most of them children.

"We've plateaued at an unacceptably high level," says Alonso. "Unless we have a highly efficacious malaria vaccine, it's going to be very hard to keep making progress."

doi: https://doi.org/10.1038/d41586-021-01806-1

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https://bit.ly/3wip74n

Pfizer and Moderna vaccines may provide years of protection from COVID-19

A small study suggests that mRNA vaccines may offer long-term protection as long as the virus doesn't evolve significantly.

By Yasemin Saplakoglu - Staff Writer

The Pfizer-BioNtech and Moderna COVID-19 vaccines will likely provide protection against the coronavirus for years if it doesn't evolve significantly, a small new study suggests.

As a massive vaccination effort continues to play out across the globe, there is still a question about how protective COVID-19 vaccines will be in the long term and whether booster shots will be necessary. Some vaccines for other viruses, such as influenza, provide only fleeting protection and need to be renewed every year, but others — such as the MMR vaccine for measles, mumps and rubella — confer lifelong protection.

The level of protection depends on how much and how quickly the virus evolves, as well as on how robust different types of vaccines

are in spurring a lasting immune response. The Pfizer-BioNTech and Moderna vaccines both use a relatively novel platform known as messenger RNA (mRNA) to train the immune system to fight SARS-CoV-2, the virus that causes COVID-19, Live Science previously reported.

While mRNA vaccines have greatly exceeded experts' expectations and have shown high efficacy in protecting people from SARS-CoV-2, including its currently circulating variants, how long this protection will last hasn't been clear.

To figure this out, a group of researchers recruited 41 participants who received two doses of the Pfizer-BioNTech vaccine; eight had previously been infected with SARS-CoV-2. The researchers collected blood samples at the start of the study and then three, four, five, seven and 15 weeks after the participants received their first dose of the vaccine.

Consistent with previous studies, the researchers found that the mRNA vaccine induced strong antibody responses and that those responses were even stronger in people who had recovered from a mild SARS-CoV-2 infection prior to being vaccinated.

The team also collected lymph node samples across this same time span from 14 people, none of whom had previously been infected with SARS-CoV-2. In response to infections and vaccinations, fleeting molecular structures known as "germinal centers" form inside the lymph nodes, the glands that hold immune system cells and typically swell in response to an infection.

In people who are infected with SARS-CoV-2, these structures form in the lymph nodes of the lungs, which are difficult to access, whereas vaccines typically spur their production in the armpits, which is more easily accessible.

"You can think of them as our boot camps for the immune cells," said senior author Ali Ellebedy, an immunologist at the Washington University School of Medicine in St. Louis. The structures train a type of immune cell known as B cells over weeks and months to bind better to a pathogen — in this case, SARS-CoV-2.

The process creates highly trained immune cells, some of which are memory cells that will remember the virus in the long-term.

Not much is known about how long these "boot camps" last inside the lymph nodes in humans; animal studies have shown that they typically last only a few weeks, Ellebedy said.

But in the new study, Ellebedy and his team found something surprising: In most of the participants who received the vaccine, their germinal centers continued to be active, training these robust immune cells for at least 15 weeks after the first dose.

'Very promising' protection

Because this germinal-center response lasted for months, it likely produced many memory cells that will last for years; and some of these memory cells will likely establish themselves inside bone marrow and produce lifelong antibodies, Ellebedy told Live Science. That's "very promising" but doesn't necessarily mean people won't need booster shots, he said.

Rather, the need for booster shots will depend on how much the virus evolves and whether the cells produced by the germinal centers are robust enough to handle significantly different variants, he added. In addition, not everyone generates the same robust immune response; some people, such as those with suppressed immune systems, will likely need booster shots, he said.

"This study, like others before it, confirms that the vaccines are eliciting the appropriate reaction from the immune system and that durable immunity is being created," said Dr. Amesh Adalja, an infectious-diseases specialist and a senior scholar at the Johns Hopkins Center for Health Security in Baltimore.

Adalja, who was not involved in the new study, agrees that it's too soon to discuss whether we will need booster shots. "If a large proportion of the fully vaccinated are contracting breakthrough

infections that land them in the hospital, that is the threshold for booster vaccinations," he told Live Science in an email.

Still, this is the first study to provide direct evidence that the germinal-center response is persistent in humans after vaccination. Although the authors didn't look at people who had received the Moderna vaccine, they think the response will likely be similar, because it's also an mRNA vaccine that showed a comparable efficacy, Ellebedy said. However, more research will be needed to see the duration of the germinal-center response from the Johnson & Johnson vaccine, because it uses a different platform (rather than mRNA), he said.

Now, Ellebedy and his team hope to continue monitoring these cells to see whether they migrate and settle permanently in bone marrow. In other words, it's still unclear whether these immune cells will "become our life partners, basically helping us for the rest of our lives" or if we will eventually need booster vaccines to make some better fighters.

The findings were published online June 28 in the journal Nature.

https://bit.ly/3AqCDWS

COVID-19 Makes Lasting Changes to Blood Cells, Which Might Explain a Lot

Why does long COVID last for so long, leaving long-haulers with symptoms that persist for months after initial infection?

Peter Dockrill

New evidence suggests the enduring imprint of COVID-19 could be due to the virus making significant alterations to people's blood – yielding lasting changes to blood cells that are still evident several months after infection is diagnosed.

"We were able to detect clear and long-lasting changes in the cells – both during an acute infection and even afterwards," explains biophysicist Jochen Guck from the Max Planck Institute for the Science of Light in Germany.

In a new study, Guck and fellow researchers analyzed patients' blood using a system developed in-house, called real-time deformability cytometry (RT-DC), which is capable of rapidly analyzing hundreds of blood cells per second, detecting if they exhibit abnormal changes in their size and structure.

