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An Ancient Meteorite Is The First Chemical Evidence of Volcanic Convection on Mars

The idea of a volcanically active Mars just got a little more real

Michelle Starr

For many years, we thought Mars was dead. A dusty, dry, barren planet, where nothing moves but the howling wind. Recently, however, pieces of evidence have started to emerge, hinting that Mars is both volcanically and geologically active.

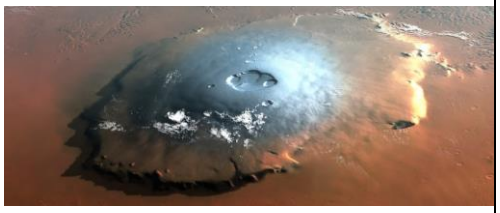


Illustration of Olympus Mons on Mars - the biggest volcano in the Solar System. **(Mark Garlick/Science Photo Library/Getty)**

Well, the idea of a volcanically active Mars just got a little more real. A meteorite that formed deep within the belly of Mars has just provided the first solid chemical proof of magma convection within the Martian mantle, scientists say.

Crystals of olivine in the Tissint meteorite that fell to Earth in 2011 could only have formed in changing temperatures as it was rapidly swirled about in magma convection currents - showing that the planet was volcanically active when the crystals formed around 574 to 582 million years ago - and it could still be intermittently so today.

"There was no previous evidence of convection on Mars, but the question 'Is Mars a still volcanically active planet?' was previously investigated using different methods," explained planetary geologist Nicola Mari of the University of Glasgow to ScienceAlert.

"However, this is the first study that proves activity in the Mars interior from a purely chemical point of view, on real Martian samples."

Olivine, a magnesium iron silicate, isn't rare. It crystallises from cooling magma, and it's very common in Earth's mantle; in fact, the olivine group dominates Earth's mantle, usually as part of a rock mass. On Earth's surface, it's found in igneous rock.

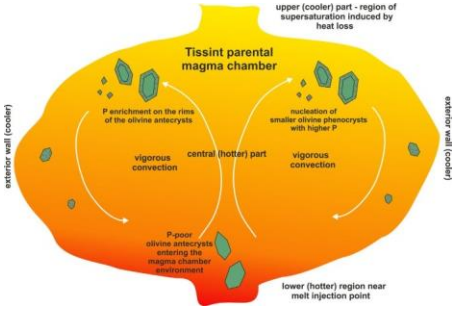
It's fairly common in meteorites. And olivine is also fairly common on Mars. In fact, the presence of olivine on the surface of Mars has previously been taken as evidence of the planet's dryness, since the mineral weathers rapidly in the presence of water.

But when Mari and his team started studying the olivine crystals in the Tissint meteorite to try to understand the magma chamber where it formed, they noticed something strange. The crystals had irregularly spaced phosphorus-rich bands.

We know of this phenomenon on Earth - it's a process called solute trapping. But it was a surprise to find it on Mars.

"This occurs when the rate of crystal growth exceeds the rate at which phosphorus can diffuse through the melt, thus the phosphorus is obliged to enter the crystal structure instead of 'swimming' in the liquid magma," Mari said.

"In the magma chamber that generated the lava that I studied, the convection was so vigorous that the olivines were moved from the bottom of the chamber (hotter) to the top (cooler) very rapidly - to be precise, this likely generated cooling rates of 15-30 degrees Celsius per hour for the olivines."



(Mari et al., Meteoritics & Planetary Science, 2020)

The larger of the olivine crystals were also revealing. Traces of nickel and cobalt are in agreement with previous findings that they originated from deep under the Martian crust, a depth of 40 to 80 kilometres (25 to 50 miles).

This supplied the pressure at which they formed; along with the equilibration temperature of olivine, the team could now perform thermodynamic calculations to discover the temperature in the mantle at which the crystals formed.

They found that the Martian mantle probably had a temperature of around 1,560 degrees Celsius in the Martian Late Amazonian period when the olivine formed. This is very close to the ambient mantle temperature of Earth of 1,650 degrees Celsius during the Archean Eon, 4 to 2.5 billion years ago.

That doesn't mean Mars is just like an early Earth. But it does mean that Mars could have retained quite a bit of heat under its mantle; it's thought that, because it lacks the plate tectonics that help to dissipate heat on Earth, Mars may cool more slowly.

"I really think that Mars could be a still volcanically active world today, and these new results point toward this," Mari told ScienceAlert.

"We may not see a volcanic eruption on Mars for the next 5 million years, but this doesn't mean that the planet is inactive. It could just mean that the timing between eruptions between Mars and Earth is different, and instead of seeing one or more eruptions per day (as on Earth) we could see a Martian eruption every n-millions of years."

We'll need more research to confidently say this hypothesis checks out. But these results also mean that previous interpretations of the planet's dryness based on surface olivine may need to be revisited. (Although let us be clear, Mars is still extremely dry.)

The ongoing NASA InSight mission that recently found evidence of Marsquakes, measures - among other things - the heat flux from the Martian crust. If Mars is still volcanically active, we may know more about it really soon.

The research has been published in *Meteoritics & Planetary Science*.

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New solar panels suck water from air to cool themselves down

Like humans, solar panels don't work well when overheated. Now, researchers have found a way to make them "sweat"—allowing them to cool themselves and increase their power output.

By Robert F. Service

It's "a simple, elegant, and effective [way] to retrofit existing solar cell panels for an instant efficiency boost," says Liangbing Hu, a materials scientist at the University of Maryland, College Park.

Today, more than 600 gigawatts of solar power capacity exists worldwide, providing 3% of global electricity demand. That capacity is expected to increase fivefold over the next decade. Most use silicon to convert sunlight to electricity. But typical silicon cells convert only 20% of the Sun's energy that hits them into current. Much of the rest turns into heat, which can warm the panels by as much as 40°C. And with every degree of temperature above 25°C, the efficiency of the panel drops. In a field where engineers struggle for every 0.1% boost in power conversion efficiency, even a 1% gain would be an economic boon, says Jun Zhou, a materials scientist at Huazhong University of Science and Technology.

Decades ago, researchers showed that cooling solar panels with water can provide that benefit. Today, some companies even sell water-cooled systems. But those setups require abundant available water and storage tanks, pipes, and pumps. That's of little use in arid regions and in developing countries with little infrastructure.

Enter an atmospheric water collector. In recent years, researchers have devised materials that can suck water vapor from the air and condense it into liquid water for drinking. Among the best is a gel that strongly absorbs water vapor at night, when the air is cool and humidity is high. The gel—a mix of carbon nanotubes in polymers with a water-attracting calcium chloride salt—causes the vapor to

condense into droplets that the gel holds. When heat rises during the day, the gel releases water vapor. If covered by a clear plastic, the released vapor is trapped, condenses back into liquid water, and flows into a storage container.

Peng Wang, an environmental engineer at Hong Kong Polytechnic University, and his colleagues thought of another use for the condensed water: coolant for solar panels. So, the researchers pressed a 1-centimeter-thick sheet of the gel against the underside of a standard silicon solar panel. Their idea was that during the day, the gel would pull heat from the solar panel to evaporate water it had pulled out of the air the previous night, releasing the vapor through the bottom of the gel. The evaporating water would cool the solar panel as sweat evaporating from the skin cools us down.

The researchers found that the amount of gel they needed depended primarily on the environment's humidity. In a desert environment with 35% humidity, a 1-square-meter solar panel required 1 kilogram of gel to cool it, whereas a muggy area with 80% humidity required only 0.3 kilograms of gel per square meter of panel.

The upshot in either case: The temperature of the water-cooled solar panel dropped by as much as 10°C. And the electricity output of the cooled panels increased by an average of 15% and up to 19% in one outdoor test, where the wind likely enhanced the cooling effect, Wang and his colleagues report today in *Nature Sustainability*.

"The efficiency increase is significant," Zhou says. But he points out that rain could dissolve the calcium chloride salt in the gel, sapping its water-attracting performance. Wang agrees, but notes the hydrogel sits beneath the solar panel, which should shield it from rain. He and his colleagues are also working on a second-generation gel that shouldn't degrade, even when wet.

Another design option, Wang says, is a setup that could trap and recondense water after it evaporates from the gel. That water, he says, could be used to clean any dust that accumulates on the solar panels, solving a second power-sapping problem at the same time. Alternatively, that same water could be stored for drinking, addressing another desperate need in arid regions.

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Antihistamines may help patients with malignant melanoma

Can a very common allergy medicine improve survival among patients suffering from the serious skin cancer, malignant melanoma?

A new study from Lund University in Sweden indicates that this may be the case. "Previous studies have shown that the same antihistamines have survival benefits in breast cancer. Now we see the same thing concerning malignant melanoma. However, more research is required to confirm the results", emphasises Professor Håkan Olsson. He is one of the researchers behind the study, which was recently published in the research journal, *Allergy*.

In the study, the researchers examined the use of six antihistamines in patients diagnosed with malignant melanoma; desloratadine, cetirizine, loratadine, clemastine, ebastine and fexofenadine.

They have matched information from three large registers (the prescribed drug register, cancer register and cause of death register) for everyone in Sweden between 2006 and 2014 who received their first diagnosis of skin cancer, a total of 24 562 individuals. Of these individuals, 1 253 were antihistamine users. Most used desloratadine (395) cetirizine (324), loratadine (251) or clemastine (192). The other antihistamines were used by considerably fewer individuals. The follow-up of individuals was carried out on 31 December 2018.

"We observed improved survival among those who used desloratadine and to a certain extent also loratadine, particularly in the age group 65 and older, when we compared with those who had not used antihistamines. The use of the other antihistamines showed no significant survival effect. The use of desloratadine and loratadine also seemed to reduce the risk of getting a new malignant melanoma", says Håkan Olsson.

"The finding is interesting for a future drug against melanoma and may also help in advanced stages of the disease. In addition, the medicines have virtually no side effects."

The research team is now planning animal experiments and randomised studies in order to understand the mechanisms behind the effect, the appropriate dose and optimum treatment period.

"We are collaborating with researchers in Barcelona and Stockholm. In Lund, we are underway with studies in both animal and human subjects, in which doses of antihistamines will be compared with the patients who do not take antihistamines, in order to measure the treatment effect", concludes Håkan Olsson.

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SwRI scientist modeled Mars climate to understand habitability

Study suggests terrestrial life unlikely to contaminate planet

SAN ANTONIO - A Southwest Research Institute scientist modeled the atmosphere of Mars to help determine that salty pockets of water present on the Red Planet are likely not habitable by life as we know it on Earth. A team that also included scientists from Universities Space Research Association (USRA) and the University of Arkansas helped allay planetary protection concerns about contaminating potential Martian ecosystems. These results were published this month in *Nature Astronomy*.

Due to Mars' low temperatures and extremely dry conditions, a droplet of liquid water on its surface would instantly freeze, boil or evaporate, unless the droplet had dissolved salts in it. This brine would have a lower freezing temperature and would evaporate more slowly than pure liquid water. Salts are found across Mars, so brines could form there.

"Our team looked at specific regions on Mars -- areas where liquid water temperature and accessibility limits could possibly allow known terrestrial organisms to replicate -- to understand if they could be habitable," said SwRI's Dr. Alejandro Soto, a senior research scientist and co-author of the study. "We used Martian climate information from both atmospheric models and spacecraft measurements. We developed a model to predict where, when and for how long brines are stable on the surface and shallow subsurface of Mars."

Mars' hyper-arid conditions require lower temperatures to reach high relative humidities and tolerable water activities, which are measures of how easily the water content may be utilized for hydration. The maximum brine temperature expected is -55 F -- at the boundary of the theoretical low temperature limit for life.

"Even extreme life on Earth has its limits, and we found that brine formation from some salts can lead to liquid water over 40% of the Martian surface but only seasonally, during 2% of the Martian year," Soto continued. "This would preclude life as we know it."

While pure liquid water is unstable on the Martian surface, models showed that stable brines can form and persist from the equator to high latitudes on the surface of Mars for a few percent of the year for up to six consecutive hours, a broader range than previously thought. However, the temperatures are well below the lowest temperatures to support life. "These new results reduce some of the risk of exploring the Red Planet while also contributing to future work on the potential for habitable conditions on Mars," Soto said.

Soto collaborated with co-authors from the Lunar and Planetary Institute (USRA) and the Arkansas Center for Space and Planetary Sciences at the University of Arkansas on the paper "Distribution and Habitability of (Meta)stable Brines on Present-Day Mars,"

published in May in Nature Astronomy. The SwRI portion of this research was funded by NASA under the Habitable Worlds program through a grant led by USRA.

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Exploring why some COVID-19 patients lose their sense of smell

Two proteins required for SARS-CoV-2 entry are produced by cells of the nasal cavity that contribute to odor detection

Doctors have reported that partial or total loss of the sense of smell is often an early symptom of infection with SARS-CoV-2, the virus that causes COVID-19. Now, researchers reporting in *ACS Chemical Neuroscience* have shown that in mice, two proteins required for SARS-CoV-2 entry are produced by cells of the nasal cavity that contribute to odor detection. Moreover, larger amounts of the proteins are made in older animals than in younger ones.

