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How Slime Molds Remember Where They Ate

These simple organisms physically encode food locations to solve complex tasks

By Lars Fischer

Physarum polycephalum. **Credit: Scott Camazine Science Source**

Like all slime molds, *Physarum polycephalum* has no brain or nervous system—yet it somehow "remembers" food sites for future reference.

In a new paper, biophysicists Mirna Kramar and Karen Alim of the Max Planck Institute for Dynamics and Self-Organization in Göttingen, Germany, describe how the organism's internal structure changes to encode past food locations.

Although slime molds are extremely simple organisms—just a system of interlaced tubes—they can solve complex optimization problems such as finding the shortest path through a maze. Pure stimulus-response activity patterns—for instance, crawling toward increasing concentrations of certain molecules or avoiding harmful mechanical stimuli—cannot explain the extent of their skill. How they can take in and retain information has long remained unclear.

The study, published in the *Proceedings of the National Academy of Sciences USA*, revealed that when parts of *P. polycephalum* come in contact with a food source, they release a substance that softens the tube network's gel-like walls, making them widen from their inherent internal pressure. The slime mold moves by expanding along wider tubes and pruning narrower ones—so the enlarged tubes effectively record past food locations, as they influence the organism's overall direction of growth even after the food is gone.

The researchers do not yet know what the softening substance is, but by modeling changes in tube diameters, they found it is likely a soluble material that spreads by flow and diffusion. The team suggests this mechanism could also be common in other "living flow networks," such as vertebrate vascular systems.

Kramar and Alim "have pinned down nicely a mechanobiological mechanism for slime mold behavior implementing something like memory," says University of Bremen physicist Hans-Günther Döbereiner, who was not involved in the study. Future research into a slime mold's ability to carry out complex tasks, he says, will require an examination of "molecular signaling, material properties and flow patterns of the cellular fluid regulating its behavior."

New Jersey Institute of Technology biologist Simon Garnier, who was also not involved in the study, adds that this work builds on prior investigations of how this organism encodes past experiences. The researchers' model "provides a nice mechanistic explanation for how slime mold achieves this feat," he says. It could lead to improved network optimization and routing algorithms, Garnier adds, similar to those inspired by ant colonies.

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Converting scar tissue to heart muscle after a heart attack

Researchers from the University of Tsukuba demonstrate the direct conversion of scar tissue cells to heart muscle cells in mice after a heart attack

Tsukuba, Japan - It is estimated that during a heart attack, one billion cells in the heart are lost. In the wake of the heart attack, the lost tissue is replaced by scar tissue, which can lead to heart failure, arrhythmia and death. In a new study, researchers from the University of Tsukuba have shown how cells in the scar tissue can be converted to heart muscle cells, effectively regenerating the injured heart.

The injured heart of humans and rodents alike does not have the capacity to regenerate after injury. Therefore, the only way for the heart to heal the wound is to build a scar tissue in the injured area.

A longstanding goal in the field has been to find a way to reprogram fibroblasts, cells that produce the connective tissue in a scar, to cardiomyocytes, the working heart muscle cells. By doing so, the lost heart muscle cells could be replaced, effectively preventing the heart from going into heart failure, a heart muscle weakness that can lead to death.

Previous studies have shown that cardiomyocytes appear to be formed by directly injecting a harmless virus carrying a set of cardiac transcription factors, proteins that drive the expression of genes that heart muscle cells need for their development and function, into the heart of rodents after a heart attack. However, the origin and functional significance of these newly formed heart muscle cells has not unequivocally been determined yet.

"Direct cardiac reprogramming holds great potential for cardiac regeneration and the treatment of myocardial infarction," says lead author of the study Professor Masaki Ieda. "However, when transcription factors are introduced, apparent cardiomyocytes may be formed either by converting fibroblasts to new cardiomyocytes or by fusing fibroblasts with existing cardiomyocytes. The difference is that only the former process, which we call 'direct reprogramming', significantly contributes to regeneration. In this study, our goal was to determine how new cardiomyocytes are formed when cardiac transcription factors are introduced after myocardial infarction."

To achieve their goal, the researchers first generated mice in which all cells emitted red fluorescence. However, the mice were modified in a way that the fibroblasts emitted green fluorescence after treatment with the drug tamoxifen. As a result, when looking at the heart after treatment with tamoxifen, cells that emitted both red and green fluorescence indicated that cell fusion between fibroblasts and cardiomyocytes had happened. Conversely, the presence of green fluorescence indicated that direct reprogramming of fibroblasts to cardiomyocytes had occurred.

Equipped with the tools to tackle their research question, the researchers used a mouse model of heart attack and treated the mice with tamoxifen. While there was no direct reprogramming in a control group, the researchers found 1-1.5% of directly reprogrammed cells when a virus carrying cardiac transcription factors was injected into the mice. Both groups exhibited minimal cell fusion. These results suggest that the main route of generating new heart muscle cells by this method is via reprogramming fibroblasts directly to cardiomyocytes.

"These are striking results that show that fibroblasts can be directly reprogrammed to cardiomyocytes. Our findings demonstrate the exciting potential of direct reprograming as a strategy for cardiac regeneration after myocardial infarction," says Professor Ieda.

The article, "Overexpression of Gata4, Mef2c, and Tbx5 Generates Induced Cardiomyocytes via Direct Reprogramming and Rare Fusion in the Heart" was published in Circulation at DOI: 10.1161/CIRCULATIONAHA.120.052799

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More Than 30,000 Years Ago, Study Suggests

The dates for the bone samples excavated from the early depositional levels of Coxcatlan Cave ranged from 33,448 to 28,279 years old.

Archaeologists have obtained radiocarbon dates for the faunal bones excavated from Coxcatlan Cave, a dry rock shelter located within the southern portion of the Tehuacan Valley, southern Puebla, Mexico. The dates for the bone samples from the early depositional levels of the cave ranged from 33,448 to 28,279 years old.

Coxcatlan Cave is a north-facing, dry rockshelter site in the southern portion of the Tehuacan Valley along the alluvial slopes of the Sierra Madre Oriental. The cave is several meters above the valley floor on a low bluff. It extends approximately 30 m in length

and 8 m in width.

Within the cave, archaeologists previously excavated to a maximum depth of 4 m, documenting 28 horizontal stratigraphic levels, or habitation zones, and 42 discrete occupational episodes.

The zones occupied by people who did not make or use pottery, referred to as the Preceramic zones, are the earliest levels of the rock shelter.

These zones have been divided into four cultural phases — the Ajuereado, El Riego, Coxcatlan, and Abejas phases — based on changes in the stone tool technology, basketry and woven matting, and settlement patterns. The earliest evidence for human occupation in the Tehuacan Valley occurred during the Ajuereado phase.

"Even though previous studies had not dated items from the bottom of Coxcatlan Cave, we were not expecting such old ages," said Dr. Andrew Somerville, a researcher in the Department of World Languages and Cultures at Iowa State University.

"The findings add to the debate over a long-standing theory that the first humans crossed the Bering Land Bridge into the Americas 13,000 years ago." "We weren't trying to weigh in on this debate or even find really old samples. We were just trying to situate our agricultural study with a firmer timeline," he added.

"We were surprised to find these really old dates at the bottom of the cave, and it means that we need to take a closer look at the artifacts recovered from those levels."

Dr. Somerville and colleagues selected a sample of 17 bones — eight lagomorphs (hares and rabbits) and nine deer specimens — from the Ajuereado levels of Coxcatlan Cave for radiocarbon dating.

The findings provide the researchers with a better understanding of the chronology of the region. However, questions still remain. Most importantly, is there a human link to the bottom layer of the cave where the bones were found?

"If closer examination of the bones provides evidence of a human link, it will change what we know about the timing and how the first people came to America," Dr. Somerville said.

"Pushing the arrival of humans in North America back to over 30,000 years ago would mean that humans were already in North America prior to the period of the Last Glacial Maximum, when the Ice Age was at its absolute worst."

"Large parts of North America would have been inhospitable to human populations. The glaciers would have completely blocked any passage over land coming from Alaska and Canada, which means people probably would have had to come to the Americas by boats down the Pacific coast."

The results appear in the journal *Latin American Antiquity*.

Andrew D. Somerville et al. New AMS Radiocarbon Ages from the Preceramic Levels of Coxcatlan Cave, Puebla, Mexico: A Pleistocene Occupation of the Tehuacan Valley? Latin American Antiquity, published online May 19, 2021; doi: 10.1017/laq.2021.26

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We Finally Have a Simple System For Naming All The Concerning COVID-19 Variants

COVID-19 variants are to be known by letters of the Greek alphabet to avoid stigmatizing nations where they were first detected, the World Health Organization announced Monday.

Robin Millard, AFP

The new system applies to variants of concern - the most troubling of which four are in circulation - and the second-level variants of interest being tracked. "They will not replace existing scientific names, but are aimed to help in public discussion," said Maria Van Kerkhove, the WHO's COVID-19 technical lead.

Under the new system, the variants of concern take on the following names: the hitherto so-called British variant B.1.1.7 becomes **Alpha**; the B.1.351 first discovered in South Africa becomes **Beta**, while the Brazilian P.1 becomes **Gamma**.

The so-called Indian variant B.1.617 is split into sub-lineages, of

which the B.1.617.2 variant of concern becomes **Delta**.
The B.1.617.1 variant of interest is called **Kappa**.
Besides these names, there are two other scientific names in use for each mutation, while different geographic names have been used to describe the same variant.
For example, within Britain, what other countries have been referring to as the British variant is often called the Kent variant - the county in southeast England where it was first discovered.
The lineage names such as B.1.1.7.2 will still continue to be used in scientific circles, for the mutation information that their name conveys.

Stigmatizing and discriminatory

"While they have their advantages, these scientific names can be difficult to say and recall, and are prone to misreporting," the WHO said in a statement.
"As a result, people often resort to calling variants by the places where they are detected, which is stigmatizing and discriminatory.
"To avoid this and to simplify public communications, WHO encourages national authorities, media outlets and others to adopt these new labels."

