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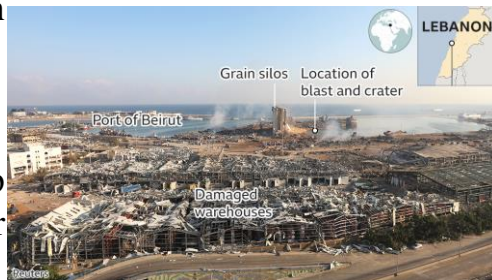
## Beirut blast was 'historically' powerful

*The blast that devastated large parts of Beirut in August was one of the biggest non-nuclear explosions in history, experts say.*

By Jonathan Amos and Paul Rincon

The Sheffield University, UK, team says a best estimate for the yield is 500 tons of TNT equivalent, with a reasonable upper limit of 1.1 kilotons. This puts it at around one-twentieth of the size of the atomic bomb dropped on Hiroshima, Japan, in 1945.

The team mapped how the shock wave propagated through the city. The group hopes its work can help emergency planners prepare for future disasters.



"When we know what the yield is from these sorts of events, we can then work out the loading that comes from that. And that tells us how to construct buildings that are more resilient," said Dr Sam Rigby from Sheffield's Blast and Impact Engineering Research Group.

"Even things like glazing. In Beirut, glazing damage was reported up 10km away from the centre of the explosion, and we know falling glass causes a lot of injuries."

The 4 August explosion was the result of the accidental detonation of approximately 2,750 tonnes of improperly stored ammonium nitrate. The blast led to some 190 deaths, as well as more than 6,000 injuries.

The Sheffield team arrived at its estimate by studying videos of the event posted on social media. When the group did this in the immediate aftermath of the blast, it produced an initial estimate in the range of 1.0-1.5 kilotons of TNT.

But this was based on only a limited set of videos, which the team

discovered may have dropped frames either when being uploaded to social media or when being pulled down.

The group has now had the chance to review many more videos from the event (16 in total) to generate a broader set of data points from which to make the calculations.

This has resulted in the yield estimate being revised downwards slightly.

"Think of it like a kid on a swing," said Dr Rigby. "If you push the child and see how far they go, you can then work out how hard the push was. That's essentially how we work out the yield."

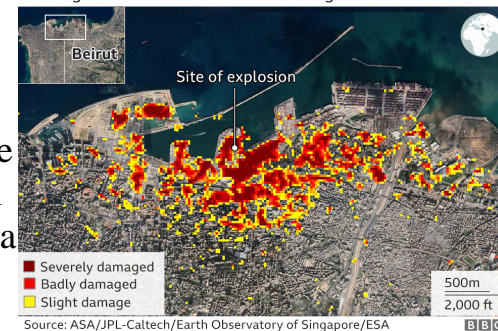
In a matter of milliseconds, the explosion released the equivalent of around 1GWh of energy. This is enough to power more than 100 homes for a year, say the researchers.

The nuclear device dropped on Hiroshima was in the range of 13-15 kilotons of TNT equivalent. By way of comparison, one of the US military's biggest conventional weapons, the GBU-43/B MOAB ("Massive Ordnance Air Blast") device, has a yield of around 11 tons.

"The Beirut explosion is interesting because it sits almost directly in a sort of no-man's land between the largest conventional weapons and nuclear weapons," said Dr Rigby. "It was about 10 times bigger than the biggest conventional weapon, and 10 to 20 times smaller than the early nuclear weapons," he told BBC News.

Dr Rigby said Beirut was in the top 10 in terms of the most powerful accidental non-nuclear explosions in history (neglecting much more energetic natural occurrences like volcanoes, asteroid impacts, etc.); and probably just outside the top 10 when some nuclear mock-up tests (such as "Minor Scale" - the largest ever

Beirut blast damage  
Damage assessment carried out 7 Aug



man-made non-nuclear explosion, which was around 3.2 kilotons of TNT) are included. Beirut was about a third of Minor Scale.

The largest accidental non-nuclear explosion in history occurred in Halifax, Nova Scotia, in 1917, when two ships (one carrying explosives) collided. That was nearly 3 kilotons of TNT equivalent, so again Beirut was around a third this size, give or take. More recently, the 2015 explosion in Tianjin (China) was only around half the yield of Beirut. This again involved ammonium nitrate.

"Beirut's certainly the most powerful non-nuclear explosion of the 21st Century," said Dr Rigby.

The new analysis is [published in the journal Shock Waves](#). Other scientists have also estimated the yield of the Beirut explosion.

The BGR group in Germany used seismic, infrasonic, and hydroacoustic data from the event; and Dr Jorge Díaz studied the physics of the evolution of the explosion fireball using Twitter videos.

"Remarkably, we all used publicly available data and found consistent results by implementing completely different methods," said Dr Díaz, who is affiliated to Indiana University, Bloomington, US.

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

## **New Reactor Design Could Produce First Ever Energy-Positive Fusion Reaction**

*A startup chasing the holy grail of energy production has published peer-reviewed scientific papers validating the physics of their approach*

By [Edd Gent](#)

Nuclear fusion has gone from a scientists' pipe dream to a technology attracting serious investment. Now one of the startups chasing this holy grail of energy production has published a series of peer-reviewed scientific papers that validate the underlying physics of their approach.

For decades, the leading hope for achieving fusion power has been the International Thermonuclear Experimental Reactor (ITER) being built in France. News earlier this year that [construction is now underway](#) has provided hope that the goal might finally be within reach.

But the project isn't expected to be fully operational until 2035, and with a price tag of at least \$22 billion, it seems there's still some way to go before the technology can go mainstream. A growing [number of startups](#) seem to think they can do things faster and cheaper, but judging the feasibility of these private endeavors has proven challenging.

Now researchers from Commonwealth Fusion Systems, one of the leaders of the pack, and their collaborators at MIT have published seven papers describing their progress in a [special issue of the Journal of Plasma Physics](#). The results are promising, suggesting their reactor design should work and could even exceed their expectations.

Like the ITER plant, the company's SPARC reactor is a tokamak, the name for a specific design of fusion reactors. The machine consists of a doughnut-shaped chamber used to contain an incredibly hot plasma made up of two different isotopes of hydrogen fusing together to create helium and a huge amount of [energy](#) as a byproduct.

Containing this roiling sea of high energy particles requires powerful magnetic fields. In conventional tokamaks they are provided by enormous electromagnets made from superconducting wires that need to be cryogenically cooled.

The secret to the SPARC reactor is that its magnets will be built from new high-temperature superconductors that require much less cooling and can produce far more powerful magnetic fields. That means the reactor can be ten times more compact than ITER while achieving similar performance.

As with any cutting-edge technology, converting principles into practice is no simple matter. But the analysis detailed in the papers suggests that the reactor will achieve its goal of producing more energy than it sucks up.

So far, all fusion experiments have required more energy to heat the plasma and sustain it than has been generated by the reaction itself.

The SPARC reactor is designed to achieve a Q factor of at least two, which means it will produce twice as much energy as it uses, but the analysis suggests that figure might actually rise to ten or more. The papers used the same physics and simulations as the ITER design team and other previous fusion experiments.

Martin Greenwald, deputy director of MIT's Plasma Science and Fusion Center, [said in a press release](#) that there are still many details to work out, particularly when it comes to actually designing and building the machine.

But the results suggest there are no major obstacles and that they should be able to meet their goal of starting construction midway through next year.

The next major milestone for the group will be the successful demonstration of the magnet technology at the heart of their design. Commonwealth [said in a press release](#) that they hope to demonstrate a 20 Tesla large-bore magnet in 2021. If everything remains on track they expect SPARC to demonstrate the first ever energy-positive fusion reaction by 2025, paving the way for a commercial fusion power plant the company calls ARC.

Cary Forest, a physicist at the University of Wisconsin, [told the \*New York Times\*](#) that the group's timelines might be a little ambitious, but the results suggest that the reactor will work as they hope. It seems like the hope of near-limitless clean energy may not be as far off as we thought.

<https://bit.ly/2GPUK10>

## Humans Are All More Closely Related Than We Commonly Think

*Humanity's most recent common ancestor and so-called genetic isopoint illustrate the surprising connections among our family trees*

By [Scott Hershberger](#)

The late esteemed English actor Christopher Lee traced his ancestry directly to Charlemagne. In 2010 Lee released a symphonic metal album paying homage to the first Holy Roman emperor—but his enthusiasm may have been a tad excessive. After all, says [geneticist Adam Rutherford](#), “literally everyone” with European ancestry is directly descended from Charlemagne.

The family tree of humanity is much more interconnected than we tend to think. “We’re culturally bound and psychologically conditioned to not think about ancestry in very broad terms,” Rutherford says. Genealogists can only focus on one branch of a family tree at a time, making it easy to forget how many forebears each of us has.

Imagine counting all your ancestors as you trace your family tree back in time. In the  $n$ th generation before the present, your family tree has  $2^n$  slots: two for parents, four for grandparents, eight for great-grandparents, and so on. The number of slots grows exponentially. By the 33rd generation—about 800 to 1,000 years ago—you have more than eight billion of them. That is more than the number of people alive today, and it is certainly a much larger figure than the world population a millennium ago.

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This seeming paradox has a simple resolution: “Branches of your family tree don’t consistently diverge,” Rutherford says. Instead “they begin to loop back into each other.” As a result, many of your ancestors occupy multiple slots in your family tree. For example,

“your great-great-great-great-great-grandmother might have also been your great-great-great-great-aunt,” he explains.