The new coronavirus still holds many secrets, one of which is how it can cause loss of smell -- even in infected people who have no other COVID-19 symptoms. SARS-CoV-2 hijacks two proteins to invade human cells: the cell surface receptor ACE2 and the protease TMPRSS2. However, scientists still aren't sure which cells in the olfactory epithelium (the tissue lining the nasal cavity) express these proteins and could potentially be infected by the virus. Finding out could help explain symptoms and aid in the development of more accurate diagnostic tests. So Rafal Butowt and colleagues studied the proteins' expression in mice and how their levels change with age.

Using several methods, the researchers found that ACE2 and TMPRSS2 are expressed in sustentacular cells -- cells of the nose that help transfer odors from the air to neurons. Older mice made more of the two proteins in nasal cells than younger mice. If also true in humans, this result could explain why older people are more susceptible to SARS-CoV-2, the researchers say. They also note that future studies should examine whether sustentacular cells can pass the virus to neurons, which could provide SARS-CoV-2 a route to infect the brain.

The authors acknowledge funding from the National Science Centre of Poland and the National Institute of General Medical Sciences of the National Institutes of Health.

The abstract that accompanies this paper can be viewed here.

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World-first saliva test detects hidden throat cancer

A simple saliva test developed by QUT biomedical scientists has detected early throat cancer in a person who had no symptom and no clinical signs of cancer.

In what is believed to be a world-first, the non-invasive test picked up HPV-DNA in a saliva sample from an infected healthy person. Persistent human papillomavirus (HPV) infection is now the leading cause of cancers in the oropharynx (tonsils and tongue base area of the throat). "The series of saliva tests raised the alert and detected an early cancer before the person had any symptoms," said QUT Faculty of Health's Associate Professor Chamindie Punyadeera, who, with Dr Kai Tang, developed the test. "This enabled removal of the tonsil which had a 2mm cancer in it, by straightforward local surgery alone.

"The incidence of high-risk human papillomavirus (HPV)-driven throat cancers is on the rise in developed countries and, unfortunately, it is often discovered only when it is more advanced, with patients needing complicated and highly impactful treatment.

"In the US, HPV-driven throat cancers have surpassed cervical cancers as the most common cancer caused by HPV but unlike cervical cancer, up until now, there has been no screening test for this type of oropharyngeal cancer."

Professor Punyadeera said the discovery was made during an HPV-prevalence study which included 665 healthy individuals.

"To take the test all the person has to do is give a salivary oral rinse sample. When the test shows HPV-16 DNA, it is repeated and if the

presence of HPV-16 is persistent over a period of time we would be suspicious that there may be underlying cancer.

"The person whom we reported in this study had been consistently HPV-16 DNA positive for 36 months, with a steadily rising count of HPV-16 DNA after testing at 6, 12 and 36 months.

"The patient was found to have a 2mm squamous cell carcinoma in the left tonsil, treated by tonsillectomy. This has given our patient a high chance of cure with very straightforward treatment. "Since the surgery, the patient has had no evidence of HPV-16 DNA in his saliva."

Professor Punyadeera said this was the first-ever case of histologically confirmed diagnosis of an asymptomatic, hidden throat cancer, diagnosed with a saliva screening test and that wider validation studies were required to confirm this finding.

"The presence of this pattern of elevated salivary HPV-DNA must be fully evaluated, as it may provide the critical marker for early cancer detection. "We now have the promise of a screening test for oropharynx cancer and there is an urgent need to undertake a major study to validate this test and the appropriate assessment pathway for people with persisting salivary HPV-DNA."

This research is part of a collaboration with Royal Brisbane and Women's Hospital's Professor Liz Kenny, Dr Sarj Vasani, Dr Touraj Taheri and Associate Professor Brett Hughes and University of Queensland's Professor Laurence J. Walsh.

The study, An Occult HPV-Driven Oropharyngeal Squamous Cell Carcinoma Discovered Through a Saliva Test was published in Frontiers in Oncology.

https://bit.ly/3dLiBKw

Coronavirus infection in children -- it may not start with a cough

Gastrointestinal symptoms, coupled with a fever or history of exposure to COVID-19, could indicate coronavirus infection in children

Children suffering from sickness and diarrhea, coupled with a fever or history of exposure to coronavirus, should be suspected of being infected with COVID-19, recommends a new study published in *Frontiers in Pediatrics*.

The research also suggests that the gastrointestinal symptoms first suffered by some children hints at potential infection through the digestive tract, as the type of receptors in cells in the lungs targeted by the virus can also be found in the intestines.

"Most children are only mildly affected by COVID-19 and the few severe cases often have underlying health issues. It is easy to miss its diagnosis in the early stage, when a child has non-respiratory symptoms or suffers from another illness," says author of this study, Dr. Wenbin Li, who works at the Department of Pediatrics, Tongji Hospital, Wuhan, China.

He continues, "Based on our experience of dealing with COVID-19, in regions where this virus is epidemic, children suffering from digestive tract symptoms, especially with fever and/or a history of exposure to this disease, should be suspected of being infected with this virus."

In this study, Li and his colleagues detail the clinical features of children admitted to hospital with non-respiratory symptoms, which were subsequently diagnosed with pneumonia and COVID-19.

"These children were seeking medical advice in the emergency department for unrelated problems, for example, one had a kidney stone, another a head trauma. All had pneumonia confirmed by chest CT scan before or soon after admission and then confirmed to have COVID-19. While their initial symptoms may have been unrelated, or their COVID-19 symptoms were initially mild or relatively hidden before their admission to hospital, importantly, 4 of the 5 cases had digestive tract symptoms as the first manifestation of this disease."

By highlighting these cases, Li hopes that doctors will use this information to quickly diagnose and isolate patients with similar symptoms, which will aid early treatment and reduce transmission.

The researchers also link the children's gastrointestinal symptoms, which have been recorded in adult patients, to an additional potential route of infection.

Li explains, "The gastro-intestinal symptoms experienced by these children may be related to the distribution of receptors and the transmission pathway associated with COVID-19 infection in humans. The virus infects people via the ACE2 receptor, which can be found in certain cells in the lungs as well as the intestines. This suggests that COVID-19 might infect patients not only through the respiratory tract in the form of air droplets, but also through the digestive tract by contact or fecal-oral transmission."

While COVID-19 tests can occasionally produce false positive readings, Li is certain all these five children were infected with the disease, but he cautions that more research is needed to confirm their findings.

"We report five cases of COVID-19 in children showing non-respiratory symptoms as the first manifestation after admission to hospital. The incidence and clinical features of similar cases needs further study in more patients."

Notes to Editors *Please link to the original research article in your reporting:* *https://www.frontiersin.org/articles/10.3389/fped.2020.00258/full*

https://bit.ly/3dLXA2f

Not all twins are identical and that's been an evolutionary puzzle, until now

When a mother gives birth to twins, the offspring are not always identical or even the same gender. Known as fraternal twins, they represent a longstanding evolutionary puzzle.

Joseph L Tomkins [*] Rebecca Sear [**] Wade Hazel [***]

Identical twins arise from a single fertilised egg that accidentally splits in two, but fraternal twins arise when two eggs are released and fertilised. Why this would happen was the puzzle.

In research published today in Nature Ecology & Evolution we used computer simulations and modelling to try to explain why natural selection favours releasing two eggs, despite the low survival of twins and the risks of twin births for mothers.

Why twins?

Since Michael Bulmer's landmark 1970 book on the biology of twinning in humans, biologists have questioned whether double ovulation was favoured by natural selection or, like identical twins, was the result of an accident.

At first glance, this seems unlikely. The embryo splitting that produces identical twins is not heritable and the incidence of identical twinning does not vary with other aspects of human biology. It seems accidental in every sense of the word.

In contrast, the incidence of fraternal twinning changes with maternal age and is heritable.

Those do not sound like the characteristics of something accidental.

The twin disadvantage

In human populations without access to medical care there seems little benefit to having twins. Twins are more likely to die in childhood than single births. Mothers of twins also have an increased risk of dying in childbirth.

In common with other great apes, women seem to be built to give birth to one child at a time. So if twinning is costly, why has evolution not removed it?

Paradoxically, in high-fertility populations, the mothers of twins often have more offspring by the end of their lives than other mothers. This suggests having twins might have an evolutionary benefit, at least for mothers.

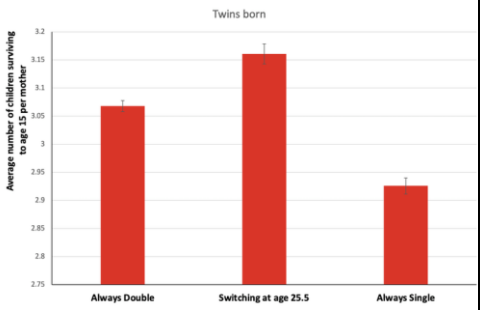
But, if this is the case, why are twins so rare?

Modelling mothers

To resolve these questions, together with colleagues Bob Black and Rick Smock, we constructed simulations and mathematical models

fed with data on maternal, child and fetal survival from real populations.

This allowed us to do something otherwise impossible: control in the simulations and modelling whether women ovulated one or two eggs during their cycles. We also modelled different strategies, where we switched women from ovulating one egg to ovulating two at different ages. We could then compare the number of surviving children for women with different patterns of ovulation.



Author provided

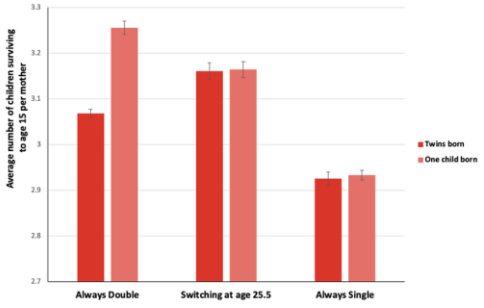
Women who switched from single to double ovulation in their mid-20s had the most children survive in our models – more than those who always released a single egg, or always released two eggs.

This suggests natural selection favours an unconscious switch from single to double ovulation with increasing age.

A strategy for prolonging fertility

The reason a switch is beneficial is fetal survival – the chance that a fertilised egg will result in a liveborn child – decreases rapidly as women age.

So switching to releasing two eggs increases the chance at least one will result in a successful birth.



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But what about twinning? Is it a side effect of selection favouring fertility in older women? To answer this question, we ran the simulations again, except now when women double ovulated the simulation removed one offspring before birth.

In these simulations, women who double ovulated throughout their lives, but never gave birth to twins, had more children survive than those who did have twins and switched from single to double ovulating. This suggests the ideal strategy would be to always double ovulate but never produce twins, so fraternal twins are an accidental side effect of a beneficial strategy of double ovulating.

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Ice Age giant sloths died in a pit of their own poop

The animals may have been sickened after feces contaminated their watering hole

By Mindy Weisberger - Senior Writer 3 days ago

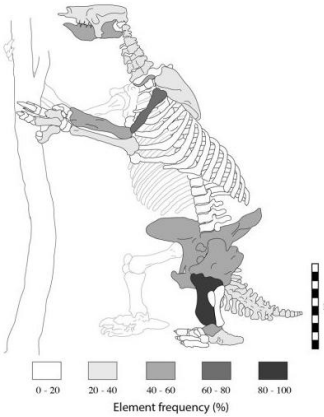
During the Ice Age, a group of giant ground sloths died together, possibly after swallowing their own feces in a contaminated pool of shallow water.

Scientists discovered the bones of nearly two dozen ground sloths (*Eremotherium laurillardi*) in a pit at a fossil-rich site called Tanque Loma in southwestern Ecuador. The bone bed dates to the end of the Pleistocene epoch (around 2.6 million to 11,700 years ago) and holds thousands of bones from large mammals.

The condition of the sloth bones and their arrangement relative to each other hinted that the animals died around the same time, the scientists wrote in a new study. And preserved vegetation helped the researchers piece together a grim picture of a marshy watering

hole saturated with sloth poo, that sickened and killed the sloths that gathered there, the researchers reported.

Giant ground sloths once roamed the Americas and are kin to the much smaller tree sloths that are around today. The biggest ground sloth, *Megalonyx jeffersonii*, reached about 10 feet (3 meters) in height and would have towered above a human. These massive herbivores first appeared in South America about 35 million years ago and died out at the end of the Pleistocene, along with most other big Ice Age mammals, such as mastodons, dire wolves and cave lions.



Bones found in Tanque Loma represent 22 sloths; adults and juveniles.
(Image: © E.L. Lindsey, E.X. Lopez Reyes, G.E. Matzke, et al., Palaeogeography, Palaeoclimatology, Palaeoecology (2020)

Some experts argue that humans hunted these mega mammals to extinction, while others say that the animals vanished as the global climate changed. But for the Tanque Loma sloths, death came for different reasons.

Researchers identified 575 bones representing 22 ground sloth adults and juveniles, dating them to around 18,000 to 23,000 years ago. The bones were preserved in a single layer without much sediment separating them, suggesting that the animals died around the same time and were submerged soon afterward, according to the study.

While there was a coating of asphalt atop the bones, it didn't extend all the way through the fossil layer. This detail told the researchers that the sticky goo seeped into the marsh after the animals were already dead, and that the sloths didn't die because they became trapped in sticky tar, as was the case in the La Brea tar pits in Los Angeles, for instance.

The scientists also analyzed the soil around the bones and plant matter at the site, identifying the location as a marsh that periodically dried up, allowing ground plants to flourish. The sloth bones were surrounded by plants that appeared to have been chewed and digested.