No country should be stigmatized for detecting and reporting variants. Globally, we need robust surveillance for variants, incl epi, molecular and sequencing to be carried out and shared. We need to continue to do all we can to reduce the spread of SARS-CoV-2 #COVID19 @WHO

— Maria Van Kerkhove (@mvankerkhove) May 31, 2021

Earlier this month, US President Joe Biden signed a hate crimes law aimed at protecting Asian Americans who have suffered a surge in attacks during the COVID-19 pandemic. US anti-extremism groups say the number of attacks and hate crimes against Asian Americans has exploded since the beginning of the crisis.
They lay some of the blame with former president Donald Trump, who repeatedly referred to COVID-19 as the "China virus".
The WHO has been trying to come up with simplified new nomenclature for the variants for several months.
The Greek alphabet contains 24 letters but there is no plan yet as to where to go next if they are exhausted. Epsilon, Zeta, Eta, Theta and Iota have already been ascribed to variants of interest.

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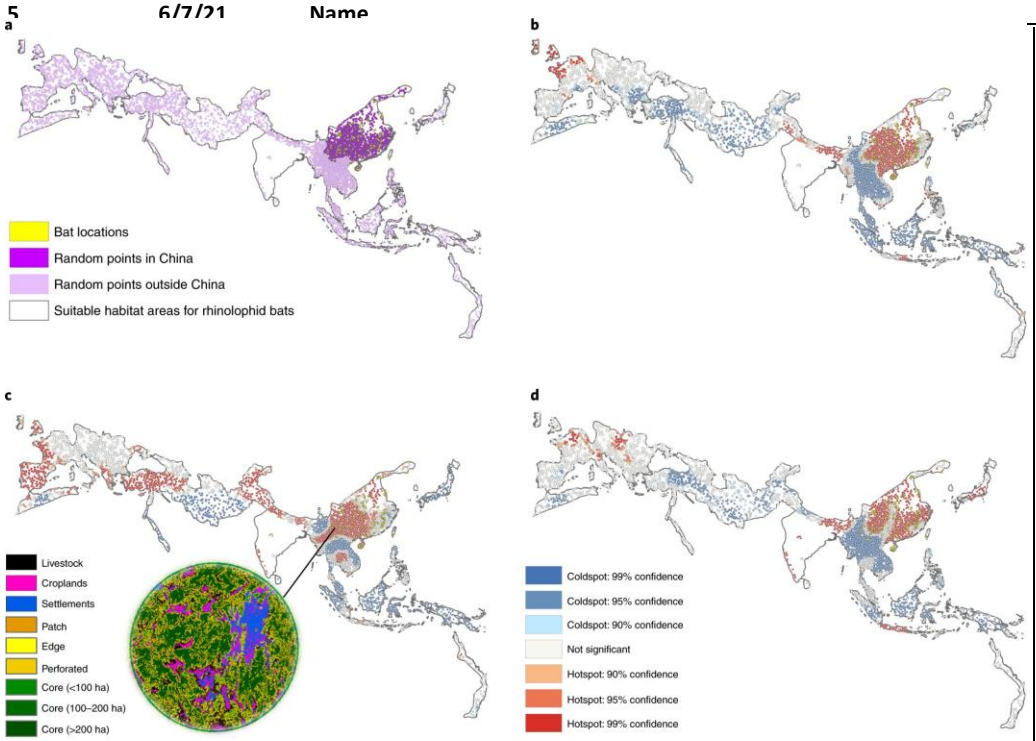
Mapping zoonotic 'hot spots' where risk of coronaviruses jumping from bats to humans is highest

Maps of possible hotspots in Asia and Europe where risk of coronaviruses jumping from bats to humans is highest

by Bob Yirka , Phys.org

A team of researchers from Politecnico di Milano, the University of California and Massey University has created maps of possible hotspots in Asia and Europe where the risk of coronaviruses jumping from bats to humans is highest. In their paper published in the journal *Nature Food*, the researchers describe how they combined data regarding human population densities, horseshoe bats, land use and other factors to create their maps and what they showed. Monia Santini, with Foundation Euro-Mediterranean Center on Climate Change, has published a News & Views piece in the same journal issue outlining the known ways that coronaviruses can jump from animals to humans and the work done by the team on this new effort.
The global COVID-19 pandemic has put a lot of pressure on scientists to learn more about coronaviruses and how they can lead to pandemics. One area of research involves the means by which coronaviruses jump from animals, such as bats, to humans. Prior research has suggested that if the places most at risk for such jumps could be identified, then they could be monitored more closely to quickly react when such jumps occur. In this new effort, the researchers have used a variety of resources to find those places most at risk and to map them.



***Univariate spatial analysis of coronavirus outbreak drivers*. Credit: Nature Food (2021). DOI: 10.1038/s43016-021-00285-x**

Prior research has shown that one of the major factors involved in viruses jumping from animals to humans is human encroachment on natural habitats. As humans take down forests, some of the animals living in them attempt to adapt by learning to live in the new environment. This leads to interactions between wild animals and domesticated animals and humans. The result can be viruses moving from animals to humans, so call zoonotic disease transfer, or jumping. Thus, to isolate likely hotspots for coronavirus jumping, the researchers looked for recent encroachments by studying satellite images. They also obtained information from existing databases that track wildlife such as horseshoe bats—the only animal that has been found to consistently host SARS-type coronaviruses. They also pulled data from human population databases and from databases that hold information about domesticated animals, most particularly livestock.

The researchers then analyzed all the data and used it to identify certain hotspots around the world. They then marked the hotspots on a map, making it easier for viewers to see patterns and to make risk assessments.

More information: *Maria Cristina Rulli et al, Land-use change and the livestock revolution increase the risk of zoonotic coronavirus transmission from rhinolophid bats, Nature Food (2021). DOI: 10.1038/s43016-021-00285-x*

Monia Santini, The land use–food–coronavirus nexus, Nature Food (2021). DOI: 10.1038/s43016-021-00290-0

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New study may help explain low oxygen levels in COVID-19 patients

U of A researchers find SARS-CoV-2 infects immature red blood cells, reducing oxygen in the blood and impairing immune response

A new study published in the journal *Stem Cell Reports* by University of Alberta researchers is shedding light on why many COVID-19 patients, even those not in hospital, are suffering from hypoxia--a potentially dangerous condition in which there is decreased oxygenation in the body's tissues. The study also shows why the anti-inflammatory drug dexamethasone has been an effective treatment for those with the virus.

"Low blood-oxygen levels have been a significant problem in COVID-19 patients," said study lead Shokrollah Elahi, associate professor in the Faculty of Medicine & Dentistry. "Because of that, we thought one potential mechanism might be that COVID-19 impacts red blood cell production."

In the study, Elahi and his team examined the blood of 128 patients with COVID-19. The patients included those who were critically ill and admitted to the ICU, those who had moderate symptoms and were admitted to hospital, and those who had a mild version of the

disease and only spent a few hours in hospital. The researchers found that, as the disease became more severe, more immature red blood cells flooded into blood circulation, sometimes making up as much as 60 per cent of the total cells in the blood. By comparison, immature red blood cells make up less than one per cent, or none at all, in a healthy individual's blood.

"Immature red blood cells reside in the bone marrow and we do not normally see them in blood circulation," Elahi explained. "This indicates that the virus is impacting the source of these cells. As a result, and to compensate for the depletion of healthy immature red blood cells, the body is producing significantly more of them in order to provide enough oxygen for the body."

The problem is that immature red blood cells do not transport oxygen--only mature red blood cells do. The second issue is that immature red blood cells are highly susceptible to COVID-19 infection. As immature red blood cells are attacked and destroyed by the virus, the body is unable to replace mature red blood cells--which only live for about 120 days--and the ability to transport oxygen in the bloodstream is diminished.

The question was how the virus infects the immature red blood cells. Elahi, known for his prior work demonstrating that immature red blood cells made certain cells more susceptible to HIV, began by investigating whether the immature red blood cells have receptors for SARS-CoV-2. After a series of studies, Elahi's team was the first in the world to demonstrate that immature red blood cells expressed the receptor ACE2 and a co-receptor, TMPRSS2, which allowed SARS-CoV-2 to infect them.

Working in conjunction with the the lab of virologist Lorne Tyrrell at the U of A's Li Ka Shing Institute of Virology, the team performed investigative infection testing with immature red blood cells from COVID-19 patients and proved these cells got infected with the SARS-CoV-2 virus.

"These findings are exciting but also show two significant consequences," Elahi said. "First, immature red blood cells are the cells being infected by the virus, and when the virus kills them, it forces the body to try to meet the oxygen supply requirements by pumping more immature red blood cells out of the bone marrow. But that just creates more targets for the virus.

"Second, immature red blood cells are actually potent immunosuppressive cells; they suppress antibody production and they suppress T-cell immunity against the virus, making the entire situation worse. So in this study, we have demonstrated that more immature red blood cells means a weaker immune response against the virus."

Following the discovery that immature red blood cells have receptors that allow them to become infected by the coronavirus, Elahi's team then began testing various drugs to see whether they could reduce immature red blood cells' susceptibility to the virus.

"We tried the anti-inflammatory drug dexamethasone, which we knew helped to reduce mortality and the duration of the disease in COVID-19 patients, and we found a significant reduction in the infection of immature red blood cells," said Elahi.

When the team began exploring why dexamethasone had such an effect, they found two potential mechanisms. First, dexamethasone suppresses the response of the ACE2 and TMPRSS2 receptors to SARS-CoV-2 in immature red blood cells, reducing the opportunities for infection. Second, dexamethasone increases the rate at which the immature red blood cells mature, helping the cells shed their nuclei faster. Without the nuclei, the virus has nowhere to replicate.

Luckily, putting Elahi's findings into practice doesn't require significant changes in the way COVID-19 patients are being treated now.

"For the past year, dexamethasone has been widely used in

COVID-19 treatment, but there wasn't a good understanding as to why or how it worked," Elahi said. "So we are not repurposing or introducing a new medication; we are providing a mechanism that explains why patients benefit from the drug."

Elahi noted that Wendy Sligl and Mohammed Osman had a crucial role in recruiting COVID-19 patients for the study. The research was supported by Fast Grants, the Canadian Institutes of Health Research and a grant from the Li Ka Shing Institute of Virology.