The consequence of humanity being “incredibly inbred” is that we are all related much more closely than our intuition suggests, Rutherford says. Take, for instance, the last person from whom everyone on the planet today is descended. In 2004 mathematical modeling and computer simulations by a group of statisticians led by Douglas Rohde, then at the Massachusetts Institute of Technology, indicated that [our most recent common ancestor](#) probably lived no earlier than 1400 B.C. and possibly as recently as A.D. 55. In the time of Egypt’s Queen Nefertiti, someone from whom we are all descended was likely alive somewhere in the world.

Go back a bit further, and you reach a date when our family trees share not just one ancestor in common but *every* ancestor in common. At this date, called the genetic isopoint, the family trees of any two people on the earth now, no matter how distantly related they seem, trace back to the same set of individuals. “If you were alive at the genetic isopoint, then you are the ancestor of either everyone alive today or no one alive today,” Rutherford says. Humans left Africa and began dispersing throughout the world at least 120,000 years ago, but the genetic isopoint occurred much more recently—somewhere between 5300 and 2200 B.C., according to Rohde’s calculations.

At first glance, these dates may seem much too recent to account for long-isolated Indigenous communities in South America and elsewhere. But “genetic information spreads rapidly through generational time,” Rutherford explains. Beginning in 1492, “you begin to see the European genes flowing in every direction until our estimates are that there are no people in South America today who don’t have European ancestry.”

In fact, even more recent than the global genetic isopoint is the one

for people with recent European ancestry. Researchers [using genomic data](#) place the latter date around A.D. 1000. So Christopher Lee’s royal lineage is unexceptional: because Charlemagne lived before the isopoint and has living descendants, everyone with European ancestry is directly descended from him. In a similar vein, nearly everyone with Jewish ancestry, whether Ashkenazic or Sephardic, [has ancestors who were expelled from Spain](#) beginning in 1492. “It’s a very nice example of a small world but looking to the past,” says Susanna Manrubia, a theoretical evolutionary biologist at the Spanish National Center for Biotechnology.

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Not everyone of European ancestry carries genes passed down by Charlemagne, however. Nor does every Jew carry genes from their Sephardic ancestors expelled from Spain. People are more closely related genealogically than genetically for a simple mathematical reason: a given gene is passed down to a child by only one parent, not both. In a simple statistical model, Manrubia and her colleagues showed that the average number of generations separating two random present-day individuals from a common *genealogical* ancestor [depends on the logarithm of the relevant population’s size](#).

For large populations, this number is much smaller than the population size itself because the number of possible genealogical connections between individuals doubles with each preceding generation. By contrast, the average number of generations separating two random present-day individuals from a common *genetic* ancestor is linearly proportional to the population size because each gene can be traced through only one line of a person’s family tree. Although Manrubia’s model unrealistically assumed the population size did not change with time, the results still apply in the real world, she says.

Because of the random reshuffling of genes in each successive

generation, some of your ancestors contribute disproportionately to your genome, while others contribute nothing at all. According to calculations by geneticist Graham Coop of the University of California, Davis, you carry genes from [fewer than half of your forebears from 11 generations back](#). Still, all the genes present in today's human population can be traced to the people alive at the genetic isopoint. "If you are interested in what your ancestors have contributed to the present time, you have to look at the population of all the people that coexist with you," Manrubia says. "All of them carry the genes of your ancestors because we share the [same] ancestors."

And because the genetic isopoint occurred so recently, Rutherford says, "in relation to race, it absolutely, categorically demolishes the idea of lineage purity." No person has forebears from just one ethnic background or region of the world. And your genealogical connections to the entire globe mean that not too long ago your ancestors were involved in every event in world history.

So the next time you hear someone claim to be descended from royalty, take heart: you are, too. "You are very special, and you are very generic, in a sense," Manrubia says.

<https://go.nature.com/36ROtNh>

**Japan's government must seek out expert scientists**  
*Ministers can be bizarrely ignorant of the pressing scientific and technical issues of the day*

[Yoshiyasu Takefuji](#)

I agree with your argument that the successor to Japan's prime minister Shinzō Abe must embrace diversity, diplomacy and better regulation in science ([Nature 585, 159; 2020](#)). However, such policy advances depend on advice from expert scientists, which is not solicited under Japan's present political system.

Japan's political parties are faction-ridden. In a quest for consensus, the prime minister appoints the ministers recommended by each

faction. These ministers can be bizarrely ignorant of the pressing scientific and technical issues of the day: Japan's cybersecurity minister, for example, claims that he has never used a computer (see [go.nature.com/32kd98a](https://go.nature.com/32kd98a)).

If an appointee's background means that they are unsuited to the task they are charged with, they will call on advice from other government officials.

Instead, ministers should follow the practice of other democratic nations and call in experts to advise on policy. Only then can the government genuinely improve how science is run.

*Nature 586, 200 (2020) doi: 10.1038/d41586-020-02813-4*

<https://bit.ly/3LA2cg1>

**Scientists Identify 24 Potentially 'Superhabitable' Exoplanets**

*24 extrasolar planets that may have conditions more suitable for life than Earth*

A team of geobiologists and astronomers from the United States and Germany has identified 24 extrasolar planets that may have conditions more suitable for life than Earth. Those alien worlds are older, a little larger, slightly warmer and possibly wetter than our own planet.

"The 24 top contenders for superhabitable planets are all more than 100 light-years away, but our study could help focus future observation efforts, such as from NASA's James Web Space Telescope, NASA's LUVIOR (Large UV/Optical/IR Surveyor) space observatory and ESA's PLATO (PLANetary Transits and Oscillations of stars) space telescope," said [Professor Dirk Schulze-Makuch](#), a geobiologist at Washington State University and the Technical University in Berlin.

"With the next space telescopes coming up, we will get more information, so it is important to select some targets."

"We have to focus on certain planets that have the most promising

conditions for complex life. However, we have to be careful to not get stuck looking for a second Earth because there could be planets that might be more suitable for life than ours.”

In the study, Professor Schulze-Makuch and his colleagues, [Dr. Rene Heller](#) from the Max Planck Institute for Solar System Research and [Dr. Edward Guinan](#) from Villanova University, identified superhabitability criteria and searched among the 4,500 known exoplanets for good candidates.

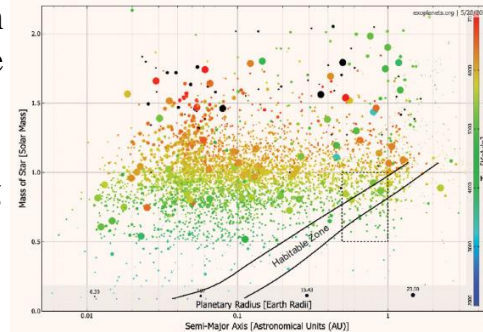
They selected stellar systems with probable terrestrial planets orbiting within the host star’s liquid water habitable zone from the [Kepler Object of Interest Exoplanet Archive](#) of transiting exoplanets.

“While the Sun is the center of our Solar System, it has a relatively short lifespan of less than 10 billion years,” they said.

“Since it took nearly 4 billion years before any form of complex life appeared on Earth, many similar stars to our Sun, called G-type stars, might run out of fuel before complex life can develop.”

“In addition to looking at systems with cooler G-type stars, we also looked at systems with K-dwarf stars, which are somewhat cooler, less massive and less luminous than our Sun. K stars have the advantage of long lifespans of 20 to 70 billion years.” “This would allow orbiting planets to be older as well as giving life more time to advance to the complexity currently found on Earth.”

Star-planet distances (along the abscissa) and mass of the host star (along the ordinate) of roughly 4,500 confirmed and candidate exoplanets. The temperatures of the stars are indicated with symbol colors (see color bar). Planetary radii are encoded in the symbol sizes (see size scale at the bottom). The conservative habitable zone,



defined by the moist-greenhouse and the maximum greenhouse limits, is outlined with black solid lines. Stellar luminosities required for the parameterization of these limits are shown along the ordinate of the diagram. The dashed box refers to the region shown in the next figure. Image credit: Schulze-Makuch *et al*, doi: 10.1089/ast.2019.2161. However, to be habitable, planets should not be so old that they have exhausted their geothermal heat and lack protective geomagnetic fields.

Earth is around 4.5 billion years old, but the scientists argue that the sweet spot for life is a planet that is between 5 billion to 8 billion years old.

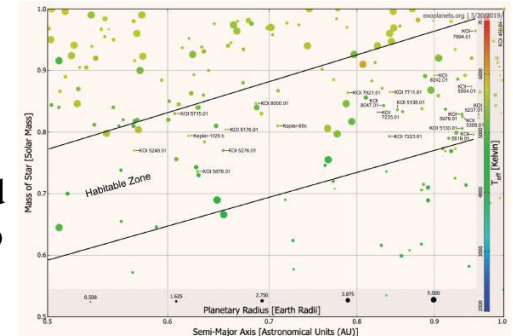
“Size and mass also matter. A planet that is 10% larger than the

Earth should have more habitable land,” they said. “One that is about 1.5 times Earth’s mass would be expected to retain its interior heating through radioactive decay longer and would also have a stronger gravity to retain an atmosphere over a longer time period.”

“Water is key to life and a little more of it would help, especially in the form of moisture, clouds and humidity.”