So, what killed all those sloths? One likely explanation is that they wallowed together in a watering hole as do modern large herbivores, such as wildebeests and hippos, to escape heat and insects. But their relief took a deadly turn; after the animals fouled the marsh with their feces, they would have later eaten contaminated plants and drunk polluted water, leading to their deaths from pathogens lurking in those feces. More recently, hippos have died en masse in marshy locations dirtied by enormous quantities of their poo, the scientists said.

In one case in the 1970s, during the dry season, a herd of hippos in Tanzania filled a shrinking watering hole with their feces; photos of the wallow showed "a small group of live hippos in the water and many hippo corpses on the shore," and the herd shrank from 140 hippos to around 40 in just one week, the researchers wrote.

Based on the evidence from Ecuador, the giant ground sloths likely met a similar fate. The findings were published online April 15 in the journal Palaeogeography, Palaeoclimatology, Palaeoecology.

https://bit.ly/3bFEPfG

Geometry guided construction of earliest known temple, built 6,000 years before Stonehenge

Hunter-gatherers built colossal Göbekli Tepe 11,500 years ago in today's Turkey as a single structure of ritual significance, say Tel Aviv University researchers

The sprawling 11,500-year-old stone Göbekli Tepe complex in southeastern Anatolia, Turkey, is the earliest known temple in human history and one of the most important discoveries of Neolithic research.

Researchers at Tel Aviv University and the Israel Antiquities Authority have now used architectural analysis to discover that geometry informed the layout of Göbekli Tepe's impressive round stone structures and enormous assembly of limestone pillars, which they say were initially planned as a single structure.

***Göbekli Tepe, Enclosure C.* Credit: Gil Haklay/AFTAU.**

Three of the Göbekli Tepe's monumental round structures, the largest of which are 20 meters in diameter, were initially planned as a single project, according to researchers Gil Haklay of the Israel Antiquities Authority, a PhD candidate at Tel Aviv University, and Prof. Avi Gopher of TAU's Department of Archaeology and Ancient Near Eastern Civilizations. They used a computer algorithm to trace aspects of the architectural design processes involved in the construction of these enclosures in this early Neolithic site. Their findings were published in *Cambridge Archaeological Journal* in May.

"Göbekli Tepe is an archaeological wonder," Prof. Gopher explains. "Built by Neolithic communities 11,500 to 11,000 years ago, it features enormous, round stone structures and monumental stone pillars up to 5.5 meters high. Since there is no evidence of farming or animal domestication at the time, the site is believed to have been built by hunter-gatherers. However, its architectural complexity is highly unusual for them."

Discovered by German archaeologist Dr. Klaus Schmidt in 1994, Göbekli Tepe has since been the subject of hot archaeological debate. But while these, and other early Neolithic remains, have been intensively studied, the issue of architectural planning during these periods and its cultural ramifications have not.

Most researchers have made the case that the Göbekli Tepe enclosures at the main excavation area were constructed over time. However, Haklay and Prof. Gopher say that three of the structures were designed as a single project and according to a coherent geometric pattern.

"The layout of the complex is characterized by spatial and symbolic hierarchies that reflect changes in the spiritual world and in the social structure," Haklay explains. "In our research, we used an analytic tool -- an algorithm based on standard deviation mapping -- to identify an underlying geometric pattern that regulated the design."

"This research introduces important information regarding the early development of architectural planning in the Levant and in the world," Prof. Gopher adds. "It opens the door to new interpretations of this site in general, and of the nature of its megalithic anthropomorphic pillars specifically."

Certain planning capabilities and practices, such as the use of geometry and the formulation of floor plans, were traditionally assumed to have emerged much later than the period during which the Göbekli Tepe was constructed -- after hunter-gatherers transformed into food-producing farmers some 10,500 years ago. Notably, one of the characteristics of early farmers is their use of rectangular architecture.

"This case of early architectural planning may serve as an example of the dynamics of cultural changes during the early parts of the Neolithic period," Haklay says. "Our findings suggest that major architectural transformations during this period, such as the transition to rectangular architecture, were knowledge-based, top-down processes carried out by specialists.

"The most important and basic methods of architectural planning were devised in the Levant in the Late Epipaleolithic period as part of the Natufian culture and through the early Neolithic period. Our

new research indicates that the methods of architectural planning, abstract design rules and organizational patterns were already being used during this formative period in human history."

Next, the researchers intend to investigate the architectural remains of other Neolithic sites throughout the Levant.

https://bit.ly/2XcHh8f

Growing mountains or shifting ground: What is going on in Earth's inner core?

Best evidence yet that the Earth's inner core is rotating

CHAMPAIGN, Ill. -- Exhaustive seismic data from repeating earthquakes and new data-processing methods have yielded the best evidence yet that the Earth's inner core is rotating - revealing a better understanding of the hotly debated processes that control the planet's magnetic field. The new study by researchers from the University of Illinois at Urbana-Champaign is published in the journal *Earth and Planetary Science Letters*.

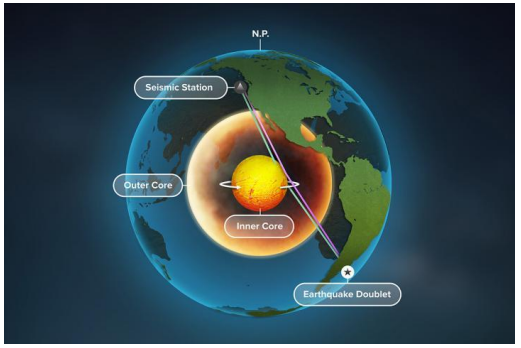
Geologists do not fully understand how the Earth's magnetic field generator works, but suspect it is closely linked to dynamic processes near the inner core-outer core boundary area, the researchers said. Shifts in the location of the magnetic poles, changes in field strength and anomalous seismic data have prompted researchers to take a closer look.

"In 1996, a small but systematic change of seismic waves passing through the inner core was first detected by our group, which we interpreted as evidence for differential rotation of the inner core relative to the Earth's surface," said geology professor and study co-author Xiaodong Song, who is now at Peking University.

"However, some studies believe that what we interpret as movement is instead the result of seismic waves reflecting off an alternately enlarging and shrinking inner core boundary, like growing mountains and cutting canyons."

The researchers present seismic data from a range of geographic locations and repeating earthquakes, called doublets, that occur in the same spot over time. "Having data from the same location but different times allows us to differentiate between seismic signals that change due to localized variation in relief from those that change due to movement and rotation," said Yi Yang, a graduate student and lead author of the study.

The team found that some of the earthquake-generated seismic waves penetrate through the iron body below the inner core boundary and change over time, which would not happen if the inner core were stationary, the researchers said. "Importantly, we are seeing that these refracted waves change before the reflected waves bounce off the inner core boundary, implying that the changes are coming from inside the inner core," Song said.



A new study of Earth's inner core used seismic data from repeating earthquakes, called doublets, to find that refracted waves, blue, rather than reflected waves, purple, change over time -- providing the best evidence yet that Earth's inner core is rotating. **Graphic by Michael Vincent**

The basis of the debate lies in the fact the prior studies looked at a relatively small pool of somewhat ambiguous data generated from a method that is highly dependent on accurate clock time, the researchers said.

"What makes our analysis different is our precise method for determining exactly when the changes in seismic signals occur and arrive at the various seismic stations across the globe," Yang said. "We use a seismic wave that did not reach inner core as a reference wave in our calculations, which eliminates a lot of the ambiguity."

This precise arrival time analysis, an extensive collection of the best quality data and careful statistical analysis performed by Yang, are what give this study its power, Song said. "This work confirms that the temporal changes come mostly, if not all, from the body of the inner core, and the idea that inner core surface changes are the sole source of the signal changes can now be ruled out," he said.

The National Science Foundation and the Natural Science Foundation of China supported this study.

Editor's notes: To reach Xiaodong Song, call 217-714-5125; email xsong@illinois.edu. The paper "Origin of temporal changes of inner-core seismic waves" is available online and from the U. of I. News Bureau. DOI: 10.1016/j.epsl.2020.116267

https://bit.ly/2X13o0W

Arthritis clinical trial shows support for dextrose injection to alleviate knee pain

Efficacy of intra-articular hypertonic dextrose (prolotherapy) for knee osteoarthritis: A randomized controlled trial

A randomized controlled trial conducted by a research team at a primary care clinic at the Chinese University of Hong Kong indicates that intra-articular-only injection therapy with hypertonic dextrose is safe and effective for alleviating symptoms of knee osteoarthritis.

Over 52 weeks of treatment, the study followed 76 patients who were between 45 and 75 years old who had been diagnosed with knee osteoarthritis and who suffered moderate to severe chronic knee pain for at least three months. One group of 38 patients received the hypertonic dextrose injection therapy, while the other had the same therapy only using normal saline. While both groups reported some improvement, the hypertonic dextrose group reported more significant reductions in pain by the conclusion of the study. The researchers note that longer-term follow-up, direct comparison with other injection therapies, and cost-effective analysis are all needed.

Efficacy of Intra-Articular Hypertonic Dextrose (Prolotherapy) for Knee Osteoarthritis: A Randomized Controlled Trial *Regina Wing Shan Sit, MBBS, et al*

The Chinese University of Hong Kong, Jockey Club School of Public Health and Primary Care, Hong Kong

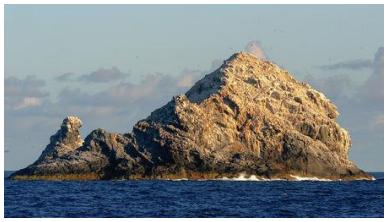
https://bit.ly/2LxdeCs

World's biggest volcano is barely visible

Two small, guano-covered islands that peek above the waves in the central North Pacific Ocean are merely the tips of our planet's largest single volcano, new research reveals.

By Sid Perkins

Pūhāhonu—Hawaiian for "turtle surfacing for air"—lies about 1100 kilometers northwest of Honolulu. It is a shield volcano—a broad dome that rises about 4500 meters from the sea floor from a single source of molten rock. In an analysis reported this month in *Earth and Planetary Science Letters*, researchers estimate that Pūhāhonu contains approximately 150,000 cubic kilometers of rock, based on a 2014 sonar survey.



NOAA

But only one-third of that volume is exposed above the sea floor; the rest is buried beneath a ring of debris, broken coral, and other material that has eroded from the peak. Pūhāhonu is so heavy, researchers note, that it has caused Earth's crust nearby—and thus the volcano itself—to sink hundreds of meters over millions of years.

From sea floor to peak, Mauna Loa, on Hawaii's Big Island, is the tallest shield volcano on Earth. But it's nowhere near as big as Pūhāhonu; a 2013 study estimates Mauna Loa's volume at 83,000 cubic kilometers. The Tamu Massif, a 4-kilometer-tall volcanic feature the size of the British Isles on the sea floor east of Japan, contains almost 7 million cubic kilometers of material and was once thought to be the world's largest shield volcano. But now it is

believed to have formed along a midocean ridge rather than over a single source of magma. That makes Pūhāhonu the world champion shield volcano … for now.

https://bit.ly/2yZ8XoS

The north magnetic pole is leaving Canada for Siberia. These 'blobs' may be the reason why.

These 'blobs' may be the reason why. - Moving away because of a fierce tug-of-war battle being waged by two giant blobs hiding deep underground

By Laura Geggel - Associate Editor

The north magnetic pole is lurching away from its traditional home in the Canadian Arctic and toward Siberia because of a fierce tug-of-war battle being waged by two giant blobs hiding deep underground, at the core–mantle boundary, a new study finds.

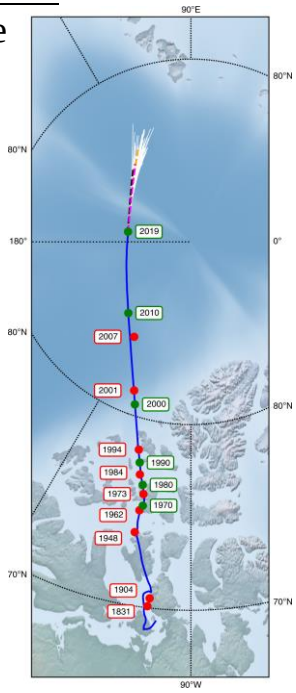
These blobs, areas of negative magnetic flow under Canada and Siberia, are in a winners-take-all struggle. Already, as these blobs change shape and magnetic intensity, a victor has emerged; from 1999 to 2019, while the blob beneath Canada weakened, the blob under Siberia slightly intensified, the researchers found. "Together, these changes caused the north magnetic pole to travel towards Siberia," the researchers wrote in the study.

"We've never seen anything like this before," study lead researcher Phil Livermore, an associate professor of geophysics at the University of Leeds in the United Kingdom, told Live Science in an email.

When scientists first located the north magnetic pole (the point where your compass needle points) in 1831, it sat in the northern Canadian territory of Nunavut. Soon, researchers realized that the north magnetic pole tended to wander, but it usually didn't stray far. Then, from 1990 to 2005, the magnetic pole's yearly jaunt jumped from a historic speed of no more than 9 miles (15 kilometers) a year to as much as 37 miles (60 km) a year, the researchers wrote in the study.