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Our Brains Have More in Common With Testicles Than You Ever Wanted to Know

That delightful saying about men thinking with their nether regions has gained a new meaning.

Tessa Koumoundouros

A new study has found an unnerving lot of similarities between men's brains and the innards of their scrotums.

Sperm production tubules under a scanning electron microscope. **(Steve Gschmeissner/SPL/Getty Images)**

"Brain and testis have the highest number of common proteins, compared with other human body tissues," a team led by biomedical scientist Bárbara Matos from the University of Aveiro in Portugal writes in their new paper.

While the brain has a highly complex role - controlling our bodies, receiving and interpreting signals from sensory organs, not to mention doing all our thinking and feeling, human testes have just two main functions - the production of sperm and hormones. (Although, many of us should be forgiven for attributing these gonads with their own thoughts and feelings too.)

Previous studies have suggested there are links between sexual dysfunction and brain disorders, and even between intelligence and semen quality. Of course, such links do not mean much by themselves, but now the team of researchers from Portugal and the UK has found an explanation for why they might exist.

They compared proteins across 33 tissue types, including the heart, intestine, cervix, ovaries and placenta, and found that testes and brains share 13,442 proteins in common. This is corroborated by gene expression studies showing these two distantly positioned organs share the highest number of genes among all the organs in the body.

Taking a closer look at the shared proteins most highly expressed in these tissues, Matos and colleagues found they're mostly involved in tissue development and cell communication. These shared proteins make sense when you consider how unexpectedly similar the two tissues are in many ways, the team explains.

The brain and testes are both greedy for energy to fuel highly demanding processes like thinking and the production of several million little sperms per day. So both organs have specialized cells to support the hard-working neurons in the brain and germ cells in the testes - to keep them well fed and physically comfortable.

Also, despite being very differently purposed cells, neurons function similarly to sperm in several ways. Both cells have important tasks involving moving stuff from within themselves to their outside environment - a process called exocytosis.

This is how brain cells pass neurotransmitters between each other. In sperm, the same process is used to release important fertilization factors.

In neurons, exocytosis is also involved in the growth of their reaching little branching arms collectively called neurites (dendrites and axons), while in sperm this process allows its innards to fuse with an egg.

"This is an underexplored topic, and the connection between these tissues needs to be clarified, which could help to understand the dysfunctions affecting brain and testis," the team wrote.

These findings raise a lot of questions, the obvious being how did two such disparate organs end up sharing so much in common? The researchers suspect it's because they're both strongly influenced by the speciation process.

Just like animals separated by millions of years of evolution and evolved half a world away from each other can develop the same traits, so too can different tissue groups within the human body.

For example, unlike most other animals, koalas have fingerprints confusingly similar to ours - thanks to the obvious selection pressure exerted by our (well, our primate ancestors') need to grip trees - despite 70 million years of evolution between us. This process is called convergent evolution.

In this case, the researchers propose the same selection pressures involved in keeping species distinct from each other may be imposed on both organs, causing them to evolve convergently. They point to 60 protein-coding genes, unique to humans, many of which are found within the brain and testis.

"The highest expression levels in cerebral cortex and testis suggested that these genes may contribute to phenotypic features that are exclusive of humans, such as the improved cognitive ability," the team wrote.

While owners of testes may not be so thrilled by these biological revelations, the rest of us might be inclined to think it makes an awful lot of sense. But before we get too ahead of ourselves, this finding means female brains share these similarities with balls, too.

Their research was published in *Royal Society Open Biology*.

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Atmospheric metal layers appear with surprising regularity

Twice a day, at dusk and just before dawn, a faint layer of sodium and other metals begins sinking down through the atmosphere, about 90 miles high above the city of Boulder, Colorado.

The movement was captured by one of the world's most sensitive "lidar" instruments and reported today in the AGU journal *Geophysical Research Letters*.

The metals in those layers come originally from rocky material blasting into Earth's atmosphere from space, and the regularly appearing layers promise to help researchers understand better how earth's atmosphere interacts with space, even potentially how those interactions help support life.

"This is an important discovery because we have never seen these dusk/dawn features before, and because these metal layers affect many things. The metals can fall into the ocean and act as fertilizer for ecosystems, the ionized metals can affect GPS radio signals," said Xinzhao Chu, CIRES Fellow, CU Boulder professor of Aerospace Engineering Sciences, and lead author of the new assessment.

It is the first time that the metal layers—which are not harmful to people—have been seen so regularly at these extreme heights in the atmosphere. Such high-altitude metal layers were discovered by Chu's group just 10 years ago above McMurdo, Antarctica, but there they occur more sporadically. Above Boulder, they're consistent, daily, and synched with winds that occur high in the atmosphere.

"Consistent daily patterns seen in our Boulder observations tell us that there are unknown processes at play, a golden opportunity for atmospheric scientists," said Jackson Jandreau who worked alongside Chu and Yingfei Chen in this study. Chen and Jandreau are both Ph.D. students in Chu's group.

The discovery also gives researchers a window into a crucial part of the atmosphere that is challenging to observe. It's a complicated region where interactions between the sun, earth and our planet's magnetic field can end up creating the environmental conditions in which surface life can thrive, protected from the harsh space

environment.

"There are metals in the atmospheres of other planetary bodies, such as Mars, and researchers look for Earth-like features on exoplanets as indicators for hospitable environments," Chu said. "Can these metal layers be one of these features?"

More information: *Xinzhao Chu et al, Mid-Latitude Thermosphere-Ionosphere Na (TINa) Layers Observed With High-Sensitivity Na Doppler Lidar Over Boulder (40.13°N, 105.24°W), Geophysical Research Letters (2021).* *DOI: 10.1029/2021GL093729*

https://wb.md/3q7BslV

Woman Donates Kidney to Hubby's Ex-Wife Days After Wedding

The tale of Jim Merthe and his two wives is a testament to how love and compassion can triumph over division

Kelli Kennedy, Associated Press

Fort Lauderdale, Fla. (AP) — Ten years after their first date, Debby Neal-Strickland put on a cream-colored lace gown and married her longtime sweetheart at their Florida church. Two days later, she put on a hospital gown and donated a kidney to Mylaen Merthe — her new husband's ex-wife.

Two days after Debby-Neal Strickland, front left, and Jim Merthe were married in November, Debby donated a kidney to James' ex-wife Mylaen Merthe, Center Back.

An unusual story? Yes. But the tale of Jim Merthe and his two wives is a testament to how love and compassion can triumph over division.

Mylaen, 59, had long struggled with kidney disease. By last year, she was ghostly pale with dark circles under her eyes, dragging herself through the workday with no energy. By the time she was admitted to the hospital in November, her kidneys were only functioning at 8%. Her brother offered to donate a kidney, but wasn't a match so Debby volunteered.

Jim and Mylaen have been divorced nearly two decades, but they got along well as they raised their two children, and as Jim fell in love with 56-year-old Debby. The women were friendly at family gatherings, though not especially close.

And Debby knew that Mylaen was about to become a grandmother for the first time — her daughter was pregnant.

She imagined Mylaen's daughter giving birth, "and her mom not being there. I just couldn't not try to change that," she said. "God told me, 'You're a match and you need to do this.'"

Giving is what Debby and Jim do. At their home in Ocala, they are raising six children — a 6-year-old girl with autism and five teenagers. Some are Debby's biological grandchildren and some they are fostering.

But Debby's desire to help Mylaen ran deeper. She spent years watching her brother die of cystic fibrosis while awaiting a double lung transplant. She offered one of her lungs, but she wasn't a match and he needed two. "When somebody needs an organ, if they don't get it, they're probably not going to make it. I know it's something that you do quickly," she said.

Debby passed the initial match for blood and tissue and began more complex testing while juggling a house full of kids — and at one point, toting a urine collection jug for 24 hours.

Mylaen tried desperately not to get her hopes up, focusing instead on her future as a grandmother.

Debby "knew that's all I ever wanted," she said. She "did it from her heart."

After months of testing and COVID delays, the transplant was set for two days after Jim and Debby's wedding. Debby was tempted to postpone the wedding, but friends discouraged her. The couple had already waited 10 years, canceling twice in deference to their

children who announced their own engagements.

They married Nov. 22. Jim wore a gray suit with a yellow shirt, "because he's my single yellow rose," Debby said.

"It was the most amazing day of my life, until two days later. That was also the most amazing day of my life," she said.

As soon as she regained consciousness, the new bride asked about Mylaen. A few floors below, Mylaen was also pleading with the nurses — "`I need to see her.' That was the first thing out of my mouth."

COVID-19 protocols were strict, but Jim was eventually allowed to wheel his new wife into his ex-wife's room.

"We had our masks on too, so we're crying, and of course our stomachs were hurting because of the incisions," Mylaen said. "We kinda laughed and cried."

Debby could already see the difference. The circles under Mylaen's eyes were gone, "she looked so alive and revitalized."

Mylaen moved in with her daughter, son-in-law and new baby Jackson to recuperate.

"I got to hold him and feed him," said Mylaen, who welcomed a second grandson in March. "I was like, 'I'm actually here to see this and I'm holding this little baby.'"

The women call themselves kidney sisters, pray for each other, coo over their grandbabies and are planning a big family trip to Lake Rabun, Georgia, this summer.

"This is what the world is about. Family. We need to stick together," Mylaen said. "She saved my life."

This story has been corrected to reflect that the husband's name is Jim Merthe, not Jim Strickland.

"One Good Thing" is a series that highlights individuals whose actions provide glimmers of joy in hard times — stories of people who find a way to make a difference, no matter how small. Read the collection of stories at https://apnews.com/hub/one-good-thing.

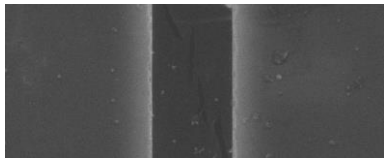
https://bit.ly/3clr7l1

This 2D Material Is Way Tougher Than Graphene, And Scientists Are Excited

A two-dimensional material with similar physical properties to graphene has now turned out to blow graphene out of the water in terms of toughness.

Michelle Starr

The material is called hexagonal boron nitride (h-BN), and it's so resistant to cracking that scientists are gobsmacked. The finding flies in the face of the fundamental description of fracture mechanics that scientists have been using to predict and define toughness since the 1920s.