The habitable zone around K-dwarf stars, the potential site for superhabitable planets. Twenty-four confirmed and candidate exoplanets that are smaller than 2 Earth radii are labeled with name tags. Uncertainties in the observed stellar, planetary, and orbital parameters propagate into the planetary radius measurements, which is why we include planets as large as 2 Earth radii, although truly superhabitable planets might be restricted to radii less than 1.1 Earth radii. Image credit: Schulze-Makuch *et al*, doi: 10.1089/ast.2019.2161.

A slightly overall warmer temperature, a mean surface temperature



of about 5 degrees Celsius (or about 8 degrees Fahrenheit) greater than Earth, together with the additional moisture, would be also better for life.

This warmth and moisture preference is seen on Earth with the greater biodiversity in tropical rain forests than in colder, drier areas. Among the 24 top planet candidates none of them meet all the criteria for superhabitable planets, but one has four of the critical characteristics, making it possibly much more comfortable for life than our home planet.

“It’s sometimes difficult to convey this principle of superhabitable planets because we think we have the best planet,” Professor Schulze-Makuch said. “We have a great number of complex and diverse lifeforms, and many that can survive in extreme environments. It is good to have adaptable life, but that doesn’t mean that we have the best of everything.”

The team’s [paper](#) was published in the journal *Astrobiology*.

Dirk Schulze-Makuch et al. *In Search for a Planet Better than Earth: Top Contenders for a Superhabitable World*. *Astrobiology*, published online September 18, 2020; doi: 10.1089/ast.2019.2161

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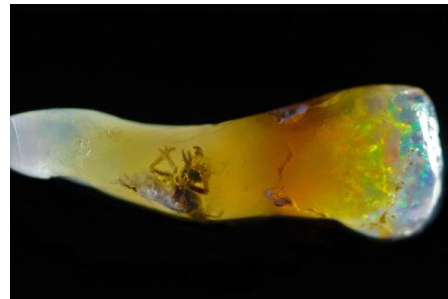
## Rare Fossil Reveals Cicada Entombed in Opal

*An insect trapped in a precious gem points to new places to search for ancient life*

By [Carolyn Wilke](#)

A bug trapped in a precious gem could offer new clues in the hunt for ancient life on Earth and Mars.

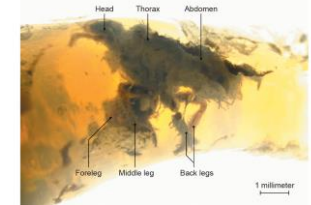
The opal, pulled from rock in Indonesia and nicknamed “Beverly,” contains the shell of a tiny cicada nymph. In June in *Scientific Reports*, [researchers explained how it likely formed](#).



*Cicada encased in opal specimen.* Credit: Brian T. Berger @velvetboxsociety

Other opal fossils have been found among silica-containing rocks that form near geysers, says Boris Chauviré, a geologist at Grenoble Alpes University in France. Hot water dissolves these rocks; when the resulting silica-rich fluid cools, it can harden to form the shimmery gem—sometimes filling in spaces left by decayed organisms or trapping creatures' bodies.

But this fossil comes from soil made by volcanic rocks eroding, and it is the first animal found entombed in opal that formed this way. Knowing this can happen, Chauviré says, suggests new places to look for ancient life.



Source: “Arthropod Entombment in Weathering-Formed Opal: New Horizons for Recording Life in Rocks,” by Boris Chauviré et al., in *Scientific Reports*, Vol. 10, Article No. 10575; 2020. <https://doi.org/10.1038/s41598-020-67412-9>

This kind of opal formation is in fact more common than the hydrothermal process, but it is slower and thus considered less likely to preserve traces of life. But the team found a layer of zeolite, a silica-rich mineral, coating the exoskeleton; the researchers' analysis suggests that the substance crystallized on the shell while it was buried in soil and exposed to silica-carrying water, preserving its structure before the surrounding liquid eventually formed opal.

“This is the first time I've seen this type of preservation,” says Frances Westall, a geologist and astrobiologist at CNRS in Orléans, France, who was not involved in the study. She says the viability of this process—somewhat analogous to more common cases of [insects preserved in amber](#), a fossilized tree resin—opens up more possibilities for finding evidence of ancient life. “The early Earth was a volcanic environment like Indonesia,” she says. “And so was early Mars.”

“Now we know that all kinds of silica can contain this kind of fossil

or biomolecules,” Chauviré says. Opal fossils that formed in volcanic settings such as early Earth or early Mars could reveal ancient underground critters that are not typically preserved in sedimentary rock or amber, he adds: “The future Jurassic Park can

<https://bit.ly/33HJ3Cw>

## Study Points to Novel Role for Microglia in Down Syndrome

*Overactive immune cells identified in a mouse model and in postmortem human brain tissue may offer a potential therapeutic target for cognitive delays associated with the condition, researchers report.*

[Catherine Offord](#)

Overactivation of the brain’s immune cells, called microglia, may play a role in cognitive impairments associated with Down syndrome, according to research published today (October 6) in [Neuron](#). Researchers in Italy identified elevated numbers of the cells in an inflammation-promoting state in the brains of mice with a murine version of the syndrome as well as in postmortem brain tissue from people with the condition. The team additionally showed that drugs that reduce the number of activated microglia in juvenile mice could boost the animals’ performance on cognitive tests.

“This is a fabulous study that gives a lot of proof of principle to pursuing some clinical trials in people,” says Elizabeth Head, a neuroscientist at the University of California, Irvine, who was not involved in the work. “The focus on microglial activation, I thought, was very novel and exciting,” she adds, noting that more research will be needed to see how the effects of drugs used in the study might translate from mice to humans.

Down syndrome is caused by an extra copy of part or all of human chromosome 21, and is the most commonly occurring chromosomal condition in the US. Children with Down syndrome often

experience cognitive delays compared to typically developing children, although there’s substantial variation and the effects are usually mild or moderate. People with the syndrome also have a higher risk of certain medical conditions, including Alzheimer’s disease.

A number of studies have identified elevated levels of inflammation in people with Down syndrome, while separate research has also connected inflammation to cognitive delay or decline in people and research animals. In their study, neuroscientists Laura Cancedda, Laura Perlino, Giovanni Morelli, and Bruno Pinto of the Italian Institute of Technology in Genoa and colleagues set out to investigate a role for microglia—which, when in a so-called activated state, release cytokines known to promote neuroinflammation.

The team focused first on a mouse model of the condition, in which part of chromosome 16—the murine equivalent of human chromosome 21—is triplicated. These so-called Dp(16) mice show some of the traits seen in people with Down syndrome, including delayed development and difficulties with motor and cognitive skills, but not the dysfunctional neurogenesis or other abnormalities in brain development characteristic of some other mouse models of the condition and people with Down syndrome.

Comparing the brains of juvenile Dp(16) mice with those of control animals, the researchers didn’t find any differences in the overall numbers of microglia. However, Dp(16) mice had higher numbers of microglia in an activated state—they showed cell morphology, electrophysiology, and protein expression patterns associated with neuroinflammation.

The fact that they’re getting the same signatures in their young mice and they’re seeing something similar in younger human brains, I thought was really convincing.

—Elizabeth Head, University of California, Irvine



The researchers next knocked out some of these activated microglia in the brains of Dp(16) mice—either by feeding animals a drug that reduces the overall number of microglia, or by injecting the animals with acetaminophen, an anti-inflammatory drug that helps inhibit microglial activation, once a day for three days. Both sets of treated mice performed better on lab measures of cognition—such as discrimination between familiar and unfamiliar objects—than Dp(16) mice that hadn't been treated. Testing acetaminophen on a different mouse model of Down syndrome produced similar results. The effect of acetaminophen wore off relatively quickly, Morelli notes: mice tested a couple weeks after their last injection showed microglial morphology and cognitive performance similar to that of untreated mice. Additional experiments showed that the drug didn't have a significant effect in adult animals, suggesting that microglial activation is particularly relevant during earlier stages of brain development, Cancedda adds.

To connect their findings to humans, the researchers examined the hippocampi of postmortem brains from people with Down syndrome who died before they reached 40. An analysis of gene and protein expression patterns and cell morphology revealed the same tell-tale signs of microglia activation that the team had found in mice.

Tarik Haydar, a neuroscientist at Children's National Hospital in Washington, DC, who was not involved in the work, says that he was impressed by the study's thoroughness. How microglia influence the developing brain, particularly as it relates to Down syndrome, has been largely unknown, he adds. The study authors "not only asked that question, they answered it quite comprehensively."

Both Haydar and Head praise the team's use of two separate mouse models and inclusion of postmortem human brain tissue, noting that similar findings across all three provide good evidence for

microglial cells' relevance in Down syndrome. "The fact that they're getting the same signatures in their young mice and they're seeing something similar in younger human brains, I thought was really convincing," says Head.

In an email to *The Scientist*, Victoria Puig, a neuroscientist at the Hospital del Mar Medical Research Institute in Barcelona, calls the study a "tour de force" and praises its use of multiple techniques. She adds that while the findings provide evidence "that abnormal microglia may be a possible cause" of cognitive delays, Down syndrome "is a complex syndrome that combines alterations not only in the brain immune system but also in other systems," including the cardiovascular system and wider patterns of gene expression. "Chronic treatment with anti-inflammatory drugs may not be sufficient" to treat cognitive problems in people, she writes, although it's worth scientists investigating to see if it could help.

Cancedda says that researchers will need to learn more about how and over what timescale acetaminophen, a widely available and relatively safe drug, acts to reduce cognitive problems in the juvenile mice they studied, with an eye to possible clinical trials in the future. She cautions that there's still a lot left to understand about acetaminophen's effects on cognition and that people should not experiment with taking the drug outside a clinical setting.