In October 2017, the north magnetic pole crossed the international date line and entered the Eastern Hemisphere, passing within 242 miles (390 km) of the geographic north pole. Then, the north magnetic pole began moving southward. The change was so rapid, that in 2019, geologists were forced to publish a new World Magnetic Model, a map that informs everything from airplane navigation to the GPS on smartphones, a year ahead of time.

It was anyone's guess why the pole was leaving Canada for Siberia. That was until Livermore and his colleagues realized that the blobs were, in large part, responsible.



The magnetic north pole has wandered away from the Canadian Arctic (solid blue line) and toward Siberia for about the past century, but it has considerably sped up over the past 20 years. The international date line is shown as a black dotted line. The data after 2019 are extrapolated lines based on different models. **(Image credit: Livermore PW, et al. Nature Geoscience (2020))**

Changing blobs

The magnetic field is generated by swirling liquid iron deep inside the Earth in the outer core. As such, changes in that sloshing iron can change the location of magnetic north.

The magnetic field isn't confined to the core, however; magnetic field lines "poke out" of Earth, Livermore said. As it turns out, these blobs are the spots where these lines pop out. "If you imagine the lines of [the] magnetic field like soft spaghetti, then these patches would be like a cluster of spaghetti sticking out of the Earth," he said.

The researchers discovered that from 1999 to 2019, the blob under Canada elongated east to west and divided into two smaller joined blobs, possibly because of a change in the pattern of core flow between 1970 and 1999. One of these blobs had a higher intensity than the other, but overall this elongation "caused the weakening of the Canadian patch at Earth's surface," the researchers wrote in the study.

Furthermore, because of the split, the Canadian blob with higher intensity became closer to the Siberian blob. This, in turn, enhanced the Siberian blob, the researchers wrote.

However, these two blobs are in a delicate balance, so "it would take only a minor readjustment of the present configuration to reverse the current trend" of the north magnetic pole's current trek toward Siberia, the researchers wrote in the study. In other words, a tweak to one blob or the other could send the North Magnetic pole back toward Canada.

Reconstructions of past north magnetic pole movements suggests that two blobs — and sometimes three — have influenced the pole's position over time. These blobs have prompted the pole to wander around northern Canada for the past 400 years, the researchers said.

"But over the last 7,000 years, [the north magnetic pole] seems to have chaotically moved around the geographic pole, showing no preferred location," the researchers wrote in the study. The pole also moved toward Siberia in 1300 B.C., according to modeling.

It's difficult to say what will happen next. "Our predictions are that the pole will continue to move towards Siberia, but forecasting the future is challenging and we cannot be sure," Livermore said.

That forecasting will rely on "detailed monitoring of the geomagnetic field from Earth's surface and space in the coming years," the researchers wrote in the study, which was published online May 5 in the journal Nature Geoscience.

https://bit.ly/2Z84z1s

Donut Sugar Could Help Stored Blood Last

Dehydrated blood that could be kept at room temperature for years may be possible thanks to a sugar used to preserve donuts—and made by tardigrades and brine shrimp so they can dry out and spring back with water.

By Susanne Bard

Download MP3

Blood donations save lives. But blood can only be stored under refrigeration for up to six weeks. After that, it's no longer usable for transfusions.

"Because of that limitation, people have to continually donate blood to meet the needs. But also, in places where refrigeration may not be available, that can also be a challenge. It's difficult to have blood available when needed."

University of Louisville bioengineer Jonathan Kopechek. He says disruptions to regular blood donations due to COVID-19 have put stress on the blood supply, and the pandemic underscores the need for more reliable long-term storage methods. Blood *can* be frozen for extended periods of time ... "But it's pretty rare because of all the challenges and complexities with that process."

Instead Kopechek's team has developed a method of preserving blood so it can be stored in a *dehydrated* state at room temperature. To do so, they turned to an unusual preservative: a sugar called trehalose, which is a common ingredient in *donuts* ...

"To help make them look fresh even when they might be months old, and you wouldn't know the difference."

The researchers chose trehalose because, in nature, it's made by hardy animals like tardigrades and sea monkeys—aka brine shrimp—famous for their ability to survive dehydration.

"So these animals can dry out completely for a long period of time and then be rehydrated and resume normal function. So we wanted to use the trehalose that's produced by these organisms and apply

that to preserving blood cells in a dried state, just like those organisms are."

But first, the researchers had to get trehalose *into* blood cells. They used ultrasound to drill temporary holes in the cell membranes—which let some trehalose get in.

"And they need to have sufficient levels of trehalose on both the inside and the outside of the cell in order to survive the dehydration and rehydration process." At that point, the blood could be dried and made into a powder. "And then we can rehydrate the blood and have it return back to normal."

The team is still trying to improve yields but thinks the dried blood could be stored at room temperature for years. The study is in the journal *Biomicrofluidics*. *[Connor S. Centner et al., Ultrasound-induced molecular delivery to erythrocytes using a microfluidic system]*

Kopechek says the technique could be ready for clinical trials in three to five years. If successful, it could be used to create stores of dried blood in case of future pandemics or natural disasters—and for humanitarian aid work, military operations or even missions to Mars. Maybe first aid kits on the Red Planet will include dried red blood cells.

https://bit.ly/2X1Bj9N

Excess coffee consumption a culprit for poor health

Study shows that excess coffee consumption can cause poor health

Cappuccino, latte or short black, coffee is one of the most commonly consumed drinks in the world. But whether it's good or bad for your health can be clarified by genetics, as a world-first study from the University of South Australia's Australian Centre for Precision Health shows that excess coffee consumption can cause poor health.

Using data from over 300,000 participants in the UK Biobank, researchers examined connections between genetically instrumented habitual coffee consumption and a full range of diseases, finding that too much coffee can increase the risk of osteoarthritis, arthropathy (joint disease) and obesity. In earlier research conducted by Professor Hyppönen and team, six cups of coffee a day were considered the upper limit of safe consumption.

Expert genetic epidemiologist, UniSA's Professor Elina Hyppönen, says understanding any risks associated with habitual coffee intakes could have very large implications for population health.

"Globally, we drink around three billion cups of coffee each day, so it makes sense to explore the pros and cons of this on our health," Professor Hyppönen says.

"Typically, the effects of coffee consumption are investigated using an observational approach, where comparisons are made against non-coffee-drinkers. But this can deliver misleading results.

"In this study, we used a genetic approach - called MR-PheWAS analysis - to establish the true effects of coffee consumption against 1117 clinical conditions. "Reassuringly, our results suggest that, moderate coffee drinking is mostly safe.

"But it also showed that habitual coffee consumption increased the risks of three diseases: osteoarthritis, arthropathy and obesity, which can cause significant pain and suffering for individuals with these conditions."

Professor Hyppönen says the prevalence of these conditions in Australia and around the world shows how important it is to determine possible causes and influencers of the diseases. "Excess coffee consumption can lead to increased risks of certain diseases," Professor Hyppönen says.

"For people with a family history of osteoarthritis or arthritis, or for those who are worried about developing these conditions, these results should act as a cautionary message. "The body generally sends powerful messages with respect to coffee consumption, so it's imperative that individuals listen to these when consuming coffee.

"While these results are in many ways reassuring in terms of general coffee consumption, the message we should always remember is consume coffee in moderation - that's the best bet to enjoy your coffee and good health too."

Notes to Editors:

***Arthropathy**, the most common form of which is arthritis, affects one in seven Australians and more than 54 million adults around the world.*

***Osteoarthritis** (a chronic and progressive arthropathy that mostly affects the hands, spine and joints such as hips, knees and ankles) affects an estimated one in 11 Australians, and 300 million people worldwide.*

***Obesity** is growing in prevalence worldwide, with nearly 40 per cent of adults over overweight and 13 percent obese. In Australia, 67 per cent of Australian adults are overweight or obese.*

https://bit.ly/3fVEYPs

Remdesivir Works Against Many Viruses. Why Aren't There More Drugs Like It?

Antivirals that work against a large number of diverse viruses would help us prepare for new diseases, but creating them is a big biological challenge

In 1947, amid the "Golden Age" of antibiotic research that yielded many of the medicines we use against bacteria today, the soil of Venezuela provided a scientific prize. Researchers at a drug company on the wane discovered chloramphenicol, a molecule that could combat a wide array of bacteria from different families. It was among the first FDA-approved broad-spectrum antibiotics and was used against typhus or meningitis. Now, chloramphenicol's side effects make it a last-resort drug, but similarly versatile treatments, referred to as broad-spectrum, remain invaluable weapons against a host of bacterial infections.

Soon after that discovery, scientists began to find ways to target another type of pathogen: viruses. The FDA approved its first antiviral (IDU, for a type of herpes) in 1963, and today we have drugs that are hyper-focused on a particular virus (like many HIV-1 treatments), some medicines that work on multiple viruses within the same family (like Tamiflu, which is approved for both major types of influenza), but precious few that stretch across viral families. The promise of antiviral drugs with a truly expansive range has remained elusive.

"That's a very challenging biological question," says Kara Carter, the president of the International Society of Antiviral Research, when asked whether a panacea for *all* viruses would be feasible. If a scientist is searching for a treatment targeting the virus itself, "There's really no common mechanism across all of them." Instead, researchers hope to expand the existing roster of broad-spectrum antivirals and find more medicines that work on all viruses of a certain family, and ideally, across more than one family.

This reality makes the search for treatments for SARS-CoV-2, the virus that causes COVID-19, all the more challenging. Currently, no broad-spectrum antiviral is approved for the treatment of all coronaviruses, of which a new strain has driven the current pandemic. Scientists are rushing to find a solution.

"If you have an antiviral that works against multiple respiratory viruses [from different families], that would be super useful," says Andrea Pruijssers, an assistant professor of research at Vanderbilt University Medical Center. "That's like shooting for the moon, but we're doing it anyway." Pruijssers researches coronavirus antivirals, including the broad-spectrum drug remdesivir, which recently became the first medication to receive FDA authorization for emergency use for COVID-19.

Why Broad-Spectrum Antivirals Are So Hard to Make

Viruses are more slippery targets than bacteria. They're often a hundred times smaller and consist only of bare-bones cellular machinery. Their tiny footprint creates a conundrum for researchers: There are simply fewer targets at which to aim antivirals, especially for drugs that would shoot for the rare viral components that remain common across diverse types of viruses.

Hepatitis C, for example, is caused by HCV viruses from *Flaviviridae*, a family that also includes the virus behind yellow fever. Some Hepatitis C treatments are so targeted that they combat only some of the six main types of HCV, and certainly not yellow fever. Scientists call this virus-pinpointing model the "one drug, one bug" approach.

An antiviral's mechanism can't be *too* generic, either. "The broader you go, the more likely you are to pick off something in the host cell," says Amesh Adalja, a senior scholar at the Johns Hopkins University Center for Health Security. For instance, a broad-spectrum antiviral called ribavirin, which fights both Hepatitis C and respiratory syncytial virus, can cause birth defects and destroy blood cells. To deal directly with the microorganisms at the root of the disease, "you want it to be very exquisitely targeted to the virus and not affect the host," Adalja says. (Broad-spectrum treatments called host-acting or host-directed antivirals are an exception to this rule, aiming for the host instead of the virus, but can come with the possibility of serious side effects.)

On top of the biological challenge of finding new broad-spectrum antiviral drugs lies an economic one. Pharmaceutical companies have little financial incentive to develop broad-spectrum drugs against emerging diseases since they have no guarantee they'll recoup the costs of research. "Big pharma is rarely interested in developing a drug against an unknown that might emerge in the future, and so consequently, the entire global response to new emerging outbreaks of viral disease is reactive rather than proactive," says microbiologist Ralph Baric, who has been investigating coronaviruses and warning of their emerging-disease potential for decades. While federal funds have bankrolled research in this area, Congress has historically been more apt to spend money on already-here crises like Ebola than on preparedness measures.

"We don't really have a drug on the shelf for all SARS-like viruses, or all Ebola-like viruses, or all flu-like viruses," Baric says. So when a virus like Ebola or SARS-CoV-2 (the novel coronavirus) jumps into humans, clinicians have few treatments to work with, and scientists must start the lengthy process of testing and developing drugs from scratch. Broad-spectrum antivirals are not miracle drugs, but they would be a helpful addition to a toolbox that is currently sparse. In a paper published last year, Adalja and another Johns Hopkins colleague called the scarcity of broad-spectrum antivirals "a major chasm in preparedness for infectious disease emergencies."

The Rise of Remdesivir

To fill that void, for the past seven years, Baric's lab has partnered with the Vanderbilt lab where Pruijssers and her colleagues work. Together, they've tested some 200,000 drugs against bat coronaviruses and identified at least two dozen that showed promise. That tally includes remdesivir, so far the only antiviral to have significantly reduced recovery times (though not mortality) for COVID-19 patients in a clinical trial.

Remdesivir's potential first drew public attention in October 2015 during an Ebola outbreak in West Africa that claimed more than 11,000 lives. The U.S. Army Medical Research Institute of Infectious Diseases announced that, in partnership with the biopharmaceutical company Gilead Sciences, it had found the first small-molecule drug that protected infected rhesus monkeys from the deadly effects of Ebola. GS-5734 (remdesivir's original name) was a fine-tuned version of a compound from Gilead's libraries that was concocted to treat other viruses. A CDC screen of 1,000 possibilities had established its broad-spectrum activity. In cells in the lab, it hampered not only Ebola viruses but also several others, including the coronavirus that caused MERS.