The technology is relatively recent, but it could go a long way in exploring what remains a significant unknown in COVID-19 science: how the coronavirus may impact blood at the cellular level.

"While the pathology is not yet fully understood, hyper-inflammatory response and coagulation disorders leading to congestions of microvessels are considered to be key drivers of the still increasing death toll," the researchers, led by first author Markéta Kubánková, write in their paper.

"Until now, physical changes of blood cells have not been considered to play a role in COVID-19 related vascular occlusion and organ damage."

In the study, the researchers analyzed blood from 55 individuals: 17 patients with severe COVID-19 (half of whom later sadly died), 14 recovered patients, and 24 healthy volunteers who showed no sign of having had the disease.

In total, over 4 million blood cells taken from these people were run through the RT-DC system, being microscopically analyzed as they flowed through a narrow channel in the device.

The results showed that red blood cells (erythrocytes) in COVID-19 patients varied more in size than those from healthy people, and showed signs of stiffness in their physical structure, exhibiting less deformability, which could affect their ability to deliver oxygen through the body.

"The physical properties of erythrocytes are crucial for microcirculatory flow and as such, these changes could impair circulation and promote hypoxemia," the researchers explain.

"The effect could persist in COVID-19 patients long after the

infection is not active anymore; we found that in recovered patients phenotype alterations were not as prominent, but still present."

In contrast, the researchers discovered that a form of white blood cells (leukocytes) called lymphocytes showed decreased stiffness in COVID-19 patients, while other white blood cells, known as monocytes, were significantly larger than in cells from the control group.

Meanwhile, neutrophils – another type of white blood cell – showed numerous changes in COVID-19 patients, seen in higher volume, with greater deformation.

Interestingly, neutrophils have a particularly short lifespan (of only about one day), but the neutrophil changes in COVID-19 patients could still be seen months after infection, a result Kubánková describes as "totally unexpected" – and yet more evidence of COVID-19 infection likely leaving a lasting influence on the immune system.

"While some of these changes recovered to normal values after hospitalization, others persisted for months after hospital discharge, evidencing the long-term imprint of COVID-19 on the body," the researchers write.

"We hypothesize that the observed changes could arise due to cytoskeletal alterations of immune cells. Mechanical properties of cells can be directly related to the cytoskeleton, an important supportive structure which also determines cellular function."

It remains to be seen how these blood cell changes may ultimately be triggered by viral infection, and it's not yet fully known how the cell alterations lead to COVID-19 symptoms, and sometimes to death.

For now, it's just more evidence for how deeply this virus invades our bodies – and why it sometimes won't let people go.

"The persistent alterations of erythrocytes and neutrophils could be connected with long term symptoms of the recovered patients, of which 70 percent described chronic headache or neurological symptoms, 54 percent had concentration disorders and 62 percent circulatory problems like cold sweat and tachycardia," the authors write.

"We hypothesize that the persisting changes of blood cell physical phenotypes could contribute to the long-term impairment of circulation and oxygen delivery linked with COVID-19."

The findings are reported in *Biophysical Journal*.

https://bit.ly/3xqpfCA

A Mouse Embryo With a Beating Heart Has Been Grown Entirely in a Petri Dish

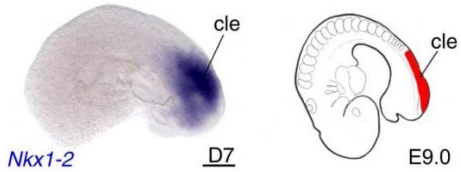
The most complex life forms ever developed entirely in Petri dishes can pump blood through tiny beating hearts, gradually growing nerves and muscles in a laboratory.

Tessa Koumoundouros

These little collections of mammalian cells form rudimentary mouse embryos, built from scratch out of stem cells - cells that have the potential to develop into any other cell type in the body.

While scientists have successfully been creating synthetic organs called organoids for a while now, these lack the full variety of cell types found in the real deal. This human-built mouse embryoid is a whole lot more intricate.

"Watching an embryo develop is a marvelous thing to behold," said developmental biologist Christine Thisse from the University of Virginia, one of the authors of the study.



Gene Nkx1-2 expression in a mouse embryoid (l); mouse embryo diagram.
(Xu et al., Nat Comm, 2021)

"What is amazing is that we can get the variety of tissues that are present in an authentic mouse embryo. [This] model shows that we

are able to induce cells to execute complex developmental programs in the right succession of steps."

The embryoid isn't a complete unborn mouse, and it can't fully develop into one as key parts are still missing - like a giant chunk of the brain. But the complexity of this experiment takes researchers a huge step towards being able to build fully functional organs in a lab.

"Human organs are made of multiple cell types that originate from different parts of the growing embryo," said developmental biologist Bernard Thisse. "The gut, for example, is made from cells that form a hollow tube. Models of this tube in a dish have been made and are called gut organoids.

"However, this tube is not enough to make a functional gut because this organ contains other components, such as smooth muscles, blood vessels and nerves that control the function of the gut and which are made from cells of different origins.

"The only way to have all the variety of cells necessary to the formation of functional organs is to develop systems in which all precursor cells are present. The embryo-like entities we have engineered using stem cells are providing just this."

Developing these fully functioning biological systems requires getting a slew of things just right - such as the correct cell type, spatial location, and timing of cell signals to get the desired outcome. Synthetically recreating these complex processes is only possible thanks to generations of research in developmental biology, including this team's previous research on zebrafish.

Many previous attempts have been built upon. These were missing things like entire types of tissues, didn't form a head structure, failed to organize tissues correctly, or develop to the embryonic stage called gastrulation.