Branching in the fracture of a h-BN sample. **(J. Lou/Rice University)**

"What we observed in this material is remarkable," said materials scientist Jun Lou of Rice University. "Nobody expected to see this in 2D materials. That's why it's so exciting."

Hexagonal boron nitride is actually extremely similar to graphene. The two materials both consist of hexagonal lattices of atoms. In the case of graphene, all those atoms are carbon; but for h-BN, each hexagon contains three boron atoms and three nitrogen atoms.

Carbon-carbon bonds are among the strongest in nature, so it's expected that graphene would be much stronger than h-BN. In general, that's true: The two materials have similar values for strength and elasticity, but h-BN's are slightly lower. Graphene has a strength of about 130 gigapascals for strength and 1.0 terapascals for elasticity; h-BN's values are 100 gigapascals and 0.8 terapascals respectively.

However, graphene also has a low resistance to cracks; in other words, it's remarkably brittle.

"We measured the fracture toughness of graphene seven years ago, and it's actually not very resistant to fracture," Lou explained. "If

you have a crack in the lattice, a small load will just break that material."

It was thought that, because h-BN's other properties are very similar to those of graphene, its brittleness would also be comparable - especially because graphene's brittleness was consistent with the Griffith theory of fracture, laid out by engineer Alan Arnold Griffith in 1921. He found that cracks will propagate when the stress placed on a material is greater than the force holding it together; and the difference in energy is released in the propagation of the crack.

When a team of researchers went to test this out, though, they found something really weird: h-BN's fracture resistance is 10 times higher than that of graphene's. That is definitely *not* consistent with the Griffith theory.

To find out why, the team applied stress to samples of h-BN, using scanning electron microscopy and transmission electron microscopy to observe in the smallest detail possible how the cracks occur. And, after over 1,000 hours of experimentation and the follow-up analysis, they figured it out.

, but they're not exactly the same. In graphene, a crack tends to zig-zag straight through the symmetrical hexagonal structure, from top to bottom. h-BN has a slight asymmetry in its hexagonal structure, due to the contrast in stress between the boron and the nitrogen, which means cracks tend to bifurcate.

This is what makes the material so much more resilient.

"If the crack is branched, that means it is turning," Lou said. "If you have this turning crack, it basically costs additional energy to drive the crack further. So you've effectively toughened your material by making it much harder for the crack to propagate."

This has implications for the development of flexible 2D materials for use in applications such as electronics. And h-BN already has a host of properties that make it an excellent prospect for these applications, including its heat resistance and chemical stability.

It could therefore provide a new way to develop technologies such as electronic textiles, stick-on electronic tattoos, and even implants.

"What makes this work so exciting is that it unveils an intrinsic toughening mechanism in a supposedly perfectly brittle material," said mechanician Huajian Gao of Nanyang Technological University in Singapore.

"Apparently, even Griffith couldn't foresee such drastically different fracture behaviors in two brittle materials with similar atomic structures." The research has been published in *Nature*.

https://bit.ly/3x2eaEy

Dunning-Kruger meets fake news

People who overrate their media savviness share more misleading material.

John Timmer

The Dunning-Kruger effect is one of the most famous—and predictable—biases in human behavior. It posits that people who don't understand a topic also lack sufficient knowledge to recognize that they don't understand it. Instead, these people know just enough to convince themselves that they completely grasp the topic, with results ranging from hilarious to painful.

Inspired by the widespread sharing of blatantly false news articles, a team of US-based researchers looked into whether Dunning-Kruger might be operating in the field of media literacy. Not surprisingly, people overestimate their ability to identify misleading news. But the details are complicated, and there's no obvious route to overcoming this bias.

Evaluating the news

Media literacy has the potential to limit the rapid spread of misinformation. Assuming people care about the accuracy of the things they like or share—something far from guaranteed—a stronger media literacy would help people evaluate if something is

likely to be accurate before pressing that share button. Assessing the credibility of sources is an essential part of that process.

Evaluating credibility is a skill—and it's one that people can clearly be bad at, leaving them open to the Dunning-Kruger effect. So the researchers arranged a set of experiments to determine whether Dunning-Kruger was an issue.

The basic test was straightforward. Relying on a couple of YouGov panels, the researchers gave the participants a set of actual headlines and asked the participants to rate them for accuracy. Without being told the test results, the participants were then asked to rate their own performance compared to the average person.

Assuming that people could rate themselves accurately, you'd expect that about half would rate themselves above average while the other half would rate themselves as below average. But that's nowhere close to what the researchers saw. Ninety percent of the participants estimated they were "above average in their ability to discern false and legitimate news headlines." The average self-reported ability outperformed 69 percent of other people.

On its own, this result could simply be representative of a general overconfidence. To determine whether the least competent were the most likely to overestimate their abilities, the researchers broke up participants into four groups based on their performance. The bottom quartile accurately judged accuracy about 10 percent of the time, and the top quartile was close to 90 percent accurate.

The top quartile also underestimated their own performance by about 15 percentage points. The above-average quartile were roughly accurate in terms of their self-assessment, and performance estimates went downhill from there. The lowest quartile showed a 40 percentage-point gap between their self-assessment and their actual performance. While the less competent didn't rate themselves as highly as the top performers, this is clearly a case of Dunning-Kruger.

In news that should surprise no one, men were more likely to have an inflated sense of their own media literacy. Republicans also fell into this category, which is not shocking given the high levels of misinformation about the election and the pandemic currently appearing on right-wing news sites.

Big mismatch, minor effects

While those are important findings on their own, the big question is how this inflated sense of competence influences people's decisions about consuming and sharing news reports. Here, the researchers benefitted from the YouGov panel, where several participants had agreed to share their browsing history anonymously (it was gathered by a combination of browser plugins and VPN service).

The researchers broke down visits to news and commentary sites based on whether the site had a history of spreading misinformation. In terms of exposure to misinformation, overconfidence was associated with a slight increase—in other words, the stronger the Dunning-Kruger effect, the more likely someone was to visit the sites that frequently post false stories. The effect, however, was minor. Those with the strongest misplaced confidence in their own abilities were only 6 percent more likely to view misinformation than those with a reasonable appraisal of their own skills.

A separate set of questions indicated that the misplaced confidence was associated with an increased willingness to share false stories, although the effect was fairly small. This willingness was influenced by whether the false story was consistent with people's political beliefs. Part of the problem is that people with overconfidence in their media savvy have a harder time discerning true and false stories than people with media skills.

Overall, we shouldn't be surprised that Dunning-Kruger applies to media literacy as well. And while the effects were small, if they replicate, they'll help improve our understanding of the misinformation landscape. The new research makes an interesting

comparison with an earlier study indicating that the average person is pretty good at recognizing misinformation but doesn't always bother to apply that skill before sharing or liking a story.

"Low performers genuinely believe in their own abilities"

The depressing part of the present research, however, is that there's a fair bit of literature on attempts to correct for Dunning-Kruger, and most of it describes failure. "Studies suggest that low performers genuinely believe in their own abilities and are not simply making face-saving expressions of self-worth," the researchers note, and they add that Dunning-Kruger is generally associated with "resistance to help, training, and corrections."

So even as we get a better grip on the factors influencing the misinformation flood we're facing, we're not necessarily getting closer to identifying what to do about it.

PNAS, 2021. DOI: 10.1073/pnas.2019527118 (About DOIs).

https://bit.ly/3cluisX

Mockingbird song decoded

Mockingbirds follow similar musical rules as those found in human music, from Beethoven to Kendrick Lamar

The North American mockingbird is famous for its ability to imitate the song of other birds. But it doesn't just mimic its kindred species, it actually composes its own songs based on other birds' melodies.

An interdisciplinary research team has now worked out how exactly the mockingbird constructs its imitations. The scientists determined that the birds follow similar musical rules as those found in human music, from Beethoven to Kendrick Lamar.

The mockingbird uses musical techniques like those of humans. **MPI for Empirical Aesthetics**

The song of the mockingbird is so complex that to investigate it required a joint effort of experts from very different fields. Neuroscientist Tina Roeske of the Max Planck Institute for Empirical Aesthetics, field biologist Dave Gammon of Elon University, and the music philosopher David Rothenberg of the New Jersey Institute of Technology combined their different approaches and areas of expertise to conduct this highly unusual study, the findings of which have just been published in the open-access journal *Frontiers in Psychology*.

Lead author Tina Roeske designed the algorithms used in testing the team's hypotheses.

"When you listen for a while to a mockingbird," she explains, "you can hear that the bird isn't just randomly stringing together the melodies it imitates. Rather, it seems to sequence similar snippets of melody according to consistent rules. In order to examine this hunch scientifically, however, we had to use quantitative analyses to test whether the data actually supported our hypotheses."

The results were unambiguous. The authors identified four compositional strategies that mockingbirds use in transitioning from one sound to the next: changing timbre, changing pitch, stretching the transition (lengthening it in time), and squeezing it (shortening it in time). The complex melodies they create are music to the ears not only of other birds but of humans as well. So, it should come as no surprise that (human) composers of varied musical styles use similar techniques in their work.

As co-author David Rothenberg explains in a YouTube video, the Tuvan throat singing group Huun-Huur-Tu presents examples of timbre change, and pitch change can be heard in the famous opening of Beethoven's Fifth Symphony; the song "Show Yourself" from the Disney film Frozen 2 itself shows the stretching of sound transitions; and if you listen very closely to Kendrick Lamar's song "Duckworth" from the album Damn, you'll hear transitions being squeezed, or shortened.

Original Publication:
Roeske, T. C., Rothenberg, D., & Gammon, D. E. (2021). Mockingbird Morphing Music: A Taxonomy of Transitions in a Complex Bird Song. Frontiers in Psychology, 12, 1143. doi:10.3389/fpsyg.2021.630115

https://bit.ly/3x2xnpF

Scientist identifies signaling underlying organ and limb regeneration

Discovery of differences in molecular signaling that promote regeneration in the axolotl while blocking it in the mouse

A team of scientists led by James Godwin, Ph.D., of the MDI Biological Laboratory in Bar Harbor, Maine, has come a step closer to unraveling the mystery of why salamanders can regenerate while adult mammals cannot with the discovery of differences in molecular signaling that promote regeneration in the axolotl, a highly regenerative salamander, while blocking it in the adult mouse. Godwin is pictured here with a tank containing an axolotl. Credit: MDI Biological Laboratory

Many salamanders can readily regenerate a lost limb, but adult mammals, including humans, cannot. Why this is the case is a scientific mystery that has fascinated observers of the natural world for thousands of years.