Remdesivir subdues a virus by interfering with replication—the way a virus copies itself. It's a common strategy among broad-spectrum antivirals because the enzymes involved tend to be conserved across many types of viruses. For example, the genetic sequences of coronaviruses' RNA polymerases are at least 70 percent identical. By contrast, the genetic code behind the "spike" that helps coronaviruses invade host cells varies more widely, Baric says.

First, the body converts remdesivir into an imposter. It becomes what's called a nucleoside analog—a genetic doppelganger that resembles adenosine, one of the four "letters" of the RNA alphabet that make up the genomes of ebolaviruses and coronaviruses. When the virus replicates, it weaves this analog into the new strand of genetic material. However, the analog's molecular makeup differs from real adenosine just enough to grind the copying process to a halt. "If the virus can't make copies of itself, the body's immune system can take over and fight off the infection," USAMRIID researcher Travis Warren explained in the 2015 announcement.

As COVID-19 swept the globe, researchers conducted an international trial of remdesivir as a treatment option. This April, the National Institutes of Health announced preliminary results: The drug reduced recovery time by 31 percent—from 15 days to 11—for severely ill COVID-19 patients, although it hadn't significantly affected the death rate. NIAID director Anthony Fauci framed the early results as a reason for optimism and a starting point for finding a better course of treatment. Experts also expect the drug to have a stronger effect when administered to patients who are at an earlier stage in their illness or who have more moderate cases of COVID-19.

EIDD-2801, another treatment option that becomes a nucleoside analog in the body, also has demonstrated broad-spectrum antiviral potential, as well as an ability to defend cells from SARS-CoV-2. It seeds the replicating coronavirus with mutations that prove lethal as the virus copies more and more of its genome. EIDD-2801, which can be administered as a pill rather than intravenously, isn't as far along in clinical trials as remdesivir. However, it appears that both can somewhat evade coronaviruses' proofreading mechanism, which (unusually for a virus) checks the copied genome's accuracy and can root out other nucleoside analogs. Both have beaten back the novel coronavirus in lab-grown versions of the airway cells SARS-CoV-2 batters. Pruijssers says both treatments are at least ten times more potent than other buzzed-about drugs, like hydroxychloroquine or camostat. Remdesivir and EIDD-2801 have also passed the laboratory safety screenings that check that they mess with only the virus' RNA and not that of the host cell, a step that derails many nucleoside analogs, as well as more advanced safety tests.

What Comes Next

Remdesivir and EIDD-2801 "aren't the only drugs that we are chasing," Baric says, though he declined to go into more detail on ongoing research. The bulk of coronavirus drug research, in Pruijssers' estimation, is predominantly focused on treatments that will work on the novel coronavirus—the crisis at hand—but not necessarily other viruses.

That eventual best treatment for COVID-19 may not be remdesivir, EIDD-2801 or any single antiviral at all. That's because stopping the virus is only part of the equation. Clinicians also must address the numerous and perplexing symptoms of the disease, and in severe cases, they must deal with the vehement immune response to the virus. Broad-spectrum antivirals could be invaluable in the short-term, especially because remdesivir and other repurposed drugs have already had their safety in humans assessed. Baric and Pruijssers both suggest that such antivirals could be especially useful when combined with other treatments.

For example, when remdesivir reached rigorous clinical trials in the Democratic Republic of the Congo as a treatment for Ebola—admittedly, a very different disease—it didn't become the recommended treatment. Although the drug reduced Ebola's mortality rate to 50 percent, it turned out that two antibody-based treatments worked better at preventing deaths.

Right now, people shouldn't expect one versatile uber-drug that routinely quashes diverse viruses affecting different organ systems. "I would emphasize that it's not going to be one broad-spectrum antiviral that works for all future pandemics we might have," says Jassi Pannu, who researched pandemic preparedness policy for Oxford University's Future of Humanity Institute and is now an incoming internal medicine resident at Stanford University Hospital. "The most likely scenario is we're going to have a suite of these drugs and a lot of them will never be used…but the goal is that you have, at least, an array of them [to try out]."

Last year, Adalja wrote that developing more broad-spectrum antivirals that work reliably within (or ideally, across) families will be "difficult" but "not impossible." He suggested increased screening of new drugs to see whether they work against more than just the virus they were designed for, the same way scientists uncovered the versatility of remdesivir. Such research needs funding, and on the federal level, more money may soon be available. "The NIH is really starting to push the concept of one drug, many bugs," Baric says, noting that the institute helped to establish the antiviral development center that sponsors his research. "They want to move, certainly the academic side of the antiviral drug development community, toward broad-based inhibitors."

But, Pannu warns, we've been here before. The early success of remdesivir suggests that broad-spectrum antivirals will get their moment in the scientific limelight. After a pandemic passes, though, the surge in interest about a multipurpose treatment wanes. This time around, doctors confronted with a new disease had no clinically proven treatments to offer COVID-19 patients. Next time could be different—if research budgets prioritize accordingly.

https://bit.ly/2Z5RSnP

Sitting in a freezer for years, potential SARS vaccine now ready for trial on usefulness against coronavirus

Thousands of doses of a potential vaccine for Severe Acute Respiratory Syndrome have been sitting in a freezer in Houston, Texas, shelved since 2016 after most of the world lost interest in the disease.

Mark Johnson

Now, four years later, they have been given new life because scientists hope they will also work for COVID-19.

Depending on the amount given to patients, anywhere from 23,000 to 230,000 doses of vaccine are currently at a storage facility called Cryogene in Houston.

"We just could not get any money. Not from the government and not from private industry," said Maria Elena Bottazzi, a professor of pediatrics at Baylor College of Medicine and one of the vaccine's developers.

Three organizations have agreed to shepherd the vaccine through clinical trials, and to ensure that it is safe and affordable. The protein-based vaccine is made using yeast, a similar method to the one employed in the manufacture of hepatitis B vaccines used around the world.

"There's a lot of knowledge and a lot of safety with this method," said Bottazzi, who is co-director of the Texas Children's Hospital Center for Vaccine Development. She said researchers hope to receive clearance from the U.S. Food and Drug Administration to start clinical trials as soon as September. Worldwide there are now about 100 vaccines against COVID-19 under development.

The three partners working to get Bottazzi's vaccine into clinical trials are Baylor College of Medicine, Texas Children's Hospital Center for Vaccine Development and PATH, a 43-year-old global nonprofit dedicated to improving public health.

Deborah Higgins, PATH's senior director for vaccine development, said that because the SARS virus and the new coronavirus "have so many similarities, we realized that there was reasonable potential for the vaccine to address the current pandemic. ..."

"Instead of having to start from ground zero in developing a vaccine, this candidate is virtually ready to go into the clinic. It is very much ahead of the game."

PATH, which works in more than 70 countries, has partnered in developing vaccines against Japanese encephalitis, meningitis A and malaria.

The SARS vaccine, known as RBD219N1, was developed by Bottazzi and colleague Peter Hotez, co-director of Texas Children's Hospital Center for Vaccine Development.

It works by targeting the key mechanism used by both SARS and the new coronavirus to infect cells.

Both viruses use their spike protein to dock onto the outside of human cells, specifically onto one part of the cell, a receptor called ACE-2. Once the protein has docked, the virus is then able to penetrate the cells.

The vaccine hinders this connection between the virus and human cell by blocking the portion of the spike protein that latches onto an ACE-2 receptor.

Bottazzi said the vaccine has been tested on animals. It has also been tested successfully on a pseudovirus, a lab-made virus that closely resembles SARS-CoV-2 but is incapable of causing disease.

Before hitting a funding wall in 2016, a consortium spent about five years and $6 million in grants from the National Institutes of Health to develop and test the vaccine. The consortium included Baylor College of Medicine, Texas Children's Hospital Center, the New York Blood Center, Walter Reed Army Institute of Research and the University of Texas Medical Branch in Galveston. Once developed, the vaccine was manufactured by the Army.

Then, in 2016, when researchers were ready to proceed to clinical trials, interest in the SARS vaccine vanished. Pharmaceutical companies weren't interested. Neither was the Army or other U.S. government agencies.

Other researchers eager to study SARS encountered similar problems. Although the disease faded away in 2004, a number of scientists worried that another coronavirus would surface.

Nevan J. Krogan, a molecular biologist at University of California, San Francisco, said he applied for a grant to do SARS research but was unable to get funding.

"There should have been a ton of research into SARS, but the money dried up," Krogan said. "It was short-sightedness, not just on the part of government agencies, but also scientists themselves."

He stressed that scientists do the peer-review work that's often used to make decisions on which grants receive funding and which do not.

Now, after four years in limbo, the Baylor team has managed to find support for its shelved vaccine.

"It's exciting to now join in this endeavor with PATH to address this important global health threat," said Hotez, co-developer of the vaccine with Bottazzi.

Bottazzi stressed that researchers hope to keep the cost of their vaccine to less than $1 or $2 a dose.

"It is becoming increasingly apparent that this virus poses great risk to low- and middle-income countries of South and Central America, Africa and Asia," she said. "Our goal is to ensure that our development efforts lead to COVID-19 vaccines with global access,

so populations can benefit in the many low-resource countries where it is so greatly needed."

https://bit.ly/2X00ObO

Surplus antioxidants are pathogenic for hearts and skeletal muscle

This discovery may have clinical importance in management of heart failure

Birmingham, Ala. - Many heart diseases are linked to oxidative stress, an overabundance of reactive oxygen species. The body reacts to reduce oxidative stress -- where the redox teeter-totter has gone too far up -- through production of endogenous antioxidants that reduce the reactive oxygen species. This balancing act is called redox homeostasis.

But what happens if the redox teeter-totter goes too far down, creating antioxidative stress, also known as reductive stress? Rajasekaran Namakkal-Soorappan, Ph.D., associate professor in the University of Alabama at Birmingham Department of Pathology, and colleagues have found that reductive stress, or RS/AS, is also pathological. This discovery, they say, may have clinical importance in management of heart failure.

They report that RS causes pathological heart enlargement and diastolic dysfunction in a mouse model. This study, published in the journal Antioxidants and Redox Signaling, was led by Namakkal-Soorappan and Pei Ping, Ph.D., David Geffen School of Medicine at the University of California-Los Angeles.

"Antioxidant-based therapeutic approaches for human heart failure should consider a thorough evaluation of antioxidant levels before the treatment," they said. "Our findings demonstrate that chronic RS is intolerable and adequate to induce heart failure."

The study used transgenic mice that had upregulated genes for antioxidants in the heart, which increased the amounts of antioxidant proteins and reduced glutathione, creating RS. One mouse line had low upregulation, and one had high upregulation, creating chronic low RS and chronic high RS, respectively, in the hearts of the mice.

The mice with high RS showed pathological heart changes called hypertrophic cardiomyopathy, and had an abnormally high heart ejection fraction and diastolic dysfunction at 6 months of age. Sixty percent of the high-RS mice died by 18 months of age.

The mice with low RS had normal survival rates, but they developed the heart changes at about 15 months of age, suggesting that even moderate RS can lead to irreversible damage in the heart over time.

Giving high-RS mice a chemical that blocked biosynthesis of glutathione, beginning at about 6 weeks of age, prevented RS and rescued the mice from pathological heart changes.

Gobinath Shanmugam, Ph.D., postdoctoral fellow in the UAB Department of Pathology, and Namakkal-Soorappan point out that a 2019 survey found about 77 percent of Americans are consuming dietary supplements every day, and within this group, about 58 percent are consuming antioxidants as multivitamins. Thus, a chronic consumption of antioxidant drugs by any individual without knowing their redox state might result in RS, which can induce pathology and slowly damage the heart.

Effect of RS on skeletal muscle

In a related study, published in the journal *Redox Biology*, Namakkal-Soorappan looked at the impact of RS on myosatellite cells, which are also known as muscle stem cells. These cells, located near skeletal muscle fibers, are able to regenerate and differentiate into skeletal muscle after acute or chronic muscle injury. The regulation of myosatellite cells is of interest given the loss of skeletal muscle mass during aging or in chronic conditions like diabetes and AIDS.

Recently, Namakkal-Soorappan reported that tilting the redox teeter-totter to oxidative stress impaired regeneration of skeletal muscle. Now, in the *Redox Biology* paper, he has shown that tilting the redox to RS also causes significant inhibition of muscle satellite cell differentiation.

Rather than genetic manipulation to induce RS, as was done in the heart study, the researchers used the chemical sulforaphane or direct augmentation of intracellular glutathione to induce RS in cultured mouse myoblast cells. Both treatments inhibited myoblast differentiation.

Finally, authors attempted to withdraw antioxidative stress by growing cells in medium without sulforaphane, which removes the RS and accelerates the differentiation. Namakkal-Soorappan and colleagues found that a pro-oxidative milieu, through a mild generation of reactive oxygen species, was required for myoblast differentiation. The researchers also showed that genetic silencing of a negative regulator of the antioxidant genes also inhibited myoblast differentiation.