Many of these issues involved the need to spatially confine the developmental chemical signals within the forming embryoid. Thisse and colleagues developed a way to do this in their zebrafish experiments - creating centers for the signalling chemicals that provide the cell clusters with a sense of direction - back and front, head and tail.

They could then control the timing, size, and strength of these signals.

Their work has now culminated in these miraculously functioning mouse embryos, with all the normal early embryonic tissue layers. The correctly organized cells and tissues are arranged properly around the embryoid spinal cord precursor (the notochord), including developing digestive, muscular, nervous and circulatory systems and a beating heart.

However, the embryoid is still missing parts of the brain, and the team suspects this may be because the chemical signal telling the cells they're at the butt end (called a WNT morphogen) spread too far.

"With the techniques we have developed, we should be able, at some point, to manipulate molecular signals that control embryo formation, and this should lead to generating embryo-like entities containing all tissues and organs including the anterior brain," said Bernard Thisse.

The researchers hope to learn how to fully control and manipulate the embryoid development, and think it may become a powerful tool for studying diseases.

"Having all the variety of tissues made allows us to hope that the scientific community will be able to build organs with a proper vascularization, innervation and interactions with other tissues," Christine Thisse said.

"This is essential to be able one day to produce functional human replacement organs in a dish. This would overcome the shortage of organs for transplants."

Their research was published in *Nature Communications*.

https://bbc.in/3whEeLd

Pets can catch Covid from owners, study suggests

Covid is common in pet cats and dogs whose owners have the disease, research suggests.

By Jim Reed Health reporter

Swabs were taken from 310 pets in 196 households where a human infection had been detected. Six cats and seven dogs returned a positive PCR result, while 54 animals tested positive for virus antibodies.

"If you have Covid, you should avoid contact with your cat or dog, just as you would do with other people," Dr Els Broens, from Utrecht University, said. "The main concern is not the animals' health but the potential risk that pets could act as a reservoir of the virus and reintroduce it into the human population."

The authors of the study said no evidence of pet-to-owner transmission had been recorded to date but it would be difficult to detect while the virus was still spreading easily between humans. Most infected pets tend to be asymptomatic or display mild Covid symptoms.

Researchers from Utrecht University sent a mobile veterinary clinic to households in the Netherlands that had tested positive for Covid at some point in the past 200 days.

Swabs were taken from their pet cats and dogs to test for evidence of a current infection, while blood samples were also tested for antibodies suggesting a past exposure to Covid.

The results were presented at the European Congress of Clinical Microbiology and Infectious Diseases:

- ***4.2% showed evidence of a current infection***
- ***17.4% tested positive for antibodies***

Follow-up tests showed all the PCR-positive (polymerase chain reaction) animals cleared the infection and went on to develop antibodies. The researchers say the most likely route of virus transmission is from human to animal, rather than the other way round.

"We can't say there is a 0% risk of owners catching Covid from their pets," Veterinary Microbiological Diagnostic Centre Dr Broens said. "At the moment, the pandemic is still being driven by human-to-human infections, so we just wouldn't detect it." Vets in Russia have started vaccinating some animals against the disease.

But Dr Broens said: "I don't see the scientific evidence for that now. "It seems unlikely that pets play a role in the pandemic."

A separate study run by the University of Guelph in Ontario, Canada, found cats that slept on their owner's bed seemed to be at particular risk of infection.

A total of 48 cats and 54 dogs from 77 households were tested for Covid antibodies and their owners asked about their interaction with their pets. About 67% of the owned cats and 43% of the owned dogs tested positive, compared with 9% of dogs and cats from an animal shelter and 3% of stray cats in the area.

A quarter of the pets displayed a symptom of the disease, from loss of appetite to difficulty breathing. And although most cases were mild, three were severe.

The study's authors said cats' biology may make them more susceptible to Covid. Cats are also more likely to sleep near their owner's face than dogs, increasing their exposure to any infection.

'Robustly conducted'

Cambridge University veterinary medicine department head Prof James Wood said the two studies added to other evidence suggesting a substantial proportion of cats and dogs may catch the virus from their owners.

"The Dutch study is robustly conducted and shows that around 20% of exposed pets may be infected and that they eventually clear the infection just as most humans do," he said. "Most reports are that this infection appears to be asymptomatic. "It also seems that the

virus does not normally transmit from dogs and cats to either other animals or their owners."

https://bit.ly/3dGydl1

Massive DNA study finds rare gene variants that protect against obesity

For some people, no amount of exercise and dieting keeps the kilograms off. For others, leanness comes naturally. Now, scientists might know one reason why.

By Rodrigo Pérez Ortega

In one of the most comprehensive studies of the genetics of obesity to date, a research team has identified rare gene variants that protect lucky carriers from putting on weight.

The work is "a tour de force of genetics," says Sadaf Farooqi, an obesity researcher at the University of Cambridge who was not involved with the study. Geneticists generally look for mutations that cause disease, but people can also carry subtly different versions of a gene that promote good health. Finding rare variants that offer protection against a disease is very hard because sequencing studies are usually small, Farooqi notes. Yet such variants can lead to new drug targets, she adds.

At least 2.8 million people die every year from being overweight or clinically obese. Obesity increases the risk of developing type 2 diabetes, heart disease, some cancers, and even severe COVID-19.

Diet and exercise can help people with obesity lose weight, but genetics also strongly influence whether a person develops the disease. Studies that focused on people with extreme obesity have identified common gene variants—like a "broken" copy of the *MC4R* gene, linked to appetite regulation—that make people more likely to be overweight. Other work has found thousands of genetic variants, each of which has a tiny impact on body weight; together, they can significantly increase the likelihood of obesity.