Now, a team of scientists led by James Godwin, Ph.D., of the MDI Biological Laboratory in Bar Harbor, Maine, has come a step closer to unraveling that mystery with the discovery of differences in molecular signaling that promote regeneration in the axolotl, a highly regenerative salamander, while blocking it in the adult mouse, which is a mammal with limited regenerative ability.

"Scientists at the MDI Biological Laboratory have been relying on comparative biology to gain insights into human health since its founding in 1898," said Hermann Haller, M.D., the institution's president. "The discoveries enabled by James Godwin's comparative studies in the axolotl and mouse are proof that the idea of learning from nature is as valid today as it was more than one hundred and twenty years ago."

Instead of regenerating lost or injured body parts, mammals typically form a scar at the site of an injury. Because the scar creates a physical barrier to regeneration, research in regenerative medicine at the MDI Biological Laboratory has focused on understanding why the axolotl doesn't form a scar—or, why it doesn't respond to injury in the same way that the mouse and other mammals do.

"Our research shows that humans have untapped potential for regeneration," Godwin said. "If we can solve the problem of scar formation, we may be able to unlock our latent regenerative potential. Axolotls don't scar, which is what allows regeneration to take place. But once a scar has formed, it's game over in terms of regeneration. If we could prevent scarring in humans, we could enhance quality of life for so many people."

The axolotl as a model for regeneration

The axolotl, a Mexican salamander that is now all but extinct in the wild, is a favorite model in regenerative medicine research because of its one-of-a-kind status as nature's champion of regeneration. While most salamanders have some regenerative capacity, the axolotl can regenerate almost any body part, including brain, heart, jaws, limbs, lungs, ovaries, spinal cord, skin, tail and more.

Since mammalian embryos and juveniles have the ability to regenerate—for instance, human infants can regenerate heart tissue and children can regenerate fingertips—it's likely that adult mammals retain the genetic code for regeneration, raising the prospect that pharmaceutical therapies could be developed to encourage humans to regenerate tissues and organs lost to disease or injury instead of forming a scar.

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In his recent research, Godwin compared immune cells called macrophages in the axolotl to those in the mouse with the goal of identifying the quality in axolotl macrophages that promotes regeneration. The research builds on earlier studies in which Godwin found that macrophages are critical to regeneration: When they are depleted, the axolotl forms a scar instead of regenerating, just like mammals.

The recent research found that although macrophage signaling in the axolotl and in the mouse were similar when the organisms were exposed to pathogens such as bacteria, funguses and viruses, when it came to exposure to injury it was a different story: The macrophage signaling in the axolotl promoted the growth of new tissue while that in the mouse promoted scarring.

The paper on the research, entitled "Distinct TLR Signaling in the Salamander Response to Tissue Damage" was recently published in the journal *Developmental Dynamics*. In addition to Godwin, authors include Nadia Rosenthal, Ph.D., of The Jackson Laboratory; Ryan Dubuque and Katya E. Chan of the Australian Regenerative Medicine Institute (ARMI); and Sergej Nowoshilow, Ph.D., of the Research Institute of Molecular Pathology in Vienna, Austria.

Godwin, who holds a joint appointment with The Jackson Laboratory, was formerly associated with ARMI and Rosenthal is ARMI's founding director. The MDI Biological Laboratory and ARMI have a partnership agreement to promote research and education on regeneration and the development of new therapies to improve human health.

Specifically, the paper reported that the signaling response of a class of proteins called toll-like receptors (TLRs), which allow macrophages to recognize a threat such an infection or a tissue injury and induce a pro-inflammatory response, were "unexpectedly divergent" in response to injury in the axolotl and the mouse. The finding offers an intriguing window into the mechanisms governing regeneration in the axolotl.

Being able to 'pull the levers of regeneration'

The discovery of an alternative signaling pathway that is compatible with regeneration could ultimately lead to regenerative medicine therapies for humans. Though regrowing a human limb may not be realistic in the short term, significant opportunities exist for therapies that improve clinical outcomes in diseases in which scarring plays a major role in the pathology, including heart, kidney, liver and lung disease.

"We are getting closer to understanding how axolotl macrophages are primed for regeneration, which will bring us closer to being able to pull the levers of regeneration in humans," Godwin said. "For instance, I envision being able to use a topical hydrogel at the site of a wound that is laced with a modulator that changes the behavior of human macrophages to be more like those of the axolotl."

Godwin, who is an immunologist, chose to examine the function of the immune system in regeneration because of its role in preparing the wound for repairs as the equivalent of a first responder at the site of an injury. His recent research opens the door to further mapping of critical nodes in TLR signaling pathways that regulate the unique immune environment enabling axolotl regeneration and scar-free repair.

More information: *Ryan J. Debuque et al, Distinct toll-like receptor signaling in the salamander response to tissue damage, Developmental Dynamics (2021). DOI: 10.1002/dvdy.340*

https://bit.ly/2SdjC8T

The dream team: Scientists find drug duo that may cure COVID-19 together

Preclinical experiments show that the drugs cepharanthine and nelfinavir may be effective treatments for COVID-19

COVID-19 continues to claim lives across the world and is infecting millions more. Although several vaccines have recently become available, making significant strides towards preventing COVID-19, what about the treatment of those who already have the infection? Vaccines aren't 100% effective, highlighting the need--now more than ever--for effective antiviral therapeutics. Moreover, some people can't receive vaccines due to health issues, and new variants of SARS-CoV-2, the virus that causes COVID-19, that can penetrate vaccine-conferred immunity, are being reported, indicating that we need to think beyond prevention.

Given this need, a team of researchers based in Japan, the US, and the UK launched a project to develop effective therapeutics. This team included several researchers based at Tokyo University of Science: Visiting Professor Koichi Watashi, Dr. Hirofumi Ohashi, Professor Shin Aoki, Professor Kouji Kuramochi, and Assistant Professor Tomohiro Tanaka. Their goal was clear and simple: finding a cure for COVID-19.

To achieve this goal, the researchers first established an experimental system for screening drugs that may help to control infections. This system used a type of cells called VeroE6/TMPRSS2 cells, which were manipulated to efficiently be infected with and produce SARS-CoV-2. "To determine whether a drug of interest could help combat infection by SARS-CoV-2, we simply had to expose VeroE6/TMPRSS2 cells to both the drug and SARS-CoV-2 and then observe whether the drug's presence served to hinder the virus's efforts to infect cells," explains Professor Watashi.

The researchers used this experimental system to screen a panel of drugs that are already approved for clinical use, including drugs like remdesivir and chloroquine that have already being approved or are being trialed as treatments for COVID-19. In an exciting outcome, the researchers found two drugs that provided effective SARS-CoV-2 suppression: cepharanthine, which is used to treat inflammation, and nelfinavir, which is approved for the treatment of HIV infection.

Cepharanthine inhibited the entry of the virus into cells by preventing the virus from binding to a protein on the cell membrane, which it uses as a gateway. In contrast, nelfinavir worked to prevent the virus from replicating inside the cell by inhibiting a protein that the virus relies on for replication. Given that these drugs have distinct antiviral mechanisms, using both of them together could be especially effective for patients, with computational models predicting that combined cepharanthine/nelfinavir therapy can hasten the clearance of SARS-CoV-2 from a patient's lungs by as few as 4.9 days.

So, does this mean we will be seeing these new drugs in COVID-19 treatment centers? Of course, the drug duo isn't ready to be rolled out into healthcare systems just yet. These findings justify further research into the clinical potential of cepharanthine/nelfinavir therapy, and only following this can we say for sure that it is useful and helpful.

Nevertheless, given the ongoing nature of the COVID-19 pandemic and the ever-increasing death toll, the development of cepharanthine/nelfinavir therapy may provide clinicians and patients with a much-needed new treatment option.

Reference

Title of original paper: Potential anti-COVID-19 agents, cepharanthine and nelfinavir, and their usage for combination treatment Journal: iScience
DOI: https://doi.org/10.1016/j.isci.2021.102367
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https://bit.ly/2S3KiJt

Researchers: Culture drives human evolution more than genetics

In a new study, University of Maine researchers found that culture helps humans adapt to their environment and overcome challenges better and faster than genetics.

After conducting an extensive review of the literature and evidence of long-term human evolution, scientists Tim Waring and Zach Wood concluded that humans are experiencing a "special evolutionary transition" in which the importance of culture, such as learned knowledge, practices and skills, is surpassing the value of genes as the primary driver of human evolution.

Culture is an under-appreciated factor in human evolution, Waring says. Like genes, culture helps people adjust to their environment and meet the challenges of survival and reproduction. Culture, however, does so more effectively than genes because the transfer of knowledge is faster and more flexible than the inheritance of genes, according to Waring and Wood.

Culture is a stronger mechanism of adaptation for a couple of reasons, Waring says. It's faster: gene transfer occurs only once a generation, while cultural practices can be rapidly learned and frequently updated. Culture is also more flexible than genes: gene transfer is rigid and limited to the genetic information of two parents, while cultural transmission is based on flexible human learning and effectively unlimited with the ability to make use of information from peers and experts far beyond parents. As a result, cultural evolution is a stronger type of adaptation than old genetics.

Waring, an associate professor of social-ecological systems modeling, and Wood, a postdoctoral research associate with the School of Biology and Ecology, have just published their findings in a literature review in the *Proceedings of the Royal Society B*, the flagship biological research journal of The Royal Society in London.

"This research explains why humans are such a unique species. We evolve both genetically and culturally over time, but we are slowly becoming ever more cultural and ever less genetic," Waring says.

Culture has influenced how humans survive and evolve for millenia. According to Waring and Wood, the combination of both culture and genes has fueled several key adaptations in humans such as reduced aggression, cooperative inclinations, collaborative abilities and the capacity for social learning. Increasingly, the researchers suggest, human adaptations are steered by culture, and require genes to accommodate.