Co-authors with Namakkal-Soorappan and Ping, and first-author Shanmugam, in the Antioxidants and Redox Signaling study, "Reductive stress causes pathological cardiac remodeling and diastolic dysfunction," are Silvio H. Litovsky and Rajesh Kumar Radhakrishnan, UAB Department of Pathology; Ding Wang, UCLA; Sellamuthu S. Gounder, Kevin Whitehead, Sarah Franklin and John R. Hoidal, University of Utah School of Medicine; Jolyn Fernandes and Dean P. Jones, Emory University, Atlanta, Georgia; Thomas W. Kensler, Fred Hutch Cancer Research Center, Seattle, Washington; Louis Dell'Italia, UAB Department of Medicine; Victor Darley-Usmar, UAB Department of Pathology; and E. Dale Abel, University of Iowa.

In the Redox Biology study, "Reductive stress impairs myogenic differentiation," co-authors with Namakkal-Soorappan are Sandeep Balu Shelar, UAB Department of Pathology; Dean P. Jones, Emory University; and John R. Hoidal, University of Utah School of Medicine.

Support for both studies came from National Institutes of Health grants HL118067 and AG042860, American Heart Association grant BGIA 0865015F, the University of Utah, and UAB.

In the two studies, Namakkal-Soorappan's name is listed as Namakkal S. Rajasekaran.

https://nyti.ms/3fZG7W5

Praying Mantises: More Deadly Than We Knew

New research shows these ferocious insects don't just hunt like robots.

By Cara Giaimo

A praying mantis watches intently as a fly bobs by. In less than a blink, she's snatched it up. When the tape is played back in slow motion, we see the mantis pause and calibrate, almost like an experienced baseball catcher who has realized she's dealing with a knuckleball.

It's an impressive highlight reel. As detailed in a paper published this week in Biology Letters, it's also evidence that mantises strike less like automatons and more like active hunters, calibrating their attacks to more efficiently capture their prey as it flies by at different speeds.

Predatory animals are traditionally divided into two categories based on how they catch their meals. The first group, pursuit predators, run down their prey. The action heroes of the animal world, they tend to be flashy and attention-getting, whether they're large like cheetahs or tiny like robber flies. Researchers have uncovered "extraordinary examples of how flexible their pursuit can be," said Sergio Rossoni, who performed the new study as a master's student at the University of Sussex, and is now a zoology doctoral student at the University of Cambridge.

The second group, known as sit-and-wait predators, skulk until the time is right, and then, bam — they strike. In the past, such predators were "thought to be quite stereotypical in their behavior," Mr. Rossoni said, almost like windup toys. Researchers had described praying mantis strikes in particular as always occurring "at the same rate with the same movements," he said.

Recently, though, this view has been called into question. Work on mantis shrimp, which deploy an ultrafast punch to smash their prey, has shown that they are able to vary their strike speed, and a 2016 study of praying mantises found that they displayed flexibility when "catching" stationary bugs. Mr. Rossoni and his then-supervisor Jeremy Niven, a zoology professor at the University of Sussex, decided to test praying mantises further, and see whether they varied their approach with slow or speedy prey.

For his experiment, Mr. Rossoni placed one Madagascan marbled mantis at a time on a raised platform underneath a bright light. (Other species preferred to hunt upside-down, which made filming difficult.) He then swung a target — either a dead bug, or a bead that looked like one — toward the mantis on a transparent wire.

The target could move at three speeds, each meant to approximate a different type of mantis prey. The slowest, 200 millimeters per second, is the average flight speed of a fruit fly. The fastest, 730 millimeters per second — or a little over one and a half miles per hour — mimicked a blow fly.

He put eight different mantises through dozens of swings, filming each with a high-speed camera. He then analyzed the insects' recorded moves.

The strike of a praying mantis has two phases. In the first, the approach phase, a mantis extends its arms up and outward. In the second, the sweep phase, the mantis scoops the prey out of the air and pulls it in to eat.

Mr. Rossoni and Dr. Niven found that the mantises did indeed adjust their strike speed, according to how quickly the target was moving. Most of that modulation occurred in the approach phase — when presented with a slower target, the mantises would raise their limbs more slowly or pause in the middle, in a zombielike pose.

And if they initially miscalculated the speed of their prey, the mantises would often "correct their own mistakes" with a similar pause, Mr. Rossoni said. "Considering that some of the strikes are less than a tenth of a second, this is quite extraordinary."

It also adds to a growing conversation about what insects — from wasps that can logically infer to ants that can roll down inclines — are capable of.

"Historically, they were viewed more as almost miniature robots that were following very simple sets of rules," Dr. Niven said. "I think that there is new research coming out that suggests that that rule book might be much more complicated."

https://bit.ly/2AF8Ejx

Treatment with interferon-α2b speeds up recovery of COVID-19 patients in exploratory study

Treatment with IFN-α2b was shown for the first time to improve virus clearance and decrease levels of inflammatory markers in a cohort of COVID-19 patients

Treatment with antivirals such as interferons may significantly improve virus clearance and reduce levels of inflammatory proteins in COVID-19 patients, according to a new study in *Frontiers in Immunology*. Researchers conducting an exploratory study on a cohort of confirmed COVID-19 cases in Wuhan found that treatment with interferon (IFN)-α2b significantly reduced the duration of detectable virus in the upper respiratory tract and reduced blood levels of interleukin(IL)-6 and C-reactive protein (CRP), two inflammatory proteins found in the human body. The findings show potential for the development of an effective antiviral intervention for COVID-19, which is an ongoing global pandemic caused by the novel coronavirus, SARS-CoV-2.

"Interferons are our first line of defence against any and all viruses - but viruses such as corona-viruses have co-evolved to very specifically block an interferon response", says lead author Dr Eleanor Fish of the Toronto General Hospital Research Institute & University of Toronto's Department of Immunology, adding: "This

informs us of the importance of interferons for the clearance of virus infections. Treatment with interferon will override the inhibitory effects of the virus."

Fish says that the research team considered IFN-α therapy for COVID-19 after they demonstrated interferons had therapeutic benefits during the SARS outbreak of 2002 and 2003. "My group conducted a clinical study in Toronto to evaluate the therapeutic potential of IFN-α against SARS. Our findings were that interferon treatment sped up the resolution of lung abnormalities in patients treated with interferon compared with those not treated with interferon" says Fish.

In this study, the authors examined the course of disease in a cohort of 77 individuals with con-firmed COVID-19 admitted to Union Hospital, Tongii Medical College, Wuhan, China, between January 16th and February 20th 2020. The individuals evaluated in this study consisted of only moderate cases of COVID-19, as none of the patients required intensive care or oxygen supple-mentation or intubation. Patients were either treated with IFN-α2b, arbidol (ARB), which is a broad-spectrum antiviral, or a combination of IFN-α2b plus ARB, and viral clearance was defined as two consecutive negative tests for virus at least 24 hours apart, from throat swab samples.

The researchers demonstrated a significantly different rate of viral clearance for each treatment group and notably, IFN-α2b treatment accelerated viral clearance by approximately 7 days. Treatment with IFN-α2b, whether alone or in combination with ARB, accelerated viral clearance when compared to ARB treatment alone. IFN treatment was also demonstrated to significantly reduce circulating levels of IL-6 and CRP, whether alone or in combination with ARB. The influence of age, co-morbidities and sex did not negate the effects of IFN treatment on viral clearance times or on the reduction in the inflammatory proteins IL-6 and CRP.

Despite the study's limitations of a small, non-randomised cohort, the work provides several important and novel insights into COVID-19 disease, notably that treatment with IFN-α2b accelerated viral clearance from the upper respiratory tract and also reduced circulating inflammatory biomarkers, hinting at functional connections between viral infection and host end organ damage by limiting the subsequent inflammatory response in the lungs of patients.

Fish argues, "Rather then developing a virus-specific antiviral for each new virus outbreak, I would argue that we should consider interferons as the 'first responders' in terms of treatment. Interferons have been approved for clinical use for many years, so the strategy would be to 'repurpose' them for severe acute virus infections."

As an uncontrolled, exploratory study, Fish says a randomized clinical trial is a crucial next step: "A clinical trial with a larger cohort of infected patients that are randomized to treatment with interferon-alpha or to a placebo would further this research".

In the meantime, the findings from this study are the first to suggest therapeutic efficacy of IFN-α2b as an available antiviral intervention for COVID-19, which may also benefit public health measures by shortening the duration of viral clearance and therefore slowing the tide of the pandemic.

Notes to Editors

Please link to the original research article in your reporting: Interferon-alpha2b treatment for COVID-19

https://www.frontiersin.org/articles/10.3389/fimmu.2020.01061/full

https://bit.ly/2ybjFbo

The Amazon Could Easily Be The Next Source of Coronaviruses, Scientist Warns

Human encroachment on animals' habitats is soaring there because of rampant deforestation

Paula Ramon, Afp

The next pandemic could come from the Amazon rainforest, warns Brazilian ecologist David Lapola, who says human encroachment on animals' habitats - a likely culprit in the coronavirus outbreak - is soaring there because of rampant deforestation.

Researchers say the urbanization of once-wild areas contributes to the emergence of zoonotic diseases - those that pass from animals to humans.

That includes the new coronavirus, which scientists believe originated in bats before passing to humans in China's rapidly urbanizing Hubei province, probably via a third species.

Lapola, who studies how human activity will reshape the future ecosystems of tropical forests, says the same processes are in play in the Amazon.

"The Amazon is a huge reservoir of viruses," he told AFP in an interview. "We'd better not try our luck."

The world's biggest rainforest is disappearing at an alarming rate.

Last year, in far-right President Jair Bolsonaro's first year in office, deforestation in the Brazilian Amazon surged 85 percent, to more than 10,000 square kilometers (3,900 square miles) - an area nearly the size of Lebanon.

The trend is continuing this year. From January to April, 1,202 square kilometers were wiped out, setting a new record for the first four months of the year, according to data based on satellite images from Brazil's National Space Research Institute (INPE).

That is bad news, not just for the planet but for human health, said Lapola, who holds a PhD in earth system modeling from the Max Planck Institute in Germany and works at the University of Campinas in Brazil.

"When you create ecological disequilibrium... that's when a virus can jump" from animals to humans, he said.

HIV, Ebola, dengue

Similar patterns can be seen with HIV, Ebola and dengue fever - "all viruses that emerged or spread on a huge scale because of ecological imbalances," he said.

So far, most such outbreaks have been concentrated in South Asia and Africa, often linked to certain species of bats.

But the Amazon's immense biodiversity could make the region "the world's biggest coronavirus pool," he said - referring to coronaviruses in general, not the one behind the current pandemic.

"That's one more reason not to use the Amazon irrationally, like we're doing now," he said.

And one more reason to be alarmed by the surge in deforestation by illegal farmers, miners and loggers, he added.

Bolsonaro, a climate-change skeptic who wants to open protected indigenous lands to mining and agriculture, deployed the army to the Amazon this week to fight deforestation, in a rare protective move.

But Lapola said he would rather see the government reinforce the existing environmental agency, IBAMA, which has faced staffing and budget cuts under Bolsonaro.

"I hope under the next administration we'll pay more attention to protecting what may be the planet's greatest biological treasure," Lapola said.

"We need to reinvent the relationship between our society and the rainforest."

Otherwise, the world faces more outbreaks - "a very complex process that is difficult to predict," he said.

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Antarctic Penguins Poop Out So Much Laughing Gas, It Has a Funny Effect on Researchers

Researchers went a little "cuckoo" studying them

Antarctica's king penguins emit such copious amounts of nitrous oxide, or laughing gas, via their faeces that researchers went a little "cuckoo" studying them, according to a Danish scientific study published Thursday.

"Penguin guano produces significantly high levels of nitrous oxide around their colonies," said the head of the study, Professor Bo Elberling, of the University of Copenhagen's Department of Geosciences and Natural Resource Management.

While studying colonies of king penguins on the Atlantic island of South Georgia between South America and Antarctica, "the researchers went 'cuckoo' from being surrounded by penguin poop", he said.

Besides being a strain on the climate, nitrous oxide has an effect very similar to the sedative laughing gas used at the dentist's.

"After nosing about in guano for several hours, one goes completely cuckoo. One begins to feel ill and get a headache," Elberling said.

Nitrous oxide is 300 times more polluting to the environment than carbon dioxide.

The nitrous oxide is explained by the penguin diet of krill and fish, which contains high levels of nitrogen.

Nitrogen is released from the penguins' faeces into the ground and soil bacteria then convert it into nitrous oxide, a greenhouse gas.

"While nitrous oxide emissions in this case are not enough to impact Earth's overall energy budget, our findings contribute to new knowledge about how penguin colonies affect the environment around them, which is interesting because colonies are generally becoming more and more widespread," Elberling said.

https://wb.md/2WZNmo4

COVID-19 Diary Week 4: The 'New Normal' Is Not Temporary

Normalcy is not happening

Don S. Dizon, MD

It is a widely embraced concept in oncology, both by those of us who treat cancer and those who live through its often debilitating treatments: the new normal. It describes the reality after cancer and how a life once imagined (and lived) has been permanently changed.

For my patients who have completed what I hope to be curative treatments, I talk about this shift cautiously. They are not the people they were before cancer; priorities have shifted and relationships have changed. Some of my patients grow closer to their loved ones and others grow apart. Some have become frustrated because things they once did with ease are now challenging, whether it be running, working, or concentrating. I try my best to prepare them for a life that, while not "back to normal," can be pretty great if given time. In fact, I often say that recovering from chemotherapy can take up to a year before someone feels anything resembling who they were before cancer.