In the new study, researchers sequenced the genomes of more than 640,000 people from Mexico, the United States, and the United Kingdom, homing in on only the exome—the part of the genome that codes for proteins. That's "a massive amount of work," says Ruth Loos, a human geneticist at the University of Copenhagen not involved in the study. Just as a photo with thousands of pixels reveals tiny details of a scene, she says, the large number of study participants provided a "very high resolution to get to the rarest variants."

Then, the researchers looked at mutations within genes that were associated with a lower or higher body mass index (BMI), the most generally accepted, if imperfect, measure of obesity. Of the 16 genes tied to BMI, five encoded cell surface proteins known as G-protein coupled receptors. Adding to the evidence they influence weight, scientists found that all five of these genes are expressed in the hypothalamus, a brain region that regulates hunger and metabolism.

Variants of one of these genes— *GPR75*—had the largest effect on BMI. Individuals carrying mutations that inactivated one copy of that gene weighed an average of 5.3 kilograms less and had half the odds of being obese compared with those with working versions, the researchers report today in *Science*.

To see how *GPR75* affected weight gain, the researchers engineered mice to lack a working copy of the gene. When fed a high-fat diet, the rodents gained 44% less weight compared with control mice. The modified mice also had better control of blood sugar and were more sensitive to insulin.

Still, the *GPR75* variants that inactivate the gene are rare: Only one in 3000 people seem to carry them. "It influences a very tiny group of the world," says Giles Yeo, a geneticist at Cambridge who was not involved in the study. The fact that the lack of *GPR75* has such clear, strong protective effect in the mice suggests it's involved in metabolic pathways related to obesity, he says. "[That] tells us a lot

of new biology that can influence everybody in the world."

As such, *GPR75* could be a potential drug target, the scientists say; there are two proven molecules that activate the GPR75 receptor, but drugs that switch it off could offer new medication options for patients struggling with obesity.

The work also shows that "it's possible to generalize this approach to other traits and diseases," such as type 2 diabetes and other metabolic disorders, says Luca Lotta, a genetic epidemiologist at Regeneron Genetics Center who led the study.

Still, for Loos, the real value of the research lies in the scale of the sequencing. "It confirms that to study complex diseases such as obesity, we need enormous sample sizes."

https://bit.ly/3dDafHh

Going 'Blank' Looks a Lot Like Parts of The Brain Falling Asleep, Neuroscientists Find

Scientists have a better idea of what happens in our brains when we 'zone out'

Jacinta Bowler

It's easy to get distracted - whether you're daydreaming about a special someone while you should be working, or completely going blank and just taking a brain break.

Now, scientists have gained a better idea of what actually happens in our brains when we 'zone out', and it looks a lot like a part of the brain is... sort-of falling asleep.

"Attentional lapses occur commonly and are associated with mind wandering, where focus is turned to thoughts unrelated to ongoing tasks and environmental demands, or mind blanking, where the stream of consciousness itself comes to a halt," the team – led by neuroscientist Thomas Andrillon – wrote in their new paper.

"Our results suggest attentional lapses share a common physiological origin: the emergence of local sleeplike activity within the awake brain."

When you go to sleep, your brain experiences 'slow waves' of brain activity in the delta (1–4 Hz) or theta (4–7 Hz) ranges during non-rapid eye movement sleep. This is the slow descent before you get to the deep, dream-filled rapid eye movement (REM) sleep.

In contrast, there's this 'sleeplike activity' while you're awake – called local sleep by scientists. It's relatively well studied by researchers and it happens while you're completely awake, but localized brain activity enters a state which resembles sleep.

There are pretty specific times when we know that local sleep happens, particularly when we're really, *really* tired. But the researchers discovered something that looks very similar to local sleep in well-rested volunteers when their minds were wandering or blanking.

"The concept of local sleep builds upon a recent questioning of the classical view of sleep as an all-or-nothing phenomenon," the team explained.

The researchers took 26 healthy and well-rested participants and took readings of their brain waves using an electroencephalogram (EEG). They made the subjects partake in what might be one of the most boring tests in science, called a Go/NoGo test.

It goes like this. The volunteers stared at images of faces or numbers that changed every second or so for over an hour, pressing a button (Go) when they saw neutral faces or any number other than three, but *not* pressing the button (NoGo) when they saw a smiling face or the number three. Every 30 to 60 seconds they were interrupted to report on their mental state.

Unsurprisingly, there were a lot of brains wandering off the task.

"The Go/NoGo tests require participants' sustained attention, but our participants declared focusing on the task only in ~48 percent of the probes," the team wrote.

"The rest of the time, they declared thinking about something else or thinking about nothing."

When the researchers looked at the preceding 20 seconds before the participants said that their mind had wandered off, the EEG recorded a particular type of slow wave in localized areas of the brain, similar to what is seen across someone's entire brain when they're falling asleep.

Interestingly, while slow waves in areas at the front of the brain preceded mind wandering, when they occurred in regions farther back in the brain, the detection was followed by the participants reporting a blank mind.

"We speculate that the slow waves we report here are generated by similar neural mechanisms as slow waves in sleep," the researchers wrote.

"Future studies could use direct evidence from intracranial recordings or sleep deprivation to more solidly establish this interpretation."

The brain is a tricky organ, and we don't yet know if this really is the same as what happens in sleep, or just our meat engines finding some other way of giving ourselves a rest.

Watch this space, and maybe have a daydream in the meantime.

This research has been published in *Nature Communications.*

https://bit.ly/2SKnrTk

Benjamin Franklin's fight against a deadly virus when colonial America was divided over smallpox inoculation

Exactly 300 years ago, in 1721, Benjamin Franklin and his fellow American colonists faced a deadly smallpox outbreak.

by Mark Canada and Christian Chauret, The Conversation

Their varying responses constitute an eerily prescient object lesson for today's world, similarly devastated by a virus and divided over vaccination three centuries later.