Head agrees that the findings provide good justification for studies in humans, but should be interpreted with caution until carefully controlled trials have provided more information—not least because of potentially harmful side-effects or interactions between acetaminophen and other medications.

"The proper way to do this, just as the authors suggest, is to do a controlled clinical trial," says Head. "I would love to see a clinical trial do that."

*B. Pinto et al., "Rescuing over-activated microglia restores cognitive performance in juvenile animals of the Dp(16) mouse model of Down syndrome," [Neuron](https://doi.org/10.1016/j.neuron.2020.09.010), doi:10.1016/j.neuron.2020.09.010, 2020.*

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

## Irregular Menstruation Was Just Linked to Early Death. Here's Why That's Important

*Length and quality of a person's menstrual cycle can be a crucial indicator of overall health*

[Carly Cassella](#)

The length and quality of a person's menstrual cycle can be a crucial indicator of their overall health, and new research suggests doctors should be monitoring this cycle in adolescence and later adulthood.

A 24-year-long study on nearly 80,000 healthy nurses in the United States has now provided some of the first real evidence on menstruation and premature death.

According to the longitudinal research, which began in 1989, people who experience irregular and long menstrual cycles in adolescence and throughout adulthood are more likely to die before the age of 70 compared to those with shorter and more regular cycles. This link was particularly strong for cardiovascular-related deaths and, to a lesser extent, [cancer](#)-related deaths.

"What this study will hopefully achieve is to raise awareness about menstrual irregularity, increase education and encourage women and doctors to consider the menstrual cycle when assessing health," [says](#) King's College reproductive physiologist Kim Jonas, who was not involved in the research.

"However, this study does not mean that all women who have experienced irregular menstrual cycles should be concerned. There is a lot more research to be done in this area and many factors are likely to be at play."

Menstruation is sometimes called the '[fifth vital sign](#)' - following temperature, pulse, breathing and blood pressure - and irregular cycles have been linked to a whole bunch of other health factors, including sexual and reproductive disease, bone and heart disease,

cancer, mental health problems, and other chronic health conditions. This doesn't mean irregular menstruation is *causing* these health effects; it might simply be that whatever causes a longer and more irregular cycle reflects poorer health overall.

Given this association, it's not altogether surprising that irregular menstruation is linked to early death, and yet health care providers [often fail to ask](#) about a patient's menstrual cycle when assessing their overall cardiovascular health.

Investigating possible correlations is not easy. Large, reliable datasets on menstruation are [hard to come by](#), and most of these results are based on self-reported surveys or menstrual tracking apps.

The current study is plagued by some of the same limitations, although its length and high follow-up rate with volunteers is impressive.

The research is based on a large cohort of nurses in the United States, aged 25 to 42 years, who were sent mailed or online questionnaires every two years to collect information on their lifestyle, diet, medical history and any disease.

At the start of the study, in 1989, female nurses were asked to recall their menstrual cycles during adolescence (between 14- and 17-years-old) and in early adulthood (between 18- and 22 years-old).

In 1993, the same cohort was asked about the usual length and regularity of their current menstrual cycles, when they were aged between 29 and 46 years.

"We found that the risk of premature mortality was higher among women who reported long or irregular cycles later in life," the authors [write](#).

While this might be due to a diminished recall of previous menstrual cycles in earlier life, it could also be a sign of persistent poor health.

In the older age groups, those who experienced menstruation cycles

longer than 40 days were more likely to die prematurely than those who reported a more typical cycle of 26 to 31 days.

This was particularly strong among female nurses who experienced continuous irregular cycles in adolescence and early adulthood, and also those who smoked.

This latter finding makes sense, as smoking is known to impact cardiovascular, immune and metabolic health, and irregular menstruation might be a sign of poor health in these areas.

"This interaction, however, should be interpreted with caution given the marginal statistical significance of the tests," the authors [warn](#).

What's more, most of the nurses in this study were white women of the same profession, and this career requires irregular hours of work, which can impact long-term health and disrupt menstrual regularity.

More research is needed to tease out the associations between menstruation and potentially fatal health conditions, but the new study provides some of the strongest indication to date that irregular menstruation could be linked to poorer health, whether in adolescence or in later adult life.

"These relations were also stronger when long and irregular cycles were consistently present during adolescence and throughout adulthood," the authors [write](#).

Even when other influential factors, like age, weight, lifestyle and family medical history, were taken into account, the results stayed the same, although the authors note they can't be sure they haven't missed out on other contributing factors.

Jacqueline Maybin, a research fellow and gynaecologist at the University of Edinburgh, [said](#) the methods of the study were sound and the results important, but for those with irregular periods there's no reason to freak out.

"It is also important to remember that irregular menstruation is a symptom and not a diagnosis. Therefore, a specific underlying cause of irregular menstruation may increase the risk of premature

death, rather than the irregular bleeding, per se."

For instance, the authors of the current study speculate that the link between premature death and an irregular cycle might sometimes reflect a disrupted hypothalamic-pituitary-ovarian axis, which is the part of the brain that closely controls female hormones, and is a sign of overall general health.

Given that a random trial is unachievable, the authors of the new study [say](#) theirs is the "best available evidence for understanding the long-term health consequences of menstrual cycle characteristics."

Primary care providers should therefore ask patients about their menstrual cycle throughout adolescence and adulthood, as this could be a key factor in assessing their overall health.

"This study should not be a cause of concern for all young women with irregular and/or long menstrual cycles as there are many other factors involved," [says](#) maternal scientist Rachel Tribe from Kings College London, who was not involved in the study.

"But I would hope that the information would raise awareness and encourage healthcare providers (as well as women) to investigate irregular menstrual cycles; an approach that has potential to improve reproductive health and subsequent longer term outcomes."

The study was published in [BMJ](#).

<https://bit.ly/34ETwy3>

## **Faraway Magma Reservoirs Complicate Volcano Monitoring**

*One third of volcanoes may have molten reserves kilometers away*

By [Katherine Kornei](#)

Magma—the molten rock that nourishes volcanoes—can lurk in underground pockets surprisingly far from where it emerges, new research shows. This means the instruments placed on a volcano's flanks might fail to pick up signs of moving magma that can signal

an [impending eruption](#).

University of Oregon volcanologist Allan Lerner and his colleagues focused on 56 volcanoes in subduction zones (geologically active areas where one tectonic plate is diving under another) on five continents for a new paper, [published in July](#) in *Geophysical Research Letters*. Compiling volcano data from other studies, the team estimated the center of each volcano's magma reservoir and compared it with the estimated center of the volcano's aboveground portion. The reservoirs had been found through processes such as measuring the earth's surface moving up or down and tracing how the planet's crust conducts electricity.

The researchers calculated that roughly one third of volcanoes were more than four kilometers away from their magma reservoirs. Five volcanoes, including two in Japan, two in Indonesia and one in Mexico, had offsets of more than 10 kilometers. "It was a surprise," Lerner says, because a long-standing tenet of volcanology is that magma reservoirs are located directly underneath volcanoes.

Advertisement

Offset magma reservoirs have been reported before, but the researchers say their investigation is the first to focus on an ensemble of volcanoes. Thanks to their large sample size, Lerner and his collaborators were also able to demonstrate correlations. They showed that smaller volcanoes tended to be farther from their magma reservoirs than larger volcanoes. This makes sense, the team suggests, because geologic structures such as fault lines essentially create an underground obstacle course for magma. The large quantities of magma that feed big volcanoes carry enough heat to blow straight through such natural boundaries, but the smaller reservoirs associated with smaller volcanoes must forge convoluted paths to the surface. "In small volcanoes, the magma that ascends is kind of at the mercy of preexisting crustal structures," Lerner says.

These results have implications for how volcanoes are monitored. Researchers usually aim to place ground-based instruments on or near a volcano, says Diana Roman, a volcanologist at the Carnegie Institution for Science, who was not involved in the research. But this new study indicates that such a strategy might not be best. "This tells us we should be looking farther afield, especially for volcanoes with relatively small edifices," Roman says.

Studying more volcanoes, including those not in subduction zones, would be valuable to see if these same trends persist, Lerner says: "A very clear next direction would be to expand this study to look at volcanoes in other tectonic settings."

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

## **Newly discovered viruses suggest 'German measles' jumped from animals to humans**

*Researchers found one of the viruses related to rubella in apparently healthy cyclops leaf-nosed bats caught in Uganda*

By [Ann Gibbons](#)

The virus that causes rubella, or German measles, finally has company. Scientists had never identified close relatives of the virus, leaving it as the only member of its genus, *Rubivirus*. But with a report in this week's issue of *Nature*, [rubella has gained a family](#). One of its two newfound relatives infects bats in Uganda; the other killed animals from three different species in a German zoo and was found in wild mice living nearby as well.

The findings strongly suggest that at some point in the past, a similar virus jumped from animals to humans, giving rise to today's rubella virus, the researchers say. Although neither of the new viruses is known to infect humans, the fact that a related virus jumped species raises concerns that the two viruses or other, as-yet-unknown relatives could cause human outbreaks. "We would be remiss not to be concerned, given what's going on in the world today," says epidemiologist Tony Goldberg of the University of

Wisconsin, Madison, a senior author of the study.

Highly infectious, the rubella virus usually causes rashes and fever, but in pregnant women it can lead to miscarriages, stillbirth, and babies born with congenital rubella syndrome, which includes deafness and eye, heart, and brain problems. An estimated 100,000 newborns are affected by the syndrome annually, mostly in Africa, the western Pacific, and the eastern Mediterranean; in many other countries the measles, mumps, and rubella (MMR) vaccine has made it a rarity.