This lived experience can be even more dramatic for those with metastatic cancer, who constantly need to shift their expectations, goals, and outlook as they walk a path that few of their peers will ever truly understand.

Having spoken to hundreds of cancer patients over decades, I thought I understood this. In truth, it's taken a global pandemic for me to truly appreciate this concept. And it worries me that this paradigm has not been discussed very much in the news media, where the focus has been on "opening up the economy." Those who say we aren't ready point to the lack of testing nationally, the lack of personnel for contact tracing, and in some areas, the growing numbers of infections. Those who say we must open back up point

to the economic devastation and the loss of personal liberty that has resulted from mandated stay-at-home orders.

This disconnect has me worried about something altogether different: our expectations. I'm only now realizing that opening up businesses and lifting shelter-in-place restrictions aren't going to bring back the lives we all enjoyed before the pandemic. It's going to be very different. A new normal is going to set in for all of us.

Up to this point, I've put away my suits in favor of scrubs during clinic, I wear masks when I'm out and about, and I've stayed home for the longest stretch of time in my academic career. I've taken up rotations on the inpatient service for the first time in two decades and embraced telehealth as a routine part of outpatient care.

And yet throughout, I have assumed that all of this was temporary, that once the pandemic was "over" I would simply resume my normal routine. I'm now realizing that this assumption is very wrong. Flattening the curve has not meant that this pandemic is over. Normalcy is not happening.

I see it in my 17-year-old daughter who is having a hard time dealing with the premature end of her senior year and with it the cancellation of prom and graduation. I see it in my partner who has been furloughed from his job in pediatrics. I see it in my mom who has had to cancel cruises and international trips that she was very much looking forward to.

Even as some states have started their phased openings, there isn't anything normal about it. Retail stores contend with physical distancing regulations and crowd controls, salons operate with masks on their barbers, and restaurants struggle to continue on with a skeleton crew for curbside pickup. We are opening back up in a society that has been devastated financially. I wonder whether I will ever sit in a crowded auditorium at an ASCO annual meeting again. Or enjoy a night out at the movies with my kids. Or a celebratory holiday dinner with my colleagues at a restaurant.

Professionally, I worry about what this new normal means for us as cancer specialists. Tina Rizack, MD, an oncologist colleague from Massachusetts, summed up what I think many of us are feeling: "I think medicine is going to be hit very hard. People haven't been coming in for health maintenance visits and they haven't been getting screened for cancer. We are about to see people presenting with late-stage cancers due to delays in care, and I don't think this is going to be limited to oncology. We are going to see people with more advanced disease, whether it be strokes or heart attacks, coming in too late. It's happening now and I fear it's not going to stop."

As I ponder the new normal, I wonder whether anything good will come of it. Personally, I've appreciated being home with my kids, something I realize I missed out on in traveling for work. Dr Rizack pointed out what she hopes will be a much larger contribution to our society: compassion.

"Before the pandemic, there was an anonymity in our society," she told me. "We wore headphones on the subway, looking down at our smartphones. We walked past neighbors without acknowledging them. I think now we've realized collectively just how important connection is, especially for us healthcare workers. Hopefully this translates into more moments of kindness, connectedness, and the experience of having been seen and heard—that yes, your voice and your life do indeed count."

Don S. Dizon, MD, is an oncologist who specializes in women's cancers. He is the director of women's cancers at Lifespan Cancer Institute and director of medical oncology at Rhode Island Hospital.

https://bit.ly/2WGOWwv

Global cooling event 4,200 years ago spurred rice's evolution, spread across Asia

Scientists use genomics, archeology, and climate data to reconstruct history of rice

A major global cooling event that occurred 4,200 years ago may have led to the evolution of new rice varieties and the spread of rice into both northern and southern Asia, an international team of researchers has found.

Their study, published in *Nature Plants* and led by the NYU Center for Genomics and Systems Biology, uses a multidisciplinary approach to reconstruct the history of rice and trace its migration throughout Asia.

Rice is one of the most important crops worldwide, a staple for more than half of the global population. It was first cultivated 9,000 years ago in the Yangtze Valley in China and later spread across East, Southeast, and South Asia, followed by the Middle East, Africa, Europe, and the Americas. In the process, rice evolved and adapted to different environments, but little is known about the routes, timing, and environmental forces involved in this spread.

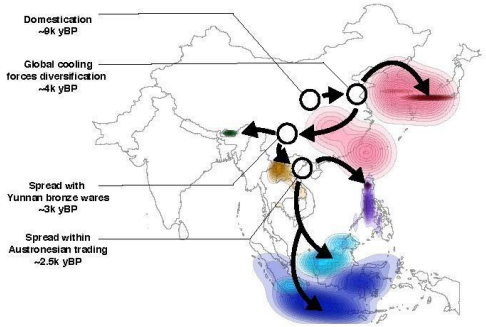
In their study, the researchers reconstructed the historical movement of rice across Asia using whole-genome sequences of more than 1,400 varieties of rice--including varieties of japonica and indica, two main subspecies of Asian rice--coupled with geography, archaeology, and historical climate data.

For the first 4,000 years of its history, farming rice was largely confined to China, and japonica was the subspecies grown. Then, a global cooling event 4,200 years ago--also known as the 4.2k event, which is thought to have had widespread consequences, including the collapse of civilizations from Mesopotamia to China--coincided with japonica rice diversifying into temperate and tropical varieties. The newly evolved temperate varieties spread in northern China, Korea and Japan, while the tropical varieties and spread to Southeast Asia.

"This abrupt climate change forced plants, including crops, to adapt," said Rafal M. Gutaker, a postdoctoral associate at the NYU Center for Genomics and Systems Biology and the study's lead author. "Our genomic data, as well as paleoclimate modeling by our collaborators, show that the cooling event occurred at the same time as the rise of temperate japonica, which grows in milder regions. This cooling event also may have led to the migration of rice agriculture and farmer communities into Southeast Asia."

"These findings were then backed up by data from archaeological rice remains excavated in Asia, which also showed that after the 4.2k event, tropical rice migrated south while rice also adapted to northern latitudes as temperate varieties," said Michael D. Purugganan, the Silver Professor of Biology at NYU, who led the study.

After the global cooling event, tropical japonica rice continued to diversify. It reached islands in Southeast Asia about 2,500 years ago, likely due to extensive trade networks and the movement of goods and peoples in the region--a finding also supported by archeological data.



***A simplified map shows the spread of rice into both northern and southern Asia following a global cooling event approximately 4,200 years before present (yBP).* Credit: Rafal Gutaker, New York University**

The spread of indica rice was more recent and more complicated; after originating in India's lower Ganges Valley roughly 4,000 years ago, the researchers traced its migration from India into China approximately 2,000 years ago.

While the researchers had thought that rainfall and water would be the most limiting environmental factor in rice diversity, they found temperature to be the key factor instead. Their analyses revealed that heat accumulation and temperature were very strongly

associated with the genomic differences between tropical and temperate japonica rice varieties.

"This study illustrates the value of multidisciplinary research. Our genomic data gave us a model for where and when rice spread to different parts of Asia, archaeology told us when and where rice showed up at various places, and the environmental and climate modeling gave us the ecological context," said Purugganan. "Together, this approach allows us to write a first draft of the story of how rice dispersed across Asia."

Understanding the spread of rice and the related environmental pressures could also help scientists develop new varieties that meet future environmental challenges, such as climate change and drought--which could help address looming food security issues.

"Armed with knowledge of the pattern of rice dispersal and environmental factors that influenced its migration, we can examine the evolutionary adaptations of rice as it spread to new environments, which could allow us to identify traits and genes to help future breeding efforts," said Gutaker.

In addition to Purugganan and Gutaker, study authors include other members of the Purugganan laboratory at the NYU Center for Genomics and Systems Biology, and collaborators at Pennsylvania State University, Universidade Nova de Lisboa in Portugal, the Crow Canyon Archaeological Center, Carnegie Mellon University, the University of Manitoba, University College London, North-West University in China, University College Dublin, and the University of California San Diego.

The research at NYU was supported by the Zegar Family Foundation and the National Science Foundation Plant Genome Research Program (IOS-1546218).

https://bit.ly/3cCKXGF

SARS-CoV-2–Fighting T Cells Found in Recovered Patients

While the finding doesn't prove people become immune to the virus after infection, it is good news for vaccine development.

Shawna Williams

Even as researchers around the world rush to develop a vaccine against the virus that causes COVID-19, and some pin their hopes on the idea that enough people will recover from infections to achieve herd immunity in the meantime, questions about whether exposure to the virus induces immunity to it have lingered. If the virus itself does not prompt immunity, a vaccine against it might not either.

Although it doesn't provide a conclusive answer, a study published yesterday (May 14) in *Cell* appears to be good news on the immunity front. Researchers at the La Jolla Institute for Immunology in California took blood from 20 adults who'd recovered from COVID-19 and exposed the samples to proteins from the SARS-CoV-2 virus. All of the patients had $CD4^+$ helper T cells that recognized the virus's spike protein, and 70 percent of them had $CD8^+$ killer T cells that responded to the same protein. "Our data show that the virus induces what you would expect from a typical, successful antiviral response," says coauthor Shane Crotty in an institute press release.

The authors also tested blood samples collected between 2015 and 2018 to see whether people who were never exposed to SARS-CoV-2 might nevertheless have some immunity to it. They detected $CD4^+$ T cell responses to SARS-CoV-2 in about half of those samples, which they suggest could be due to exposure to other coronaviruses that cause a cold.

Science notes that the results align with those of another study, led by researchers at Charité University Hospital in Berlin and reported in a preprint last month, that found $CD4^+$ T cells that recognized the spike protein in blood from 83 percent of COVID-19 patients and 34 percent of healthy people tested.

"This is encouraging data," Columbia University virologist Angela Rasmussen, who was not involved in either study, tells *Science*. Although not conclusive, the T cell response "bodes well for the development of long-term protective immunity" among people who

have recovered from COVID-19, she says, and could be useful in designing vaccines.

The results suggest that “one reason that a large chunk of the population may be able to deal with the virus is that we may have some small residual immunity from our exposure to common cold viruses,” viral immunologist Steven Varga of the University of Iowa tells *Science*. But neither study tested whether that is the case.

https://bbc.in/3cCRqdq

Coronavirus: A third of hospital patients develop dangerous blood clots

Up to 30% of patients who are seriously ill with coronavirus are developing dangerous blood clots, according to medical experts.

By Richard Galpin BBC News

They say the clots, also known as thrombosis, could be contributing to the number of people dying. Severe inflammation in the lungs - a natural response of the body to the virus - is behind their formation.

Patients worldwide are being affected by many medical complications of the virus, some of which can be fatal.

Back in March, as coronavirus was spreading across the globe, doctors started seeing far higher rates of clots in patients admitted to hospital than they would normally expect. And there have been other surprises, including the discovery of hundreds of micro-clots in the lungs of some patients.

The virus has also increased cases of deep vein thrombosis - blood clots usually found in the leg - which can be life-threatening when fragments break off and move up the body into the lungs, blocking blood vessels.

'Serious trouble'

Artist Brian McClure was rushed to hospital last month suffering from the pneumonia brought on by coronavirus. But soon after he arrived, he had a scan showing he was in a bigger fight for his life.

"I went for a lung screening and that showed blood clots in the lungs. I was told that was very dangerous," he said.

"That was when I really started to get worried. I got the picture that if I didn't improve then I would be in serious trouble."

He is now continuing his recovery at home.

"With a huge outpouring of data over the past few weeks I think it has become apparent that thrombosis is a major problem," says Roopen Arya, professor of thrombosis and haemostasis at King's College Hospital, London. "Particularly in severely affected Covid patients in critical care, where some of the more recent studies show that nearly half the patients have pulmonary embolism or blood clot on the lungs."

He believes the number of critically ill coronavirus patients developing blood clots could be significantly higher than the published data in Europe of up to 30%.

The professor's blood sciences team in the hospital has been analysing samples from patients showing how coronavirus is changing their blood making it much more sticky. And sticky blood can lead to blood clots. This change in the blood is the result of severe inflammation in the lungs, a natural response of the body to the virus. "In severely affected patients we are seeing an outpouring of chemicals in the blood and this has a knock-on effect of activating the blood clotting," says Prof Arya.

And all this ultimately causes a patient's condition to deteriorate.

According to thrombosis expert Prof Beverley Hunt, sticky blood is having wider repercussions than just blood clots - it's also leading to higher rates of strokes and heart attacks.

"And yes sticky blood is contributing to high mortality rates," she says.

Blood thinners trial

To add to all these medical challenges, there are studies showing that the blood thinners currently being used to treat the blood clots

are not always working. And ramping up doses to much higher levels risks patients suffering major bleeding which can be fatal.

The balance between treating the thrombosis and causing bleeds is "a precarious one", according to Prof Arya.

But there is now a big push to get medical teams from around the world to co-operate in finding the safest and most effective way of tackling the blood clot problem thrown up by the virus.

Trials are under way to find a standard dosage of blood thinners to be used in all countries.