As a microbiologist and a Franklin scholar, we see some parallels between then and now that could help governments, journalists and the rest of us cope with the coronavirus pandemic and future threats.

Smallpox strikes Boston

Smallpox was nothing new in 1721. Known to have affected people for at least 3,000 years, it ran rampant in Boston, eventually striking more than half the city's population. The virus killed about 1 in 13 residents—but the death toll was probably more, since the lack of sophisticated epidemiology made it impossible to identify the cause of all deaths.


THE
New-England Courant.
MONDAY August 7. 1721.
Hoc non omnes Negant: Or, Jack of all Trades.

From its first edition, The New-England Courant covered inoculation.

Credit: Wikimedia Commons

What was new, at least to Boston, was a simple procedure that could protect people from the disease. It was known as "variolation" or "inoculation," and involved deliberately exposing someone to the smallpox "matter" from a victim's scabs or pus, injecting the material into the skin using a needle. This approach typically caused a mild disease and induced a state of "immunity" against smallpox.

Even today, the exact mechanism is poorly understood and not much research on variolation has been done. Inoculation through the skin seems to activate an immune response that leads to milder symptoms and less transmission, possibly because of the route of infection and the lower dose. Since it relies on activating the immune response with live smallpox variola virus, inoculation is different from the modern vaccination that eradicated smallpox using the much less harmful but related vaccinia virus.

The inoculation treatment, which originated in Asia and Africa, came to be known in Boston thanks to a man named Onesimus. By 1721, Onesimus was enslaved, owned by the most influential man in all of Boston, the Rev. Cotton Mather.

Known primarily as a Congregational minister, Mather was also a scientist with a special interest in biology. He paid attention when Onesimus told him "he had undergone an operation, which had given him something of the smallpox and would forever preserve him from it; adding that it was often used" in West Africa, where he was from.

Inspired by this information from Onesimus, Mather teamed up with a Boston physician, Zabdiel Boylston, to conduct a scientific study of inoculation's effectiveness worthy of 21st-century praise. They found that of the approximately 300 people Boylston had inoculated, 2% had died, compared with almost 15% of those who contracted smallpox from nature.

The findings seemed clear: Inoculation could help in the fight against smallpox. Science won out in this clergyman's mind. But others were not convinced.

Stirring up controversy

A local newspaper editor named James Franklin had his own affliction—namely an insatiable hunger for controversy. Franklin, who was no fan of Mather, set about attacking inoculation in his newspaper, The New-England Courant.

One article from August 1721 tried to guilt readers into resisting inoculation. If someone gets inoculated and then spreads the disease to someone else, who in turn dies of it, the article asked, "at whose hands shall their Blood be required?" The same article went on to say that "Epidemeal Distempers" such as smallpox come "as Judgments from an angry and displeased God."

In contrast to Mather and Boylston's research, the Courant's articles were designed not to discover, but to sow doubt and distrust. The argument that inoculation might help to spread the disease posits something that was theoretically possible—at least if simple precautions were not taken—but it seems beside the point. If inoculation worked, wouldn't it be worth this small risk, especially since widespread inoculations would dramatically decrease the likelihood that one person would infect another?

Franklin, the Courant's editor, had a kid brother apprenticed to him at the time—a teenager by the name of Benjamin.

Historians don't know which side the younger Franklin took in 1721—or whether he took a side at all—but his subsequent approach to inoculation years later has lessons for the world's current encounter with a deadly virus and a divided response to a vaccine.

Independent thought

You might expect that James' little brother would have been inclined to oppose inoculation as well. After all, thinking like family members and others you identify with is a common human tendency.

That he was capable of overcoming this inclination shows Benjamin Franklin's capacity for independent thought, an asset that would serve him well throughout his life as a writer, scientist and statesman. While sticking with social expectations confers certain advantages in certain settings, being able to shake off these norms when they are dangerous is also valuable. We believe the most successful people are the ones who, like Franklin, have the intellectual flexibility to choose between adherence and independence.

Truth, not victory

What happened next shows that Franklin, unlike his brother—and plenty of pundits and politicians in the 21st century—was more interested in discovering the truth than in proving he was right.

Perhaps the inoculation controversy of 1721 had helped him to understand an unfortunate phenomenon that continues to plague the U.S. in 2021: When people take sides, progress suffers. Tribes, whether long-standing or newly formed around an issue, can devote their energies to demonizing the other side and rallying their own.

Instead of attacking the problem, they attack each other.

Franklin, in fact, became convinced that inoculation was a sound approach to preventing smallpox. Years later he intended to have his son Francis inoculated after recovering from a case of diarrhea. But before inoculation took place, the 4-year-old boy contracted smallpox and died in 1736. Citing a rumor that Francis had died because of inoculation and noting that such a rumor might deter parents from exposing their children to this procedure, Franklin made a point of setting the record straight, explaining that the child had "receiv'd the Distemper in the common Way of Infection."

Writing his autobiography in 1771, Franklin reflected on the tragedy and used it to advocate for inoculation. He explained that he "regretted bitterly and still regret" not inoculating the boy, adding, "This I mention for the sake of parents who omit that operation, on the supposition that they should never forgive themselves if a child died under it; my example showing that the regret may be the same either way, and that, therefore, the safer should be chosen."

A scientific perspective

A final lesson from 1721 has to do with the importance of a truly scientific perspective, one that embraces science, facts and objectivity.

Inoculation was a relatively new procedure for Bostonians in 1721, and this lifesaving method was not without deadly risks. To address this paradox, several physicians meticulously collected data and compared the number of those who died because of natural smallpox with deaths after smallpox inoculation. Boylston essentially carried out what today's researchers would call a clinical study on the efficacy of inoculation. Knowing he needed to demonstrate the usefulness of inoculation in a diverse population, he reported in a short book how he inoculated nearly 300 individuals and carefully noted their symptoms and conditions over days and weeks.