*Researchers found one of the viruses related to rubella in apparently healthy cyclops leaf-nosed bats caught in Kibale National Forest in Uganda. Emily Julka*

Goldberg and his former graduate student Andrew Bennett discovered one of the new viruses in apparently healthy cyclops leaf-nosed bats, netted at night in Kibale National Park in Uganda. They named it ruhugu virus, after the Ruteete region of Uganda and the local word for bat. The architecture of ruhugu's genome is identical to that of the rubella virus, and 56% of the amino acids in its eight proteins matched those in rubella. The protein that interacts with the host's immune cells was almost identical in both viruses.

As they were getting ready to publish, the researchers learned that a team led by Martin Beer at the Friedrich-Loeffler Institute had detected another rubella relative in brain tissue from a donkey, a kangaroo, and a capybara—a giant rodent native to South America—that all died from encephalitis, an inflammation of the brain, at an unnamed zoo. They found the same virus in wild yellow-necked field mice caught in the zoo or within a 10-kilometer radius. The mice appeared to be fine, suggesting they were a natural reservoir from which the virus spilled over to the zoo animals. Comparing their data, the teams realized their viruses were related,

although ruhugu was closer to rubella than the second relative, rustrela virus, named after a lagoon in the Baltic Sea. The teams decided to publish jointly.

Two other viruses that primarily affect children, measles and mumps, also came from animals, Goldberg notes. “Now we know that every disease in the letters of the MMR vaccine has a zoonotic origin,” he says. Given the genetic distance between rubella and the ruhugu and rustrela viruses, the researchers don't think either of them made the jump to humans—but they suspect they'll find other *Rubiviruses* if they look closely.

The paper is “really important because there's very little understanding of where rubella came from,” says molecular anthropologist Anne Stone of Arizona State University, Tempe. “It was all by itself without any close relative.” The finding underscores the importance of the One Health approach, which recognizes that the health of people is closely connected to that of animals and the environment, she says.

Both viruses bear close watching, researchers say. It's “really interesting” that rustrela was able to infect both placental and marsupial mammals, and “was actively jumping between species,” says evolutionary virologist Edward Holmes of the University of Sydney. That flexibility could spell trouble, says vaccinologist Gregory Poland of the Mayo Clinic. “Who knows, if it could move from mice to other mammals, could it move to humans?” he asks. “In the end, the bugs win.”

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

## **Stomach Acid & Heartburn Drugs Linked with COVID-19 Outcomes**

*While sick with COVID-19, President Trump is taking an antacid. Doctors have been exploring whether these medicines can treat SARS-CoV-2 infections, and the results are mixed.*

[Ashley Yeager](#)

The uncertainty of the COVID-19 pandemic has made our stomachs churn, and now, evidence suggests that intense heartburn may be linked with worse symptoms of the disease. Some drugs that neutralize stomach acid, such as famotidine, which President Donald Trump is [taking](#), are associated with reduced severity, but others, such as Prilosec, correlate with higher infection rates and risk of death, at least in patients hospitalized with SARS-CoV-2 infections.

“Everyone has some level of acid reflux,” says Helder Nakaya, a systems biologist at the University of São Paulo in Brazil who has been studying the link between stomach acid and SARS-CoV-2 infection. “But I want to be clear . . . we cannot claim that this increased risk of death would apply to everyone with reflux.” The link does suggest that stomach acid might be a factor that’s often overlooked when it comes to COVID-19, Nakaya says.

In a retrospective analysis posted on [medRxiv](#) of roughly 1,300 hospitalized COVID-19 patients, Nakaya and colleagues found that individuals taking proton-pump inhibitors, including Prilosec, had a two- to three-fold higher risk of death compared with hospitalized patients not taking those drugs. This observation falls in line with a study published in late August in the [American Journal of Gastroenterology](#) by doctors in the US that also found people taking a proton-pump inhibitor twice a day for acid reflux had higher odds of testing positive for SARS-CoV-2 compared with individuals taking that type of drug once a day or individuals who took a histamine-2 receptor blocker such as Pepcid AC.

Proton-pump inhibitors “may undermine the gastric barrier to SARS-CoV-2 entry and reduce microbial diversity in the gut,” increasing patients’ risk getting COVID-19, the US-based team writes in its report.

Another retrospective analysis published online in [Gastroenterology](#) in May found that in a cohort of 1,620

hospitalized patients, proton-pump inhibitors had no relationship to the patients’ outcomes. Meanwhile, famotidine, which blocks the histamine-2 receptor on cells, correlated with a reduced risk of patients being intubated or dying from COVID-19.

Prior to analyzing that larger dataset, the authors had heard anecdotal evidence that famotidine might make COVID-19 symptoms less severe, and they’d heard that a few in vitro experiments also backed up the idea, study coauthor Joseph Conigliaro, the division chief of general internal medicine in the Department of Medicine at Northwell Health in Manhasset, New York, tells *The Scientist* in an email. In April, Northwell partnered with Alchem Laboratories and launched a randomized [clinical trial](#) to test whether giving up to 360 milligrams of famotidine intravenously to COVID-19 patients would improve their health outcomes compared with patients who received standard of care treatment. The trial later came under fire with a government whistleblower complaint for being hastily approved with little evidence and for the high doses of the drug being used, according to the [Associated Press](#). Still, Northwell’s research on famotidine continued.

“Neither the whistleblower complaint or the AP story had much of an effect on us. However, they are part of an overall trend to politicize Covid-19 clinical trials and prospective therapies that is deeply unfortunate,” Matthew Libassi, a spokesperson for the Feinstein Institutes for Medical Research, the research arm of Northwell, writes in an email to *The Scientist*. “That trend concerns us as it makes it harder to conduct medical science research.”

Despite the pushback, additional evidence for famotidine’s role in fighting viruses has surfaced. A [past study](#) published in 1996 had shown that famotidine could reduce viral replication of HIV, and a more recent [computational analysis](#) published in May also suggested that the histamine blocker could have some antiviral

properties—specifically, that it might inhibit a protease that processes proteins vital for SARS-CoV-2 replication.

The computational results offered “some biological plausibility” to the hypothesis that famotidine could reduce the severity of COVID-19, but “in the end I think the computational models do not tell the whole picture,” Conigliaro says. That’s why he and his colleagues conducted their retrospective study on patients admitted to the hospital between February 25 and April 13 (these patients were not part of Northwell’s clinical trial testing famotidine). Analysis of the data revealed a distinct difference in the overall health outcomes of the patients taking famotidine compared with patients not given the drug. (Why the patients were given famotidine is not clear, though some had a history of gastric reflux and were taking the medication prior to being admitted to the hospital.)

“I expected a difference, but not so pronounced,” Conigliaro says. “I was also surprised that doses as low as what was used in the retrospective study would work that well. We had anticipated that bigger doses would be needed.” In the study, some patients received up to **20 milligrams of famotidine per day for five days intravenously**, while others received it orally; when used for acid reflux, patients can take as much as 160 milligrams four times a day. Those who got the drug in the hospital fared better than those who did not, the study found.

Julian Abrams, a gastroenterologist at Columbia University Irving Medical Center-New York Presbyterian Hospital and a collaborator of Conigliaro, notes that the link between famotidine and reduced severity of COVID-19 is still only correlational at this point, but nevertheless warrants more attention.

### **Stomach acid and ACE2**

Nakaya’s work offers a bit more insight into potential mechanisms. He and his team weren’t initially planning to look at the relationship between antacids and COVID-19 at all. Their project

began as an offshoot of another one that Nakaya’s graduate student, Leandro Jimenez, had started. Jimenez had been analyzing the transcriptome data of patients with Barrett’s esophagus, a condition in which acid reflux causes damage to the lining of the esophagus.

The [data](#) from biopsies and a gene expression repository revealed increased expression of the gene *ACE2*, which encodes a cell surface protein that SARS-CoV-2 uses to enter human cells, in individuals with Barrett’s esophagus compared with individuals who did not have the condition. That increased gene activity was tied to regulation of intracellular pH pathways, specifically, their enrichment. That connection suggests increased *ACE2* expression is linked with lower pH inside cells, possibly a result of exposure to stomach acid.

“And that raised a flag,” Nakaya says, which led his team to hypothesize that Barrett’s esophagus, as well as gastroesophageal reflux disease (GERD), may be a comorbidity of COVID-19 that hadn’t been identified before.

To test the link between stomach acid and SARS-CoV-2 infectivity, Nakaya’s team exposed human monocytes in cell culture to different pH conditions and then to SARS-CoV-2. The researchers used these immune cells because they are known to be susceptible to infection by the coronavirus, Nakaya says. Under normal oxygen levels, the cells in culture with a pH lower than 7.4 had a higher expression of *ACE2* and also a higher viral load. The result indicates that intracellular pH may influence the ability of SARS-CoV-2 to infect cells and replicate within them.

But it wasn’t clear, says Nakaya, that the finding would have any clinical relevance, so the team dug into the medical records of patients in Manaus and São Paulo, Brazil, who had been hospitalized for COVID-19 and found that proton-pump inhibitors correlated with an increased risk of death. That association, Nakaya says, suggests that it might not be the drugs themselves that lead to

worsening COVID-19 symptoms, but instead that the proton-pump inhibitors are, the team writes, “important markers of hidden comorbidities that involve the damage caused by the excess stomach acid in GI tissues.” In other words, the low pH that cells are dealing with—and that patients are trying to treat with a proton-pump inhibitor—might make the cells more vulnerable to SARS-CoV-2 infection.