Waring and Wood say culture is also special in one important way: it is strongly group-oriented. Factors like conformity, social identity and shared norms and institutions—factors that have no genetic equivalent—make cultural evolution very group-oriented, according to researchers. Therefore, competition between culturally organized groups propels adaptations such as new cooperative norms and social systems that help groups survive better together.

According to researchers, "culturally organized groups appear to solve adaptive problems more readily than individuals, through the compounding value of social learning and cultural transmission in groups." Cultural adaptations may also occur faster in larger groups than in small ones.

With groups primarily driving culture and culture now fueling human evolution more than genetics, Waring and Wood found that evolution itself has become more group-oriented.

"In the very long term, we suggest that humans are evolving from individual genetic organisms to cultural groups which function as superorganisms, similar to ant colonies and beehives," Waring says. "The 'society as organism' metaphor is not so metaphorical after all. This insight can help society better understand how individuals can fit into a well-organized and mutually beneficial system. Take the coronavirus pandemic, for example. An effective national epidemic response program is truly a national immune system, and we can therefore learn directly from how immune systems work to improve our COVID response."

More information: *Timothy M. Waring et al, Long-term gene–culture coevolution and the human evolutionary transition, Proceedings of the Royal Society B: Biological Sciences (2021). DOI: 10.1098/rspb.2021.0538*

https://bit.ly/3ik9vtC

2 types of flu viruses may have gone extinct

There's been so little flu transmission during the COVID-19 pandemic that some types of flu viruses may have disappeared.

By Rachael Rettner - Senior Writer

There's been so little flu transmission during the COVID-19 pandemic that some types of flu viruses may have gone extinct, according to news reports.

During the COVID-19 pandemic, flu cases dropped to historic lows — a phenomenon experts attribute to mask wearing and other precautions to combat the novel coronavirus.

Interestingly, two types of flu viruses haven't shown up on anyone's radar for a year, meaning there have been no reported cases of these viruses anywhere in the world, STAT reported. Experts don't yet know if these types have gone extinct, but if so, officials could have an easier time picking the strains of flu viruses included in the seasonal flu shot, STAT reported.

To explain which flu viruses may have gone extinct, it helps to understand how flu viruses are classified. Two families of flu viruses cause seasonal flu: influenza A and influenza B. Influenza A viruses are divided into "subtypes" based on two proteins on their surface known as hemagglutinin (H) and neuraminidase (N), according to the Centers for Disease Control and Prevention (CDC). Currently, H1N1 and H3N2 circulate in people, and each of these subtypes is further broken down into "clades."

Influenza B viruses, on the other hand, don't have subtypes or clades but are divided into two lineages known as B/Yamagata and B/Victoria.

One clade of H3N2, known as 3c3.A, hasn't been detected since March 2020. The same is true of the lineage B/Yamagata, according to STAT.

"I think it has a decent chance that it's gone. But the world's a big place," Trevor Bedford, a computational biologist at the Fred Hutchinson Cancer Research Center in Seattle, told STAT, referring to the H3N2 clade.

Florian Krammer, a virologist at the Icahn School of Medicine Mount Sinai in New York, shared similar thoughts about the B/Yamagata lineage. "Just because nobody saw it doesn't mean it has disappeared completely, right? But it could" have disappeared, Krammer told STAT.

Less diversity among flu viruses would be a good thing. Each year, scientists make the flu vaccine months before flu season actually starts by seeing what strains are circulating in the world and then predicting which flu strains are likely to be the most common during the upcoming season. Lower flu virus diversity means a smaller pool of circulating viruses to choose from and a greater chance that the strains in the shot will match those circulating.

H3N2 viruses are a particularly diverse group, and prior to the COVID-19 pandemic, their clades seemed to be getting more genetically diverse each year, according to STAT. So a drop in diversity for this subtype would be a "great thing," Richard Webby, director of the World Health Organization Collaborating Center for

Studies on the Ecology of Influenza in Animals and Birds, based at St. Jude Children's Hospital in Memphis, told STAT. "Currently, when we sit down to make recommendations for vaccine strains, it's always the 'headache' virus."

Webby cautioned that these virus types might still be out there even if they haven't been reported in official databases. But the dramatic drop in flu cases this year is likely to bring some changes for flu.

"Without doubt, this is definitely going to change something in terms of the diversity of flu viruses out there," Webby told STAT. "The extent to which it changes and how long it stays changed are the big question marks. But we have never seen this before."

https://bit.ly/3qfaTei

Electrochemical cell harvests lithium from seawater

KAUST researchers have developed a method to extract lithium, a vital element in autonomous vehicle batteries, from seawater in a more economically viable way.

Lithium is a vital element in the batteries that power electric vehicles, but soaring lithium demand is expected to exhaust land-based reserves by 2080. KAUST researchers have now developed an economically viable system that can extract high-purity lithium from seawater.

The oceans contain about 5,000 times more lithium than the land but at extremely low concentrations of about 0.2 parts per million (ppm). Larger ions, including sodium, magnesium and potassium, are all present in seawater at much higher concentrations; however, previous research efforts to tease lithium from this mixture have yielded little.

The KAUST team solved this problem with an electrochemical cell containing a ceramic membrane made from lithium lanthanum titanium oxide (LLTO). Its crystal structure contains holes just wide enough to let lithium ions pass through while blocking larger metal ions. "LLTO membranes have never been used to extract and concentrate lithium ions before," says postdoc Zhen Li, who developed the cell.

The cell contains three compartments. Seawater flows into a central feed chamber, where positive lithium ions pass through the LLTO membrane into a side compartment that contains a buffer solution and a copper cathode coated with platinum and ruthenium. Meanwhile, negative ions exit the feed chamber through a standard anion exchange membrane, passing into a third compartment containing a sodium chloride solution and a platinum-ruthenium anode.

The researchers tested the system using seawater from the Red Sea. At a voltage of 3.25V, the cell generates hydrogen gas at the cathode and chlorine gas at the anode. This drives the transport of lithium through the LLTO membrane, where it accumulates in the side-chamber. This lithium-enriched water then becomes the feedstock for four more cycles of processing, eventually reaching a concentration of more than 9,000 ppm. Adjusting the pH of this solution delivers solid lithium phosphate that contains mere traces of other metal ions—pure enough to meet battery manufacturers' requirements.

The researchers estimate that the cell would need only US$5 of electricity to extract 1 kilogram of lithium from seawater. The value of hydrogen and chlorine produced by the cell would more than offset this cost, and residual seawater could also be used in desalination plants to provide freshwater.

"We will continue optimizing the membrane structure and cell design to improve the process efficiency," says group leader Zhiping Lai. His team also hopes to collaborate with the glass industry to produce the LLTO membrane at large scale and affordable cost.

More information: *Zhen Li et al, Continuous electrical pumping membrane process for seawater lithium mining, Energy & Environmental Science (2021).* *DOI: 10.1039/D1EE00354B*

https://bit.ly/2RuB8oY

The world saw a shark-pocalypse 19 million years ago, and we don't know why

Researchers find evidence of a huge shark die-off but aren't sure what happened.

Doug Johnson

Sharks have been swimming and hunting in the world's oceans for 450 million years, and though their numbers have recently declined because of human activity, they're still with us. But the world once had many more, and many more varieties of, the large marine predators compared to today. In fact, new research published in Science suggests that 19 million years ago, the vast majority of sharks and shark species died off. We don't understand why or how this large extinction event occurred.

“Sharks have... weathered a large number of mass extinctions. And this extinction event is probably the biggest one they've ever seen. Something big must have happened,” Elizabeth Sibert, one of the authors of the paper, told Ars.

Sibert is a Hutchinson postdoctoral fellow at the Yale Institute for Biospheric Sciences, and she was a junior fellow in the Harvard Society of Fellows for the initial phases of this research back in 2017.

Back then, the team analyzed ancient sediment core samples, one from the South Pacific and one from the North. The International Ocean Discovery Program collected these samples in 1983 and 1992, but the material they contain dates back hundreds of millions of years. Each centimeter down on the cores represents a few 100,000 years back in time.

Embedded in the sample were 1,381 tiny shark scale, or denticle, fossils. The team looked at the raw number of scales and the different types of scales that appeared in the different layers of sediment. "Dermal denticles offer an incredible window into the past of these ancient and elusive marine predators and thus the state of ocean ecosystems through time,” Leah Rubin, another author on the paper, told Ars.

Sharp decline in sharks

Prior to 19 million years ago, the researchers found a wealth of shark biodiversity and abundance. But after that point, they saw a stark decrease in the number of scale fossils and fewer varieties of them. In all, there was a 90 percent decrease in terms of raw population and a 70 percent decrease in species diversity. Sharks never really recovered to the dizzying highs of pre-history.

Though the sediment cores are from the Pacific, Sibert suspects that the team's findings could hold true for other parts of the deep. According to Sibert, some core samples from the Atlantic Ocean show an abundance of shark life 30 million years ago. There are also more recent samples, from only a few million years ago, that similarly show a decline—but so far, there are no Atlantic samples from the timeframe of the extinction event.

Whatever led to the shark-pocalypse is still unknown. The oxygen and carbon isotopes—which are used to reconstruct what the temperatures and carbon cycles were like in the past—don't show anything amiss. In fact, they were so normal that researchers haven't spent much time studying 19 million years ago. However, Sibert noted that with more research and more sediment samples, the mystery is quite likely to be solved.

“One of the challenges with this particular bit of research is 'what happened to the sharks at this time and why was there this massive die-off?' The answer is 'we really don't know right now,'” she said, adding that the team hopes to look into how the die-off impacted other oceanic species.

We’re gonna need a bigger data set

According to Seth Finnegan, associate professor at the University of California, Berkeley's department of Integrative Biology, the

paper's findings are intriguing, but they rely on only two samples. He noted that it is also possible that the large shark die-off only happened in the Northern and Southern Pacific. But that's probably not the case, as something affecting one part of the ocean will usually affect others, he said.