The recent emergency-use authorization of mRNA-based and viral-vector vaccines for COVID-19 has produced a vast array of hoaxes, false claims and conspiracy theories, especially in various social media. Like 18th-century inoculations, these vaccines represent new scientific approaches to vaccination, but ones that are based on decades of scientific research and clinical studies.

We suspect that if he were alive today, Benjamin Franklin would want his example to guide modern scientists, politicians, journalists and everyone else making personal health decisions. Like Mather and Boylston, Franklin was a scientist with a respect for evidence and ultimately for truth.

When it comes to a deadly virus and a divided response to a preventive treatment, Franklin was clear what he would do. It doesn't take a visionary like Franklin to accept the evidence of medical science today.

https://wb.md/3jQJGC6

Heartburn Drugs Improve Blood Sugar Control in People With Diabetes

PPIs improved blood sugar control in people with diabetes but did not reduce the risk of diabetes in the general population

Washington—Antacids improved blood sugar control in people with diabetes but had no effect on reducing the risk of diabetes in the general population, according to a new meta-analysis published in the Endocrine Society's Journal of Clinical Endocrinology & Metabolism.

Type 2 diabetes is a global public health concern affecting almost 10 percent of people worldwide. Doctors may prescribe diet and lifestyle changes, diabetes medications, or insulin to help people with diabetes better manage their blood sugar, but recent data points to common over the counter antacid medicines as another way to improve glucose levels.

"Our research demonstrated that prescribing antacids as an add-on to standard care was superior to standard therapy in decreasing hemoglobin A1c (HbA1c) levels and fasting blood sugar in people with diabetes," said study author Carol Chiung-Hui Peng, M.D., of the University of Maryland Medical Center Midtown Campus in Baltimore, Md.

"For people without diabetes, taking antacids did not significantly alter their risk of developing the disease," said study author, Huei-Kai Huang M.D., of the Hualien Tzu Chi Hospital in Hualien, Taiwan.

The researchers performed a meta-analysis on the effects of proton pump inhibitors (PPIs)—a commonly used type of antacid medication—on blood sugar levels in people with diabetes and whether these medications could prevent the new onset of diabetes in the general population. The analysis included seven studies (342 participants) for glycemic control and 5 studies (244, 439 participants) for risk of incident diabetes. The researchers found antacids can reduce HbA1c levels by 0.36% in people with diabetes and lower fasting blood sugar by 10 mg/dl based on the results from seven clinical trials. For those without diabetes, the results of the five studies showed that antacids had no effect on reducing the risk of developing diabetes.

"People with diabetes should be aware that these commonly used antacid medications may improve their blood sugar control, and providers could consider this glucose-lowering effect when prescribing these medications to their patients," said study author Kashif Munir, M.D., associate professor in the division of endocrinology, diabetes and nutrition at the University of Maryland School of Medicine in Baltimore, Md.

Other authors of the study include: Yuting Huang and Khulood Bukhari of the University of Maryland Medical Center Midtown Campus in Baltimore, Md.; Yu-Kang Tu of the National Taiwan University and the Taipei Medical University in Taipei, Taiwan; Gin Yi Lee of the Danbury Hospital in Danbury, Conn.; Rachel Huai-En Chang of the Johns Hopkins Bloomberg School of Public Health in Baltimore, Md.; Yao-Chou Tsai of the Taipei Medical University; Yunting Fu of the University of Maryland in Baltimore, Md.

The manuscript received no external funding.

The manuscript, "Effects of Proton Pump Inhibitors on Glycemic Control and Incident Diabetes: A Systematic Review and Meta-analysis," was published online, ahead of print.

https://bit.ly/3qKQ7Z7

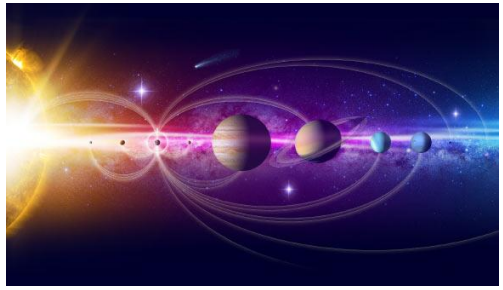
Proximity to Sun's Magnetic Field Determines Composition of Rocky Planets, Study Says

Model shows that density, mass and iron content of a rocky planet's core are influenced by its distance from the Sun's magnetic field

Terrestrial planets (Mercury, Venus, Earth, and Mars) are differentiated into three layers: a metallic core, a silicate shell (mantle and crust), and a volatile envelope of gases, ices, and, for the Earth, liquid water.

Each layer has different dominant elements (e.g., increasing iron content with depth and increasing oxygen content to the surface). University of Maryland's Professor William McDonough and Tohoku University's Dr. Takashi Yoshizaki have now developed a model showing that the density, mass and iron content of a rocky planet's core are influenced by its distance from the magnetic field of the Sun.

The new model developed by Professor McDonough and Dr. Yoshizaki shows that during the early formation of our Solar System, when the young Sun was surrounded by a swirling cloud of dust and gas, grains of iron were drawn toward the center by the Sun's magnetic field.