The results, however, don’t explain why famotidine correlated with better outcomes in Conigliaro and Abrams’s study, supposing the drug is also used to suppress stomach acid. “We don’t think that stomach acid is the explanation for the findings,” says Abrams. “We really don’t know why we found what we did.”

The past work on HIV and the computational analysis suggest that famotidine works as an antiviral, and a [study](#) published today (October 7) also supports that idea. The research shows that ranitidine bismuth citrate, another histamine antagonist and antibiotic combo used to treat stomach acid, suppresses SARS-CoV-2 replication in infected golden Syrian hamsters and improves their virus-related pneumonia symptoms. But famotidine, Conigliaro says, may have benefits other than being antiviral; it may actually prevent patients’ immune systems from overreacting to a SARS-CoV-2 infection and causing a life-threatening [cytokine storm](#). There’s some [evidence](#) for this from an observational study in which patients severely ill with COVID-19 were given a cocktail of histamine blockers, one of which was famotidine, and had better outcomes than did patients receiving the standard of care whose cases were reported elsewhere. Those data and other research “seem to suggest that the anti-histamine effect is what prevents patients from getting the cytokine storm,” Conigliaro explains, noting that cells with the histamine-2 receptor are in the lungs as well as the stomach.

The results from those studies and his team’s work, he says, bolster

the case for the clinical trial launched last spring to test famotidine as a treatment for COVID-19, results of which are still pending. The Department of Defense under the Discovery of Medical Countermeasures Against Novel Entities, or DOMANE, program is also studying famotidine as a COVID-19 treatment, according to [Vanity Fair](#), and another [famotidine trial](#) in Bangladesh is also now recruiting patients.

“Most people’s attention is shifting towards vaccines rather than treatments,” Abrams says, but “with President Trump having COVID, that brings to light again the issue of treatments, especially since he was getting famotidine.”

<https://go.nature.com/33Mlxo8>

### **Can’t smell stinky fish? It might be in your genes**

*When exposed to the reek of fish, people with a particular mutation tend to misidentify the odour — or not detect it at all.*

The pungent aroma of fish prompts many people to hold their noses. But for individuals with a particular genetic variant, even rotten fish can smell like caramel.

Rosa Gisladdottir and Kari Stefansson at deCODE Genetics in Reykjavik and their colleagues scoured the DNA of more than 11,000 people in Iceland for genetic variants that influence odour perception. The researchers asked study participants to sniff a range of scents, including those of liquorice, cinnamon and fish. After inhaling an odour, the participants named the smell and rated its intensity and pleasantness.

The researchers found that a mutation in a gene that encodes a specific odour receptor in the nasal cavity affects a person’s perception of a compound in spoiled and fermented fish. Compared with the general population, people with the mutation found the fishy odour less intense and less unpleasant, and they tended to identify the smell as an item unrelated to seafood, such as potatoes, caramel or rose. Some people with the mutation could not detect the



fish odour at all.

People with other genetic variants tended to find liquorice or cinnamon odours more pleasant than individuals without the variants. [Curr. Biol. \(2020\)](#)

<https://bit.ly/3jNKt49>

## **A NASA mission is about to capture carbon-rich dust from a former water world**

*If successful, OSIRIS-REx could carry up to 1 kilogram of carbon-rich material from the dawn of the Solar System*

By [Paul Voosen](#)

OSIRIS-REx is ready to get the goods. On 20 October, [after several years of patient study](#) of its enigmatic target, NASA's \$800 million spacecraft will finally stretch out its robotic arm, swoop to the surface of the near-Earth asteroid Bennu, and sweep up some dust and pebbles. The encounter, 334 million kilometers from Earth, will last about 10 seconds. If it is successful, OSIRIS-REx could steal away with up to 1 kilogram of carbon-rich material from the dawn of the Solar System for return to Earth in 2023.

Since OSIRIS-REx (short for Origins, Spectral Interpretation, Resource Identification, Security, Regolith Explorer) arrived in 2018, Bennu has yielded surprises, not all of them welcome. The 500-meter-wide asteroid was not smooth, as expected, but studded with more than 200 large boulders that could upset the sampling maneuver. And every so often, the asteroid [ejected coin-size pebbles](#), probably propelled by meteoroid impacts or solar heating. The boulder hazard, in particular, forced the team to target an area just 16 meters across for sampling, 10 times smaller than planned. "Bennu has not made things easy for us," says Mike Moreau, the mission's deputy project manager at NASA's Goddard Space Flight Center.

Despite the logistical challenge, the boulders contain a prize: [veins of carbonate minerals thicker than your hands](#), the team reports in

one of six studies published today in *Science* and *Science Advances*. The minerals, which precipitate out of hot water, popped out of data gathered during a close flyby of [light-colored boulders](#) near the target site, called Nightingale. Researchers believe the veins grew in channels of fluid circulating within Bennu's parent body, a larger planetesimal [thought to have formed beyond Jupiter's orbit](#) soon after the dawn of the Solar System 4.56 billion years ago, before being smashed apart in the asteroid belt within the last billion years. Heat from the decay of radioactive elements in its interior presumably drove the churning, and the presence of so much carbonate "suggests large-scale fluid flow, possibly over the entire parent body," says Hannah Kaplan, a planetary scientist at Goddard who led the work.

This ancient water world is consistent with the idea that objects like Bennu delivered much of Earth's water when they struck the planet billions of years ago, says Dante Laretta, the mission's principal investigator and a planetary scientist at the University of Arizona. The veins also suggest watery bodies like Bennu were a cauldron for the organic chemistry that generated the amino acids and [other unusual prebiotic compounds](#) found in carbon-rich meteorites.

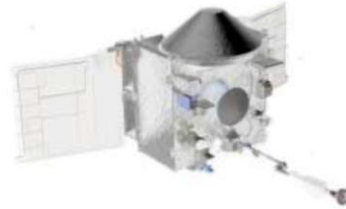
OSIRIS-REx won't be sampling the carbonate veins directly: The chamber at the end of its robotic arm is designed to suck up grit smaller than a penny. That's all right, however, because the small pebbles strewn across Nightingale also contain [signs of carbonates and other organic molecules](#), the team reports today. "This gives me a hint that my dream is going to come true," Laretta says. "I want to bring back something we've never seen before."

The team [picked Nightingale](#) for its abundant pebbles and because the site appears young, probably because an impact exposed it in recent geological time, leaving it largely unaltered by bombarding cosmic rays. But navigating the van-size spacecraft to a safe touchdown still won't be easy; the site is ringed with building-size

rocks, including one nicknamed Mount Doom, along with smaller boulders throughout. Observations suggest many of these boulders are porous, almost fluffy, and would crumble if touched. But the team doesn't want to take that chance: Using its cameras to navigate, the spacecraft will automatically abort its approach at an altitude of 5 meters if the site appears hazardous.

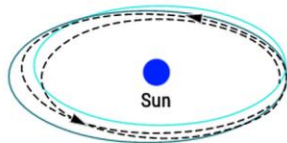
### To an asteroid and back

On 20 October, OSIRIS-REx, a NASA asteroid sample return mission, will attempt to gather up to 1 kilogram of dust and pebbles for eventual return to Earth. Its carbon-rich target, Benu, could hold organic molecules from the Solar System's earliest days.



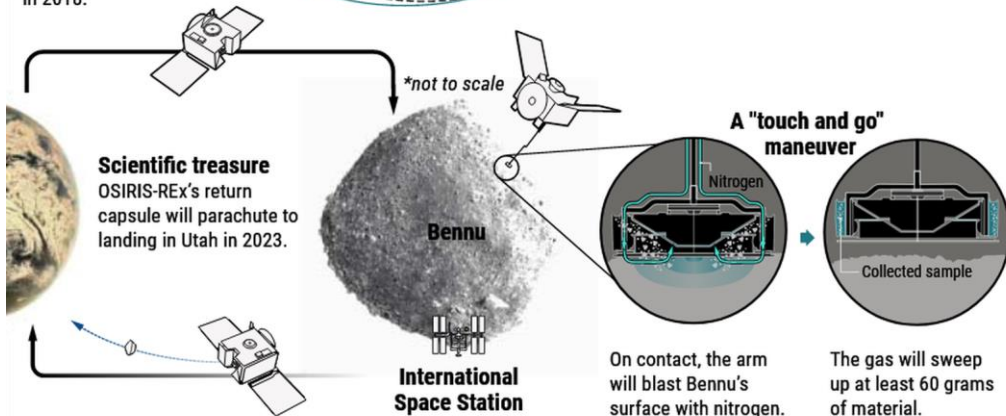
#### Local target

Benu's orbit brings it nearly as close to Earth as the Moon. OSIRIS-REx reached the asteroid in 2018.



— Earth  
— Benu  
--- Approach

The spacecraft will extend a sampling arm as it approaches its target area.



(Graphic) G. Grullón/Science;

(Images, Left To Right) Nasa; Nasa/Goddard/University Of Arizona (2)

The entire sampling attempt, lasting 4.5 hours, needs to be autonomous; Benu is currently five times farther from Earth than Mars, and radio signals take 18 minutes to reach it. After thruster maneuvers bring it to the touch point with Benu, blasts of nitrogen should push dust and pebbles into the doughnut-shaped collector at

the end of the robotic arm. It will be several days before NASA can judge how much was gathered, based on images of the target site and sampling head, and changes in how the spacecraft spins. By the end of the month, managers will decide whether to make a second attempt at a backup site in January 2021. Either way, the spacecraft will leave Benu next year and head back to Earth. It will arrive in September 2023 and eject the sample capsule, which will parachute to a landing in the Utah desert.