All the same, Finnegan noted that to get a clearer picture of what happened 19 million years ago, more samples from other parts of the ocean, and places closer to shore, would be helpful. “There are multiple levels of uncertainty here, but it's a very interesting and striking pattern. It's not subtle,” he told Ars.

It's too early to say how this research fits into our understanding of history, Finnegan said. But the study shows that sharks have been around for a long time and have seen some pretty staggering biodiversity swings. Future research into the impacts that this shark die-off had on other creatures could also outline the importance of shark conservation today. According to Finnegan, sharks are an essential part of their ecosystems, and having large swaths of them kick the bucket could produce impacts that we don't yet fully understand.

“They tend to be very important apex predators in a lot of ecosystems, very important in regulating ecosystem structures,” he said.

Among well-studied species, stumbling onto a large extinction event is quite rare, Finnegan said. However, considering fossilized shark denticles have not been thoroughly studied relative to other fossils, it's perhaps not that surprising to come across a previously undiscovered die-off.

There could be other extinction events throughout history that scientists simply haven't discovered yet, Sibert told Ars. Even today, researchers might come across some other ancient surprises. For example, her team began the work looking for background information on fish and sharks around 80 million years in the past—instead, they came across a doomsday event for the ocean predators.

“To me, that's something that's really fascinating and really exciting. If you go looking, there are probably all sorts of things we don't know about the Earth and its history.”

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https://wb.md/3iiqRao

Med Ed Is 'Rotten,' Says Outspoken Doc on a Quest for Reform

Bryan Carmody is fed up with licensing examinations.

Benjamin Mazer, MD, MBA

Disclosures June 04, 2021

He thinks that the National Board of Medical Examiners (NBME) has unacceptable financial conflicts of interest. He doesn't believe that osteopathic licensing exams should even exist. In Carmody's view, the Association of American Medical Colleges (AAMC) uses its crash-prone residency application website as a cash cow. He has no interest in working his way up through formal medical education committees just to "sit at a table where I could have a polite discussion with the CEOs of these organizations."

Instead, he has taken on these organizations as an outsider. In just a little over 2 years he has become a nationally recognized expert, one whom even the very organizations he routinely criticizes now acknowledge as an influential voice in medical education policy.

See more at the site . . .

https://bit.ly/3fTq9pd

Genius 'Shield' Implant For Diabetes Treatment Shows Great Results in Mouse Study

US researchers developing nano-fiber implant that can shield patient's own insulin-producing cells from their immune system

Mike McRae

Type 1 diabetes is fundamentally a disease of the immune system.

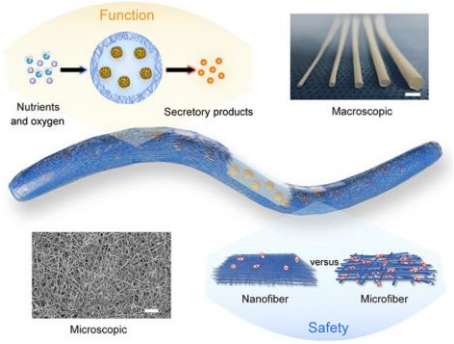
At some point, for some reason, the body's defenses destroy insulin-producing tissues in the pancreas, making it all but impossible to fine-tune the flow of glucose into cells.

Returning the pancreas to a state of functionality would give diabetics a new lease on life, yet for all the progress we've made in tissue-replacement, safely taming a traitorous immune system has been an insurmountable obstacle.

There are finally signs we might be able to clear that hurdle, with US researchers and engineers developing a nano-fiber implant that can shield a patient's own insulin-producing cells from their immune system.

Early results are encouraging, with tests in mice demonstrating it could be an effective way to treat type 1 diabetes using actual pancreatic tissue, without the need for immunosuppressive drugs.

"The device, which is about the width of a few strands of hair, is micro-porous – with openings too small for other cells to squeeze into – so the insulin-secreting cells consequently can't be destroyed by immune cells, which are larger than the openings," says medical researcher Jeffrey R. Millman from Washington University.



(Wang et al., Sci Trans Med, 2021)

For nearly a century type 1 diabetes has been treated through timely injections of the glucose-mediating hormone insulin, a process that has undoubtedly saved countless lives.

Yet getting the perfect amount of insulin from a bottle into the body is neither comfortable nor risk-free. Getting the dosage wrong could mean a life-threatening medical emergency.

While modern digital technology has made amazing progress towards mirroring an authentic pancreas, we're still a long way off matching biology's ability to meter out the right amount of hormones right where they're needed.

Advances in converting 'blank' stem cells into virtually any other cell in the body has allowed researchers to recreate a person's insulin-secreting 'islet' tissues using little more than a sample of their own cells.

Making them is one thing - transplanting them into the body without attracting unwanted attention from the person's own immune system is something else entirely.

"The problem is that in people with type 1 diabetes, the immune system attacks those insulin-secreting cells and destroys them," says Millman.

"To deliver those cells as a therapy, we need devices to house cells that secrete insulin in response to blood sugar, while also protecting those cells from the immune response."

Specially designed implants for shielding islet tissues from the ravages of the body's immune system aren't novel concepts. Some work better than others, reducing risks of scarring or providing oxygen or nutrients to extend the life of the implanted tissues.

One material with huge potential in encapsulating tissue implants is based on a polysaccharide found in the cell walls of algae, called alginate.

Its ability to avoid triggering an immune response itself makes it a suitable candidate. The challenge for researchers was to turn it into a capsule that could be removed periodically to replace the exhausted pancreatic tissue inside.

This led to the development of TRAFFIC – a thread-reinforced alginate fiber for islet encapsulation. If not for the fact the alginate is prone to swelling and breaking over time, it might have been a winning solution.

Millman and his team have now improved on TRAFFIC by weaving a medical-grade thermoplastic around an alginate hydrogel

core, giving it the right mix of stealth, robustness, and porosity.

Tested in diabetic mice, the 'nanofiber-integrated cell encapsulation' (NICE) device helped the animals maintain their glucose levels for up to 200 days using human islet cells. The devices also scaled up well for implanting and retrieval in dogs.

It's promising progress, though solutions such as these can't come soon enough for the roughly one in every 7,000 people with a type 1 diabetes diagnosis.

"The device we used in these experiments protected the implanted cells from the mice's immune systems, and we believe similar devices could work the same way in people with insulin-dependent diabetes," says Millman.

This research was published in *Science Translational Medicine*.

https://bit.ly/3qhnvlp

Early adopters of Chinese vaccines see case surges; China plows ahead anyway

China is now giving 20 million doses a day despite low efficacy.

Beth Mole

Despite a sluggish start, China is now vaccinating its people against COVID-19 at an impressive clip, currently averaging nearly 20 million doses administered per day. As of Friday, the country had given more than 720 million vaccinations since mid-December, with nearly 400 million of those were given in May alone.

The dramatic ramp up comes at an awkward time, however. Early adopters of China's vaccines have seen dramatic surges in COVID-19 cases—despite high vaccination rates—and are now backing away from the country's offerings.

Vials of the Sinopharm vaccine in Beijing on June 1. **Getty | Xinhua News Agency**

In Bahrain, for instance, officials are now offering high-risk people who have already received two doses of China's Sinopharm vaccine a third vaccine dose—but one made by Pfizer-BioNTech. The apparent vote of no confidence by officials is striking: Bahrain was one of the first countries to back and rollout Sinopharm's vaccine, and it has had a highly successful vaccination campaign. Nearly 58 percent of the Persian Gulf country has received at least one dose of a vaccine, and most of the vaccines given in Bahrain are from Sinopharm. But the country is now seeing its worst wave of COVID-19 yet and the government has recently issued a two-week lockdown to try to get transmission under control.

The Seychelles went through a similar struggle. The archipelago saw a dramatic spike in cases in mid-May, despite having around 70 percent of its population vaccinated with at least one dose. Like Bahrain, the Seychelles had largely relied on the Sinopharm vaccine.

Dubai, which has also relied on Sinopharm's vaccine, is now quietly offering residents who have been fully vaccinated with the Sinopharm vaccine the opportunity to get re-vaccinated with the Pfizer-BioNTech vaccine, according to The Wall Street Journal.

Efficacy "not high"

In a study published on May 26 in JAMA, Sinopharm researchers reported results suggesting that their inactivated virus vaccine was up to 78 percent effective against symptomatic COVID-19 cases. But the study was done mainly in young, healthy men, and the results were not conclusive regarding whether the vaccine was effective against severe disease or asymptomatic cases.

Unpublished data out of Serbia suggested that some people given the vaccine may not produce antibodies to fight off the pandemic coronavirus three months after vaccination, according to reporting by the Wall Street Journal. "The Sinopharm vaccine is not immunogenic enough, and it appears that its impact is especially low on elderly recipients," said Olgica Djurkovic-Djakovic, of the

University of Belgrade, who led the unpublished study and shared the findings with the Journal.

In April, the head of China's Centers for Disease Control and Prevention, George Gao, seemed to acknowledge this potential problem. "The efficacy of the existing vaccines is not high," he said at a conference, discussing the country's vaccines. Last month, Beijing reportedly began planning to offer third doses of the country's vaccines to try to boost protection.

Still, last month the World Health Organization granted an emergency use listing (EUL) for Sinopharm's COVID-19 vaccine, paving its way for global use. Just on Tuesday, the WHO granted an EUL to China's Sinovac vaccine. Like Sinopharm's vaccine, Sinovac's vaccine is an inactivated virus vaccine, and the two appear to have similar track records. Both Chile and Uruguay rolled Sinovac's vaccine into their mass vaccination campaigns and have seen subsequent spikes in cases.

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How a Vietnamese raw pork snack could help us keep food fresh, naturally

Fermented meat snack is helping researchers develop a safe, all-natural food preservative

A traditional Vietnamese meat snack could hold the key to developing a safe and natural food preservative, addressing the twin global problems of food waste and food-borne illnesses. The fermented pork snack, Nem Chua, is eaten raw but does not cause food poisoning when prepared correctly.

Vietnamese fermented pork snack, Nem Chua. **Credit: RMIT University**

This is because friendly bacteria that thrive in the fermented meat make a special compound that destroys more dangerous bacteria.