However, some experts believe there could be another solution: finding a way to reduce the acute inflammation in the lungs which leads to the creation of sticky blood, the source of the problem.

https://bit.ly/36aw9qb

New ECU research finds 'Dr. Google' is almost always wrong

Online symptom checkers are only accurate about a third of the time, according to new Edith Cowan University research published in the Medical Journal of Australia today

Many people turn to 'Dr Google' to self-diagnose their health symptoms and seek medical advice, but online symptom checkers are only accurate about a third of the time, according to new Edith Cowan University (ECU) research published in the *Medical Journal of Australia* today.

The study analysed 36 international mobile and web-based symptom checkers and found they produced the correct diagnosis as the first result just 36 per cent of the time, and within the top three results 52 per cent of the time.

The research also found that the advice provided on when and where to seek health care was accurate 49 per cent of the time.

It has been estimated that Google's health related searches amount to approximately 70,000 every minute. Close to 40 per cent of Australians look for online health information to self-treat.

Lead author and ECU Masters student Michella Hill said the findings should give people pause for thought.

"While it may be tempting to use these tools to find out what may be causing your symptoms, most of the time they are unreliable at best and can be dangerous at worst," she said.

Online symptom checkers ask users to list their symptoms before presenting possible diagnoses. Triage advice is about whether - or how quickly - the user should see a doctor or go to hospital.

The 'cyberchondria' effect

According to Ms Hill, online symptom checkers may be providing a false sense of security. "We've all been guilty of being 'cyberchondriacs' and googling at the first sign of a niggle or headache," she said. "But the reality is these websites and apps should be viewed very cautiously as they do not look at the whole picture - they don't know your medical history or other symptoms.

"For people who lack health knowledge, they may think the advice they're given is accurate or that their condition is not serious when it may be."

When to see a doctor

The research found that triage advice, that is when and where to seek healthcare, provided more accurate results than for diagnoses.

"We found the advice for seeking medical attention for emergency and urgent care cases was appropriate around 60 per cent of the time, but for non-emergencies that dropped to 30 to 40 per cent," Ms Hill said. "Generally the triage advice erred on the side of caution, which in some ways is good but can lead to people going to an emergency department when they really don't need to."

A balance

According to Ms Hill, online symptom checkers can have a place in the modern health system. "These sites are not a replacement for going to the doctor, but they can be useful in providing more information once you do have an official diagnosis," she said.

"We're also seeing symptom checkers being used to good effect with the current COVID-19 pandemic. For example, the UK's National Health Service is using these tools to monitor symptoms and potential 'hot spot' locations for this disease on a national basis."

Lack of quality control

Ms Hill points to the lack of government regulation and data assurance as being major issues behind the quality of online symptom checkers. "There is no real transparency or validation around how these sites are acquiring their data," she said.

"We also found many of the international sites didn't include some illnesses that exist in Australia, such as Ross River fever and Hendra virus, and they don't list services relevant to Australia."

'The quality of diagnosis and triage advice provided by free online symptom checkers and apps in Australia' was published in the *Medical Journal of Australia*.

https://wb.md/2Tfp9t6

Vitamin D: A Low-Hanging Fruit in COVID-19?

The mainstream media was flooded this week with reports speculating on what role, if any, vitamin D may play in reducing the severity of COVID-19 infection.

Becky McCall

Observational data comparing outcomes from various countries suggest inverse links between vitamin D levels and the severity of COVID-19 responses, as well as mortality, with the further suggestion of an effect of vitamin D on the immune response to infection.

But other studies question such a link, including any association between vitamin D concentration and differences in COVID-19 severity by ethnic group.

And while some researchers and clinicians believe people should get tested to see if they have adequate vitamin D levels during this pandemic — in particular frontline healthcare workers — most doctors say the best way to ensure that people have adequate levels of vitamin D during COVID-19 is to simply take supplements at currently recommended levels.

This is especially important given the fact, that during 'lockdown' scenarios, many people are spending more time than usual indoors.

Clifford Rosen, MD, senior scientist at Maine Medical Center's Research Institute in Scarborough, has been researching vitamin D for 25 years.

"There's no randomized controlled trial for sure, and that's the gold standard," he told *Medscape Medical News*, and "the observational data are so confounded, it's difficult to know."

Whether from diet or supplementation, having adequate vitamin D is important, especially for those at the highest risk of COVID-19, he says. Still, robust data supporting a role of vitamin D in prevention of COVID-19, or as any kind of 'therapy' for the infection, are currently lacking.

Rose Anne Kenny, MD, professor of medical gerontology at Trinity College Dublin, Ireland, recently coauthored an article detailing an inverse association between vitamin D levels and mortality from COVID-19 across countries in Europe.

"At no stage are any of us saying this is a given, but there's a probability that [vitamin D] — a low-hanging fruit — is a contributory factor and we can do something about it now," she told *Medscape Medical News*.

Kenny is calling for the Irish government to formally change their recommendations. "We call on the Irish government to update guidelines as a matter of urgency and encourage all adults to take [vitamin D] supplements during the COVID-19 crisis." Northern Ireland, part of the UK, also has not yet made this recommendation, she said.

Meanwhile, Harpreet S. Bajaj MD, MPH, a practicing endocrinologist from Mount Sinai Hospital, Toronto, Canada, said: "Vitamin D could have any of three potential roles in risk for COVID-19 and/or its severity: no role, simply a marker, or a causal factor." Bajaj says — as do Rosen and Kenny — that randomized controlled trials (RCTs) are sorely needed to help ascertain whether there is a specific role of vitamin D.

"Until then, we should continue to follow established public health recommendations for vitamin D supplementation, in addition to following COVID-19 prevention guidance and evolving guidelines for COVID-19 treatment."

What is the Role of Vitamin D Fortification?

In their study in the *Irish Medical Journal*, Kenny and colleagues note that in Europe, despite being sunny, Spain and Northern Italy had high rates of vitamin D deficiency and have experienced some of the highest COVID-19 infection and mortality rates in the world. But these countries do not formally fortify foods or recommend supplementation with vitamin D.

Conversely, the northern countries of Norway, Finland, and Sweden had higher vitamin D levels despite less UVB sunlight exposure, as a result of common supplementation and formal fortification of foods. These Nordic countries also had lower levels of COVID-19 infection and mortality.

Overall, the correlation between low vitamin D levels and mortality from COVID-19 was statistically significant ($P = .046$), the investigators report. "Optimizing vitamin D status to recommendations by national and international public health agencies will certainly have…potential benefits for COVID-19," they conclude.

"We're not saying there aren't any confounders. This can absolutely be the case, but this [finding] needs to be in the mix of evidence," Kenny said.

Kenny also noted that countries in the Southern Hemisphere have been seeing a relatively low mortality from COVID-19, although she acknowledged the explanation could be that the virus spread later to those countries.

Rosen has doubts on this issue too.

"Sure, vitamin D supplementation may have worked for [Nordic countries], their COVID-19 has been better controlled, but there's no causality here; there's another step to actually prove this. Other factors might be at play," he said.

"Look at Brazil, it's at the equator but the disease is devastating the country. Right now, I just don't believe it."

Does Vitamin D Have a Role to Play in Immune Modulation?

One theory currently circulating is that, if vitamin D does have any role to play in modulating response to COVID-19, this may be via a blunting of the immune system reaction to the virus.

In a recent preprint study, Ali Daneshkhah, PhD, and colleagues from Northwestern University, Chicago, Illinois, interrogated hospital data from China, France, Germany, Italy, Iran, South Korea, Spain, Switzerland, United Kingdom, and the United States. Specifically, the risk of severe COVID-19 cases among patients with severe Vitamin D deficiency was 17.3%, whereas the equivalent figure for patients with normal Vitamin D levels was 14.6% (a reduction of 15.6%).

"This potential effect may be attributed to Vitamin D's ability to suppress the adaptive immune system, regulating cytokine levels and thereby reducing the risk of developing severe COVID-19," say the researchers.

Likewise, JoAnn E. Manson, MD, chief of the Division of Preventive Medicine at Brigham and Women's Hospital in Boston, Massachusetts, in a recent commentary for Medscape, noted evidence from an observational study from three South Asian hospitals, in which the prevalence of vitamin D deficiency was

much higher among those with severe COVID-19 illness compared with those with mild illness.

"We also know that vitamin D has an immune-modulating effect and can lower inflammation, and this may be relevant to the respiratory response during COVID-19 and the cytokine storm that's been demonstrated," she noted.

Rosen said he is willing to listen on the issue of a potential role of vitamin D in immune modulation.

"I've been a huge skeptic from the get-go, and loudly criticized the data for doing nothing. I am surprised at myself for saying there might be some effect," he told *Medscape Medical News*.

"Clearly most people don't get this [cytokine storm] but of those that do, it's unclear why they do. Maybe if you are vitamin D sufficient, it might have some impact down the road on your response to an infection," Rosen said. "Vitamin D may induce proteins important in modulating the function of macrophages of the immune system."

Ethnic Minorities Disproportionately Affected

It is also well-recognized that COVID-19 disproportionately affects black and Asian minority ethnic (BAME) individuals.

But on the issue of vitamin D in this context, one recent peer-reviewed study using UK Biobank data found "no evidence to support a potential role for vitamin D concentration to explain susceptibility to COVID-19 infection either overall or in explaining differences between ethnic groups."

"Vitamin D is unlikely to be the underlying mechanism for the higher risk observed in black and minority ethnic individuals and vitamin D supplements are unlikely to provide an effective intervention," Claire Hastie, PhD, from the University of Glasgow, UK, and colleagues conclude.

But this hasn't stopped two endocrinologists from appealing to members of the British Association of Physicians of Indian Origin (BAPIO) to get their vitamin D levels tested.

"Black and Asian Minority Ethnic (BAME) population, especially front-line staff, should get their Vitamin D3 levels checked and get appropriate replacement as required," say Parag Singhal, MD, of Weston General Hospital, Weston-Super-Mare, UK, and David C. Anderson, a retired endocrinologist, in a letter to BAPIO members seen by Medscape.

Indeed, they suggest a booster dose of 100,000 IU as a one-off for BAME healthcare staff that should raise vitamin D levels for 2 to 3 months. They refer to a systematic review that concludes that "single vitamin D3 doses ≥300,000 IU are most effective at improving vitamin D status…for up to 3 months."

Commenting on the idea, Rosen remarked that in general, the high dose- 50,000-100,000-500,000 IU given as a one-off does not confer any greater benefit than a single dose of 1000 IU per day, except that the blood levels go up quicker and higher.

"Really there is no evidence that getting to super-high levels of vitamin D confer a greater benefit than normal levels," he said. "So if healthcare workers suspect vitamin D deficiency, daily doses of 1000 IU seem reasonable; even if they miss doses, the blood levels are relatively stable."

On the specific question of vitamin D needs in ethnic minorities, Rosen said while such individuals do have lower serum levels of vitamin D, the issue is whether there are meaningful clinical implications related to this.

"The real question is whether [ethnic minority individuals] have physiologically adapted for this in other ways, because these low levels have been so for thousands of years. In fact, African Americans have lower vitamin D levels but they absolutely have better bones than Caucasians," he pointed out.

Testing and Governmental Recommendations During COVID-19

The US National Institutes of Health (NIH) in general advises 400 IU to 800 IU per day intake of vitamin D, depending on age, with those over 70 years requiring the highest daily dose. This will result in blood levels that are sufficient to maintain bone health and normal calcium metabolism in healthy people.

There are no additional recommendations specific to vitamin D intake during the COVID-19 pandemic, however.

And Rosen points out that there is no evidence for mass screening of vitamin D levels among the US population.

"US public health guidance was pre-COVID, and I think high-risk individuals might want to think about their levels, for example, someone with inflammatory bowel disease or liver or pancreatic disease. These people are at higher risk anyway, and it could be because their vitamin D is low," he said.

"Skip the test and ensure you are getting adequate levels of vitamin D whether via diet or supplement [400-800 IU] per day]," he suggested. "It won't harm."

The UK's Public Health England (PHE) clarified their advice on vitamin D supplementation during COVID-19. Alison Tedstone, PhD, chief nutritionist at PHE, said: "Many people are spending more time indoors and may not get all the vitamin D they need from sunlight. To protect their bone and muscle health, they should consider taking a daily supplement containing 10 micrograms [400 IU] of vitamin D."

However, "there is no sufficient evidence to support recommending Vitamin D for reducing the risk of COVID-19," she stressed.

Bajaj is on the advisory board of Medscape Diabetes & Endocrinology. He has serve(d) as a speaker or a member of a speakers bureau for Amgen, AstraZeneca, Boehringer Ingelheim, Janssen, Merck, Novo Nordisk, and Sanofi; has received research grants from AstraZeneca, Boehringer Ingelheim, Eli Lilly, Janssen, Merck, Novo Nordisk, Sanofi, and Valeant; has received income in an amount equal to or greater than $250 from Amgen, AstraZeneca, Boehringer Ingelheim, Canadian Collaborative Research Network, CMS Knowledge Translation, Diabetes Canada Scientific Group, Janssen, LMC Healthcare, mdBriefCase, Medscape, Meducom, Merck, Novo Nordisk, sanofi-aventis, and Valeant.

Kenny, Rosen, and Singhal have disclosed no relevant financial relationships.