<https://wb.md/3jMXsmD>

## Convalescent Blood Plasma Safe, Effective for COVID-19

*Convalescent blood plasma is a useful and relatively safe therapy for COVID-19, and perhaps should be used more widely*

Priscilla Lynch

Convalescent blood plasma is a useful and relatively safe therapy for COVID-19, and perhaps should be used more widely, according to Prof Arturo Casadevall, Chair of Molecular Microbiology and Immunology at the Johns Hopkins Bloomberg School of Public Health.

Speaking during the 2020 ESCMID Conference on Coronavirus Disease (ECCVID), he said blood plasma is currently the only therapy against COVID-19 associated with a major reduction in mortality if given before ICU admission.

He quoted clinical trial data showing that convalescent blood plasma functions as an antiviral by reducing inflammation; a key issue in patients hospitalised with severe COVID-19.

Antibody levels are crucial to successful blood plasma treatment as they can vary per plasma unit so have results to date. When examining trial data, Prof Casadevall said that a number of antibody variables have to be considered — does the plasma have the specific antibodies to the SARS-CoV-2 antigen; are there sufficient antibody levels in the plasma; and was it given early enough to

have an impact?

Prof Casadeval is involved in two major randomised, placebo-controlled clinical trials on convalescent blood plasma for COVID-19 treatment and prophylaxis, which he said should provide more definitive answers.

He noted that blood plasma has not gained the attention that some novel pharmaceutical COVID-19 treatments have. However many of these new treatments have limited efficacy and are very expensive, while convalescent blood plasma is cheap, low tech and easily deployable, and "it is remarkably well tolerated", even in older sicker patients. The current challenges are figuring out if, when and how to use it effectively.

#### **Expert commentary:**

**Question:** Which COVID-19 patients should receive convalescent blood plasma?

**Casadeval:** "It depends on the patient and plasma availability, but if you have high-quality plasma, and people in hospital, all the currently available evidence is that it is reasonably safe with good outcomes."

*References: Casadeval A. Keynote Presentation: Use of convalescent plasma for COVID-19 treatment. ECCVID 2020. 24 September 2020.*

<https://wb.md/3jPyxPf>

**When the Only Clinical Choices Are 'Lose-Lose'  
Among the many tolls inflicted on healthcare workers by COVID-19 is one that is not as easily measured as rates of death or disease, but is no less tangible: moral injury.**

**Peter Yellowlees, MBBS, MD**

This is the term by which we describe the psychological, social, and spiritual impact of high-stakes situations that lead to the betrayal or transgression of our own deeply held moral beliefs and values.

The current pandemic has provided innumerable such situations that can increase the risk for moral injury, whether we deal directly

with patients infected by the coronavirus or not. Telling family members they cannot visit critically ill loved ones. Delaying code activities, even momentarily, to get fully protected with personal protective equipment. Seeing patients who have delayed their necessary or preventive care. Using video rather than touch to reassure people.

Knowing that we are following guidelines from the US Centers for Disease Control and Prevention (CDC) does not stop our feelings of guilt. The longer this pandemic goes on, the more likely it is that these situations will begin to take a toll on us.

For most of us, being exposed to moral injuries is new; they have [historically been most associated](#) with severe traumatic wartime experiences. Soldiers, philosophers, and writers have described the ethical dilemmas inherent in war for as long as recorded history. But the use of this term is a more recent development, which [the Moral Injury Project](#) at Syracuse University describes as probably originating in the Vietnam War era writings of veteran and peace activist [Camillo "Mac" Bica](#) and psychiatrist [Jonathan Shay](#). Examples of wartime events that have been thought to lead to moral injury include:

- *Causing the harm or death of civilians, knowingly but without alternatives, or accidentally;*
- *Failing to provide medical aid to an injured civilian or service member; and*
- *Following orders that were illegal, immoral, and/or against the rules of engagement or the Geneva Conventions.*

However, the occurrence of moral injuries in modern healthcare is increasingly being reported, primarily as [an adverse effect of healthcare inefficiencies](#) that can contribute to burnout. COVID-19 has now provided [an array of additional stressors](#) that can cause moral injuries among healthcare workers. A [recent guidance document on moral injury](#) published by the American Psychiatric

Association noted that in the context of a public health disaster, such as COVID-19, it is sometimes necessary to transition from ordinary standards of care to those more appropriate in a crisis, as in wartime. This forces us all to confront challenging questions for which there may be no clear answers, and to make "lose-lose" choices in which no one involved — patients, family, or clinicians — ends up feeling satisfied or even comfortable.

Moral injuries affect most of us as physicians, as well as our colleagues and families, during this unusual, painful, traumatic, and potentially lethal time. Our lives have been altered significantly, and for many, completely turned upside down by enormous sacrifices and tragic losses. Globally, physicians account [for over half of healthcare worker deaths](#). In the United States alone, over [900 healthcare workers](#) have died of COVID-19.

Most of us have felt the symptoms of moral injury: frustration, anger, disgust, guilt. [A recent report](#) describes three levels of stressors in healthcare occurring during the pandemic, which are *not dissimilar to those wartime events described previously*:

**1. Severe moral stressors, such as the denial of treatment to a COVID-19 patient owing to lack of resources, the inability to provide optimal care to non-COVID-19 patients for many reasons, and concern about passing COVID to loved ones.**

**2. Moderate moral stressors, such as preventing visitors, especially to dying patients, triaging patients for healthcare services with inadequate information, and trying to solve the tension between the need for self-preservation and the need to treat**

**3. Lower-level but common moral challenges, especially in the community — for example, seeing others not protecting the community by hoarding food, gathering for large parties, and not social distancing or wearing masks. Such stressors lead to frustration and contempt, especially from healthcare workers making personal sacrifices and who may be at risk for infection caused by these behaviors.**

Every one of us is affected by these stressors. I certainly am.

What are the outcomes? We know that moral injuries are a risk factor for the development of mental health problems and burnout, and not surprisingly we are seeing that mental health problems, suicidality, and substance use disorders have increased markedly during COVID-19, [as recently detailed by the CDC](#).

Common emotions that occur in response to moral injuries are:

- *Feelings of guilt, shame, anger, sadness, anxiety, and disgust;*
- *Intrapersonal outcomes, including lowered self-esteem, high self-criticism, and beliefs about being bad, damaged, unworthy, failing, or weak;*
- *Interpersonal outcomes, including loss of faith in people, avoidance of intimacy, and lack of trust in authority figures; and*
- *Existential and spiritual outcomes, including loss of faith in previous religious beliefs and no longer believing in a just world.*

Moral injuries tend to originate primarily from systems-based problems, as we have seen with the lack of concerted national approaches to the pandemic. On the positive side, solutions typically also involve systems-based changes, which in this case may mean changes in leadership styles nationally and locally, as well as changes in the culture of medicine and the way healthcare is practiced and managed in the modern era. We are starting to see some of those changes with the increased use of telemedicine and health technologies, as well as more of a focus on [the well-being of healthcare workers](#), now deemed "essential."

As individuals, we are not helpless. There are things we can do in our workplaces to create change. I suggest:

- 1. Acknowledge that you, like me, are affected by these stressors. This is not a secret, and you should not be ashamed of your feelings.**
- 2. Talk with your colleagues, loved ones, and friends about how you, and they, are affected. You are not alone. Encourage others to share their thoughts, stories, and feelings.**
- 3. Put this topic on your meeting and departmental agendas and discuss these moral issues openly with your colleagues. Allow**

*sufficient time to engage in open dialogue.*

4. *Work out ways of assisting those who are in high-risk situations, especially for moderate to severe injuries. Be supportive towards those affected.*

5. *Modify policies and change rosters and rotate staff between high- and low-stress roles. Protect and support at risk colleagues.*

6. *Think about difficult ethical decisions in advance so they can be made by groups, not individuals, and certainly not "on the fly."*

7. *Keep everyone in your workplace constantly informed, especially of impending staff or equipment shortages.*

8. *Maintain your inherent self-care and resilience with rest, good nutrition, sleep, exercise, love, caring, socialization, and work-life balance.*

9. *Be prepared to access the many professional support services available in our community if you are intensely distressed or if the above suggestions are not enough.*

Remember, we are in this together and will find strength in each other. This too will pass.

<https://nyti.ms/2GNiUcx>

## **What's Special About Bat Viruses? What We Don't Know Could Hurt Us**

*The immune systems of bats are weird, but we don't know how weird, how they got that way or enough about other animals.*

By [James Gorman](#)

Bats were once of interest mainly to specialists and devoted conservationists. But the global pandemic pushed the animals squarely into the spotlight as the apparent original source of the novel coronavirus. Now, once arcane research into the large number of viruses that live in bats has acquired a new urgency, along with discussions of what to do about the likelihood of diseases in animals spilling over to humans.

In the journal *Science* on Thursday, two bat researchers urged fellow scientists to examine more closely what we know for certain

about bats and viruses, and suggested how we can find out more and how that knowledge might help us.

Daniel G. Streicker, a vampire bat researcher at the University of Glasgow and Amy T. Gilbert, a disease ecologist at the National Wildlife Research Center in Fort Collins, Colo., point out a number of gaps in our knowledge, and the lack of hard numbers to prove some common perceptions.