Now researchers at RMIT University in Melbourne, Australia, have shown how this natural bacteria-killing compound could be used to keep food fresh for longer.

Food waste is a global issue that costs around $US680 billion annually in industrialised countries, consumes nearly a quarter of the water used in agriculture and produces 8% of global greenhouse emissions. Food-borne diseases like Listeria or Salmonella affect millions each year and can be life threatening for pregnant women, older people and those who are immunocompromised.

Co-lead researcher Professor Oliver Jones said changes in consumer habits have led to a greater demand for natural alternatives to artificial food preservatives.

"Scientists have known about these bacteria-killing compounds for many years but the challenge is to produce them in large enough quantities to be used by the food industry," said Jones, Associate Dean of Biosciences and Food Technology at RMIT.

"The Nem Chua compound is colourless, odourless, tasteless and very resilient. "Through this new research, we've identified the right growth conditions that would enable us to make it in large amounts, potentially at industrial scales. "With further development, we hope this could be an effective, safe and all-natural solution for both food waste and food-borne disease."

Bacteria-killing weapon

A team of RMIT researchers was inspired to investigate Nem Chua for its potential antibacterial properties after travelling to Vietnam and observing people eating the raw meat snack without getting sick, despite the hot and humid climate.

The team, led by Professor Andrew Smith (now at Griffith University) and Dr Bee May, discovered a new type of bacteria-killing compound in Nem Chua.

Plantacyclin B21AG is one of a group of compounds known as bacteriocins, which are produced by bacteria to destroy rival

bacterial strains.

Bacteriocins form holes in the membranes of target bacteria. This causes the contents of the cell to leak out - effectively killing the bacteria. The problem is most bacteriocins only work against one or two types of bacteria and they are not very stable in different environmental conditions.

Only one - Nisin, which came to market in the 1960s - is currently licensed for use as a food preservative, in a market estimated to be worth more than $US513 million in 2020, but this compound is temperature and pH sensitive limiting its use.

Tough and effective

The Nem Chua-derived compound is more robust than Nisin and is effective against a wide range of bacteria even after exposure to a range of environments typical in food processing. It can survive being heated to 90C for 20 minutes and remains stable across high and low pH levels.

The compound can also destroy a range of disease-causing organisms commonly found in food including potentially life-threating Listeria, which can survive refrigeration and even freezing.

Co-lead researcher Dr Elvina Parlindungan, who completed the new study as part of her PhD research at RMIT, is now a postdoctoral fellow at APC Microbiome, part of University College Cork in Ireland.

"Using bacteriocins as food preservatives effectively means we are turning bacteria's own toxic weapons against them - harnessing nature's smart solutions to tackle our big challenges," Parlindungan said. "In the future, these compounds might also be useful as an antibiotic in human medicine."

Researchers at RMIT's School of Science have begun experimenting with methods to further purify the compound and are planning to incorporate it into test food products.

The team is keen to collaborate with potential industry partners to further develop the technology.

This work was supported by a PhD scholarship from the Indonesian Endowment Fund for Education (LPDP), part of the Ministry of Finance of the Republic of Indonesia, awarded to Parlindungan.

'Factors that influence growth and bacteriocin production in Lactiplantibacillus plantarum B21,' with co-author Dr Chaitali Dekiwadia (RMIT Microscopy and Microanalysis Facility), is published in Process Biochemistry (DOI:?10.1016/j.procbio.2021.05.009).

https://n.pr/3ijLnrf

A New Type Of COVID-19 Vaccine Could Debut Soon

A new kind of COVID-19 vaccine could be available as soon as this summer.

It's what's known as a protein subunit vaccine. It works somewhat differently from the current crop of vaccines authorized for use in the U.S. but is based on a well-understood technology and doesn't require special refrigeration.

In general, vaccines work by showing people's immune systems something that looks like the virus but really isn't. Consider it an advance warning; if the real virus ever turns up, the immune system is ready to try to squelch it. In the case of the coronavirus, that "something" is one of the proteins in the virus — the spike protein.

The vaccines made by Johnson & Johnson, Moderna and Pfizer contain genetic instructions for the spike protein, and it's up to the cells in our bodies to make the protein itself.

The first protein subunit COVID-19 vaccine to become available will likely come from the biotech company, Novavax. In contrast to the three vaccines already authorized in the U.S., it contains the spike protein itself — no need to make it, it's already made — along with an adjuvant that enhances the immune system's response, to make the vaccine even more protective.

Protein subunit vaccines made this way have been around for a while. There are vaccines on the market for hepatitis B and pertussis based on this technology.

Article continues after sponsor message

A large test of the Novavax COVID-19 vaccine's effectiveness, conducted in tens of thousands of volunteers in the United States and Mexico, is about to wrap up. Dr. Gregory Glenn, president of research and development for Novavax, told an audience at a recent webinar hosted by the International Society for Vaccines that "we anticipate filing for authorization in the U.K., U.S. and Europe in the third quarter."

Turning plants into factories

To make the virus protein, Novavax uses giant vats of cells grown in the lab. But there's another way to make the protein: Get plants in a greenhouse to do it. That's the approach being used by the Canadian biotech firm Medicago.

The plants used are related to the tobacco plant, and have been modified to contain the genetic instructions to make the viral protein. The plants do something very valuable — they make a lipid shell that surrounds a bunch of the viral proteins, with the proteins sticking out.

"The plant will assemble the protein in a shape and form that is looking like the virus," says Nathalie Landry, Medicago's executive vice president for scientific and medical affairs. "So, if you look at an image of it, it *looks* like a virus, but it cannot induce any disease. But when [it's] injected as a vaccine your body will raise a good immune response."

Early studies suggest Medicago's candidate vaccine does just that, and the company is confident enough in those findings that it's already begun a large study in people that could involve as many as 30,000 volunteers in 11 countries.

Landry acknowledges that development of the Medicago COVID-19 vaccine has lagged behind others. "We're a latecomer, but we're coming," she says.

Another latecomer that's coming is the pharmaceutical giant Sanofi. Its protein subunit vaccine against the coronavirus is also grown in cells in the lab.

Late last year the company was getting ready to mount a large study of the vaccine's effectiveness when the early results in a smaller group of people showed it did not seem to be inducing the immune response that would be protective.

"Especially in elderly individuals in that study, it was not as immunogenic as it should be," says Dr. Paul Goepfert at the University of Alabama at Birmingham, who was one of the researchers involved in those early studies. He says the issue turned out to be an incorrect calculation of the dose of vaccine being delivered. "So instead of giving 10 micrograms of the dose, they were actually giving one microgram," Goepfert says.

Sanofi has fixed that problem and repeated the early studies with good results. The company is now enrolling volunteers in a large efficacy trial.

Goepfert says it'll be a good thing if all these vaccines make it to consumers. But that alone isn't going to solve the problem of getting people vaccinated. Why? "Because the vaccines that we have now are just beyond our wildest dreams kind of effective," he says. "And I'm living in a state right now where it just frustrates me how slow our vaccine uptake is."

Goepfert lives in Alabama. According to the latest numbers from the Centers for Disease Control and Prevention, only Mississippi has a lower per capita rate of vaccination.

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New Research Shows Māori Traveled to Antarctica at Least 1,000 Years Before Europeans

Researchers suggest the Māori have a significantly longer history with Earth's southernmost continent

Jacinta Bowler

When we think of Antarctic exploration, the narrative is overwhelmingly white. The first confirmed sighting of mainland

Antarctica was attributed to a Russian expedition in 1820, while the first landing on the mainland is attributed to an American explorer in 1821.

Now, a new paper by New Zealander researchers suggests that the indigenous people of mainland New Zealand - Māori - have a significantly longer history with Earth's southernmost continent.

The research team, led by conservation biologist Priscilla Wehi from Manaaki Whenua Landcare Research, looked at oral histories as well as 'grey literature' – meaning research, reports, technical documents and other material published by organizations outside common academic or commercial publishing channels.

"We found connection to Antarctica and its waters have been occurring since the earliest traditional voyaging, and later through participation in European-led voyaging and exploration, contemporary scientific research, fishing, and more for centuries," said Wehi.

The researchers first highlight an early 7th century southern voyage by a Polynesian chief Hui Te Rangiora and his crew. This would have likely made them the first humans to see Antarctic waters, over a thousand years before the Russian expedition and even long before Polynesian settlers' planned migration to New Zealand.

"In some narratives, Hui Te Rangiora and his crew continued south. A long way south. In so doing, they were likely the first humans to set eyes on Antarctic waters and perhaps the continent," the team writes in their paper.

"Hui Te Rangiora's voyage and return are part of the history of the Ngāti Rārua people, and these stories appear in a number of carvings."

This finding might not be much of a surprise to our Māori readers who have been telling these stories for generations, but as the paper explains, academic literature still has a long way to go to catch up to this wealth of knowledge.

"The narratives of under-represented groups and their connection to Antarctica remain poorly documented and acknowledged in the research literature," the team writes. "This paper begins to fill this gap."

But Hui Te Rangiora's voyage definitely wasn't the last time Māori and their ancestors traveled to Antarctica.

Te Atu – a Ngāpuhi man - has been called the first Māori and first New Zealander to view the coast of Antarctica in 1840 as part of the United States Exploring Expedition.

Māori were also part of the 'Heroic Age of Antarctic Exploration' in the late 19th and early 20th century, helping European explorers with medicine, construction, scientific expertise and more on journeys to Antarctica.

"Māori participation in Antarctic voyaging and expedition has continued to the present day but is rarely acknowledged or highlighted," the researchers write. "For Māori on these voyages, seafaring skills were the critical currency."

More recently, a number of Māori have or are currently participating in New Zealand's Antarctic science programs, doing research on everything from the effects of climate change to penguin population ecology, and the team behind this latest paper hopes these numbers will grow.

"Taking account of responsibilities to under-represented groups, and particularly Māori as Treaty partners, is important for both contemporary and future programs of Antarctic research, as well as for future exploration of New Zealand's obligations within the Antarctic Treaty System," said Wehi.

"Growing more Māori Antarctic scientists and incorporating Māori perspectives will add depth to New Zealand's research programs and ultimately the protection and management of Antarctica."

The research has been published in the *Journal of the Royal Society of New Zealand*.