A view of the planets of our Solar System. **Image credit: Jenny Mottar / NASA.**

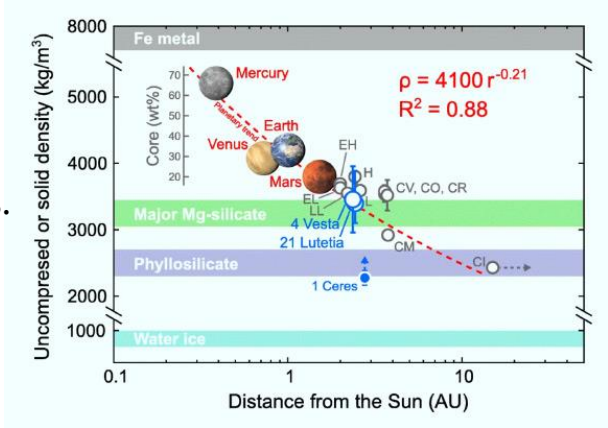
When the planets began to form from clumps of that dust and gas, planets closer to the Sun incorporated more iron into their cores

than those farther away.

The researchers found that the density and proportion of iron in a rocky planet's core correlates with the strength of the magnetic field around the Sun during planetary formation.

Their study suggests that magnetism should be factored into future attempts to describe the composition of rocky planets, including those outside our Solar System.

The composition of a planet's core is important for its potential to support life. On Earth, for instance, a molten iron core creates a magnetosphere that protects the planet from cancer-causing cosmic rays. The core also contains the majority of the planet's phosphorus, which is an important nutrient for sustaining carbon-based life.



Density of the rocky solar system bodies: uncompressed and solid densities are shown for terrestrial planets and chondrites (gray), respectively; bulk planetary densities are shown for asteroids (blue); for 1 Ceres, its bulk density is a lower limit of its solid density, given its high ice abundance and porosity; the red line shows a fit curve for the planets. **Image credit: McDonough & Yoshizaki, doi: 10.1186/s40645-021-00429-4.**

Using existing models of planetary formation, the scientists determined the speed at which gas and dust was pulled into the center of our Solar System during its formation.

They factored in the magnetic field that would have been generated by the Sun as it burst into being and calculated how that magnetic field would draw iron through the dust and gas cloud.

As the early Solar System began to cool, dust and gas that were not drawn into the Sun began to clump together.

The clumps closer to the Sun would have been exposed to a stronger magnetic field and thus would contain more iron than those farther away from the Sun.

As the clumps coalesced and cooled into spinning planets, gravitational forces drew the iron into their core.

When the authors incorporated their model into calculations of planetary formation, it revealed a gradient in metal content and density that corresponds perfectly with what scientists know about the planets in our Solar System.

Mercury has a metallic core that makes up about three-quarters of its mass. The cores of Earth and Venus are only about one-third of their mass, and Mars, the outermost of the rocky planets, has a small core that is only about one-quarter of its mass.

This new understanding of the role magnetism plays in planetary formation creates a kink in the study of exoplanets, because there is currently no method to determine the magnetic properties of a star from Earth-based observations.

"The attributes of our Solar System may be equally applicable to exoplanetary systems," the researchers said.

"The generation of a planetary magnetosphere, which nurtures life, shapes a planet's habitability."

"It is likely that life's sustainability critically depends on being sited in the Goldilocks zone and having the right amount of metallic core, which contains an appropriate amount of a light element and is not cooling too fast."

The team's paper was published in the journal *Progress in Earth and Planetary Science*.

W.F. McDonough & T. Yoshizaki. 2021. Terrestrial planet compositions controlled by accretion disk magnetic field. Prog Earth Planet Sci 8, 39; doi: 10.1186/s40645-021-00429-4

https://nyti.ms/3qYVbt7

These Plants Act Like Bees in a Hive

The plants seem to divide labor to maximize the health of their colonies that grow up the sides of trees.

By Elizabeth Preston

K.C. Burns's favorite research days are the ones where he puts on his backpack and walks into the wilderness with no agenda. On one hike on Australia's Lord Howe Island, he came across a cluster of staghorn ferns. They are common potted plants, but in nature they grow in dense colonies that cling to treetops. In the volcanic island's stunted forest, those treetops are right at eye level.

A staghorn fern in Lamington National Park in Queensland, Australia.
Credit...Mauritius Images GmbH/Alamy

"I almost looked beyond it," said Dr. Burns, a biologist at Victoria University of Wellington in New Zealand. Then he peered closer and realized the plants within the colony were doing different jobs to survive. Ferns growing higher up had waxy fronds that seemed to direct rainwater into the colony's center. Farther down, ferns grew spongier leaves that were damp to the touch. Some plants weren't reproducing at all — they seemed to have dedicated their lives to collecting water for their neighbors' entangled roots.

It struck Dr. Burns that the ferns were working together as a kind of superorganism, perhaps like bees in a hive.

"I sat down and thought, oh my God," he said. In a paper published last month in Ecology, Dr. Burns and his co-authors argued that colonies of the staghorn fern Platycerium bifurcatum show a kind of collective behavior known as eusociality. Until now, scientists had only recognized eusociality in some species of animals like bees or ants that live in colonies and divide their labor.

To measure how ferns divided labor, the researchers sampled plants growing at different heights within 24 colonies. They counted two types of leaves on each plant. One type, which they called nest fronds, are rounded and mostly brown, clasping the tree like cupped hands. The other fronds, long, green and forked like antlers, can grow spores on their undersides that will become the next generation of ferns.

Plants closer to the top of each colony had more spore-bearing fronds. Plants near the bottom had more of the cupped, non-reproducing nest fronds. About 40 percent of individual plants weren't reproducing at all, like worker bees.

Next the scientists cut out wedges from nest fronds, dried them, then soaked them in water to measure how much they sopped up. They found that nest fronds from the bottom of a colony were more

Since the colony's roots grew in a tangled network, these spongy leaves might help the whole colony stay hydrated. The scientists found that larger colonies (the biggest one they studied held 58 individual ferns) had more spore-bearing fronds per capita. Living in a big group, then, might improve the ferns' fitness.

For the most part, the groups are families. "We quickly realized the genetics is important," Dr. Burns said, because eusocial animals live in closely related groups.

When researchers analyzed DNA from 11 fern colonies, they found that most plants within a colony were as closely related as possible: They were clones. New plants arise from buds in the root systems of others, Dr. Burns said.

Being clones "means that the different individuals have aligned interests genetically," said Guy Cooper, an evolutionary biologist at the University of Oxford. By helping a neighboring clone, a plant is also helping its own genes survive.

Dr. Cooper said he would like to know more about the life cycle of

a colony, and how much the individual ferns depended on one another. Even if staghorn ferns aren't as social as bees, "it was very cool to see that there might be similar sorts of complex social behaviors happening in plants," he said.

He also pointed out that some plants that spread by cloning themselves were considered to be one individual, not many. For example, aspen trees sprout massive groves of clones from one root network. An aspen forest in Utah nicknamed Pando is sometimes called the world's largest single organism, covering 106 acres.

"You then have to wonder about some more philosophical questions about whether they are different individuals to start with," Dr. Cooper said of the ferns. Maybe the ferns within a colony are more like limbs on a body than bees in a hive.

Cloning doesn't explain the whole story of staghorn ferns, though. In some Lord Howe Island colonies, Dr. Burns and his colleagues found unrelated plants. They don't know how those ferns became part of the treetop communities.

Plants are some of the most flexible living things on Earth, said Karen Kapheim, a biologist at Utah State University who studies the evolution of social behavior in bees. Maybe it's not surprising that a fern could also evolve social tendencies, she said.

Science is revealing more and more about how plants behave and communicate, Dr. Kapheim said. "I think adding social behavior to that fits in with this new, emerging understanding of plants."

https://bit.ly/3ypIlGw

Visiting Comet 46P/Wirtanen Is 'Abnormally High' in Alcohol

Comets are boozy beasts.

They come in here to the inner Solar System from goodness-knows-where (the outer Solar System), get a little warmth, and start spewing alcoholic compounds into space, willy-nilly.

Michelle Starr

Comet 46P/Wirtanen, which visited the inner Solar System in 2018, takes the martini. According to an analysis of its atmosphere, or coma, it was giving off what scientists have called an "abnormally high" amount of alcohol. And this can tell us some really interesting things about the evolution of the Solar System.

"46P/Wirtanen has one of the highest alcohol-to-aldehyde ratios measured in any comet to date," said cometary scientist Neil Dello Russo of Johns Hopkins University Applied Physics Laboratory. "This tells us information about how carbon, oxygen, and hydrogen molecules were distributed in the early Solar System where Wirtanen formed."

Comet 46P/Wirtanen is a fairly regular visitor to the inner Solar System. It swings around the Sun every 5.4 years, occasionally veering so close to Earth that it is visible in the night sky to the naked eye. On its most recent visit, in December 2018, it came within 11.6 million kilometers (7.2 million miles) of Earth, around 30 times the average distance between Earth and the Moon.

Astronomers took full advantage of this opportunity to study this comet from relatively close quarters, using the Keck Observatory's newly-upgraded Near-Infrared Spectrograph (NIRSPEC).

This instrument can collect data on the sunlight that shines through the comet's coma so that scientists can then analyze it to determine its chemical composition.

Cometary comas can tell us a lot about the outer and early Solar System. Comets differ from asteroids in that they're filled with all sorts of frozen compounds – ices – that got bound up in them when they formed, hence the nickname "dirty snowball".

For most of a comet's orbit, these ices remain frozen, but when the comet draws close enough to the warmth of the Sun, the ices start to sublimate, dislodging dust and creating a dusty, gaseous envelope.

It's this material that forms the comet's gas and dust tails, streaming away from the Sun due to solar wind and radiation pressure.

And, because this material has been sitting locked frozen in a comet from the time the body formed – when the Solar System was a baby – until sublimation, it contains information about the composition of the cloud from which the Solar System itself formed.

In the coma of 46P/Wirtanen, NIRSPEC took just 10 to 20 minutes to detect its composition: acetylene, ammonia, ethane, formaldehyde, hydrogen cyanide (which breaks apart to create cyanogen, the compound that makes the comet glow green), methanol, and water.

The NIRSPEC data can also reveal the temperature of the coma, and here the scientists found something really odd. There was evidence of more heat than could be accounted for by just the Sun.

"We found that the temperature measured for water gas in the coma did not decrease significantly with distance from the nucleus, which implies a heating mechanism," said astronomer Erika Gibb of the University of Missouri-St. Louis.

It's unclear what this heating mechanism could be, but there are multiple possibilities. One possibility is that solar radiation could have ionized some of the molecules in the coma, close to the cometary nucleus, which would result in the release of energetic electrons. These electrons could collide with other molecules and transfer energy, which is released as heat.

Another is that solid chunks and grains of ice broke off the comet, tumbling away from the nucleus before sublimating and releasing energy via collisions in the cooler cloud at that distance, rather than closer in. The team did find a significantly higher proportion of water in the outer coma compared to other compounds, which is consistent with this model.

This may help explain how water could have been delivered to planets like Earth. Although the water ice sublimates at the comet, it may return to liquid or ice form when it lands on a planet.

Other ingredients for life have also been found on comets, so these dirty snowballs could be vitally important not just for our own existence, but for life elsewhere in the Universe.

"Comet studies like this are exciting because they serve as a launchpad for answering the million-dollar question – are we alone?" said astronomer Greg Doppmann of Keck Observatory.

"The organic compounds on comets tell us what ingredients formed our solar system and served as precursors to life. We can then look for these same prebiotic molecules in other planetary systems, which opens an exciting door to the very real possibility of finding microbial life beyond Earth – not in our kids' lifetimes, but our own lifetime."

The research has been published in *The Planetary Science Journal*.