Dr. Streicker said in an interview that we may have gotten ahead of ourselves in the focus of research. "I think we're often trying to explain why bats are special before we actually work out how they're special," he said.

First and foremost, the researchers write is the "global health conundrum" of whether bat viruses are more likely to cause outbreaks than viruses harbored by other creatures.

The common perception that bats harbor more viruses than other animals does not hold up, they write, when one looks at the huge number of bat species.

Nor are bats immune to the effects of all viruses. There is no question, they write, that many bats can live with viruses that can prove lethal in humans and other animals, such SARS and MERS.

The "key question," Dr. Streicker said, is whether bat tolerance of viruses causes the evolution of pathogens that are more dangerous for people. Science does not yet have an answer.

"We seem to be lacking really strong, compelling evidence that the viruses of bats are more diverse or more prone to infect humans or more dangerous when they do infect humans than viruses of other animals," he said.

It isn't only the internal workings of bats that needs to be understood. How bad a spillover disease is and how it spreads depends on how people interact with bats, what kind of bats are involved, where they live and how they spread viruses among each other.

“We need interactions between immunologists, virologists, ecologists and evolutionary biologists.” That’s starting to happen, he says, partly because of the pandemic.

Bat scientists had been pushing for such cross-disciplinary work before the pandemic started. For example, the National Science Foundation last week awarded a grant of \$1.67 million to the American Museum of Natural History, Texas Tech University and Stony Brook University in order to establish the [Global Union of Bat Diversity Networks](#).

Tigga Kingston, an ecologist at Texas Tech, had been getting together at meetings on bat research for half a dozen years with her colleagues at the museum and at Stony Brook, and discussing the need for more connections. There were many networks of bat researchers, some regional, some devoted to a specific subject, but not a global network to foster communication between all bat researchers.

In 2019 she said, they decided to move from planning to action just as the National Science Foundation was reaching out to promote more of the kind of “meta-network” that they were thinking about. The fit was ideal.

Then, of course, the pandemic emerged, and an effort designed for basic research and conservation took on a new urgency. Suddenly, she said, “everything we’re doing has relevance to Covid-19,” from metabolism studies to evolution to conservation questions.

“We need immunologists working next to genomicists, who are working with ecologists, who are working with people who study the physiology of the animal,” she said. Until that happens, she added, “we really don’t stand a hope of mitigating these kinds of events.”

In the Science article, Dr. Streicker and Dr. Gilbert also point to specific areas of research in which bats could serve as testing populations for new techniques in disease control, like vaccines for

animal populations.

Rabies in animals like foxes has been successfully fought with vaccines in bait that foxes eat. That wouldn’t work for bats, but, Dr. Streicker said, a vaccine could be applied to bat fur and spread by contact.

In the future, [genetic engineering techniques like Crispr](#), might even be used to try to genetically engineer bats to be resistant to some viruses, he said, something that has been tested with mosquitoes, and discussed for use with mice and Lyme disease. “I think that’s very far into the future,” Dr. Streicker said, “and there are all sorts of ethical issues.”

But there are other ways to make what is essentially a contagious vaccine, perhaps by attaching the proteins that would promote an immune response to a virus that is infectious in bats, but not harmful. To them, or us.

<https://nyti.ms/30RY5nD>

## **In a First, New England Journal of Medicine Joins Never-Trumpers**

*Editors at the world’s leading medical journal said the Trump administration “took a crisis and turned it into a tragedy.”*

By [Gina Kolata](#)

Throughout its 208-year history, The New England Journal of Medicine has remained staunchly nonpartisan. The world’s most prestigious medical journal has never supported or condemned a political candidate.

Until now.

In an editorial signed by 34 editors who are United States citizens (one editor is not) and published on Wednesday, the journal said the Trump administration [had responded so poorly to the coronavirus pandemic](#) that they “have taken a crisis and turned it into a tragedy.”

The journal did not explicitly endorse Joseph R. Biden Jr., the

Democratic nominee, but that was the only possible inference, other scientists noted.

The editor in chief, Dr. Eric Rubin, said the scathing editorial was one of only four in the journal's history that were signed by all of the editors. The N.E.J.M.'s editors join those of another influential publication, Scientific American, who last month endorsed Mr. Biden, the former vice president.

The political leadership has failed Americans in many ways that contrast vividly with responses from leaders in other countries, the N.E.J.M. said.

In the United States, the journal said, there was too little testing for the virus, especially early on. There was too little protective equipment, and a lack of national leadership on important measures like mask wearing, social distancing, quarantine and isolation.

There were attempts to politicize and undermine the Food and Drug Administration, the National Institutes of Health and the Centers for Disease Control and Prevention, the journal noted.

As a result, the United States has had tens of thousands of "excess" deaths — those caused both directly and indirectly by the pandemic — as well as immense economic pain and an increase in social inequality as the virus hit disadvantaged communities hardest.

The editorial castigated the Trump administration's rejection of science, writing, "Instead of relying on expertise, the administration has turned to uninformed 'opinion leaders' and charlatans who obscure the truth and facilitate the promulgation of outright lies."

The uncharacteristically pungent editorial called for change: "When it comes to the response to the largest public health crisis of our time, our current political leaders have demonstrated that they are dangerously incompetent. We should not abet them and enable the deaths of thousands more Americans by allowing them to keep their jobs."

Scientific American, too, had never before [endorsed a political](#)

[candidate](#). "The pandemic would strain any nation and system, but Trump's rejection of evidence and public health measures have been catastrophic," the journal's editors said.

The N.E.J.M., like all medical journals these days, is deluged with papers on the coronavirus and the illness it causes, Covid-19. Editors have struggled to reconcile efforts to insist on quality with a constant barrage of misinformation and misleading statements from the administration, said Dr. Clifford Rosen, associate editor of the journal and an endocrinologist at Tufts University in Medford, Mass.

"Our mission is to promote the best science and also to educate," Dr. Rosen said. "We were seeing anti-science and poor leadership." Mounting public health failures and misinformation had eventually taken a toll, said Dr. Rubin, the editor in chief of The New England Journal of Medicine. "It should be clear that we are not a political organization," he said. "But pretty much every week in our editorial meeting there would be some new outrage."

"How can you not speak out at a time like this?" he added. Dr. Thomas H. Lee, a professor of medicine at Harvard Medical School and a member of the journal's editorial board, did not participate in writing or voting on the editorial. But "to say nothing definitive at this point in history would be a cause for shame," he said.

Medical specialists not associated with the N.E.J.M. applauded the decision.

"Wow," said Dr. Matthew K. Wynia, an infectious disease specialist and director of the Center for Bioethics and Humanities at the University of Colorado. He noted that the editorial did not explicitly mention Mr. Biden, but said it was clearly "an obvious call to replace the president."

There is a risk that such a departure could taint the N.E.J.M.'s reputation for impartiality. While other medical journals, including

JAMA, the Lancet and The British Medical Journal, have taken political positions, the N.E.J.M. has dealt with political issues in a measured way, as it did in a [forum published in October 2000](#) in which Al Gore and George W. Bush answered questions on health care.

But it is hard to imagine such a deliberative debate in today's acrimonious atmosphere, said Dr. Jeremy Greene, a professor of medicine and historian of medicine at Johns Hopkins University.

The Trump administration, he said, had demonstrated "a continuous, reckless disregard of truth."

"If we want a forum based on matters of fact, it strikes me that no form of engagement could work," Dr. Greene added.

<https://bit.ly/2FoYPZS>

## More Humans Are Growing an Extra Artery in Our Arms, Showing We're Still Evolving

*Subtle shifts in our anatomy today demonstrate how unpredictable evolution can be*

[Mike Mcrae](#)

Picturing how our species might appear in the far future often invites wild speculation over stand-out features [such as height](#), [brain size](#), and [skin complexion](#). Yet subtle shifts in our anatomy today demonstrate how unpredictable evolution can be.

Take something as mundane as an [extra blood vessel](#) in our arms, which going by current trends could be common place within just a few generations.

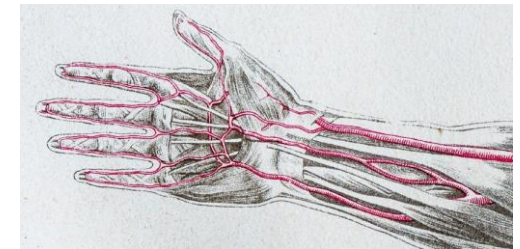
Researchers from Flinders University and the University of Adelaide in Australia have noticed an artery that temporarily runs down the centre of our forearms while we're still in the womb isn't vanishing as often as it used to. That means there are more adults than ever running around with what amounts to be an extra channel of vascular tissue flowing under their wrist.

"Since the 18th century, anatomists have been studying the

prevalence of this artery in adults and our study shows it's clearly increasing," [says](#) Flinders University anatomist Teghan Lucas.

"The prevalence was around 10 percent in people born in the mid-1880s compared to 30 percent in those born in the late 20th century, so that's a significant increase in a fairly short period of time, when it comes to evolution."

The [median artery](#) forms fairly early in development in all humans, transporting blood down the centre of our arms to feed our growing hands.



*Three major arteries in the forearm - median in the centre (ilbusca/Digital Vision Vectors/Getty Images)*

At around 8 weeks, it usually regresses, leaving the task to two other vessels – the radial (which we can feel when we take a person's pulse) and the ulnar arteries.

Anatomists have known for some time that this withering away of the median artery isn't a guarantee. In some cases, it hangs around for another month or so.

Sometimes we're born with it still pumping away, feeding either just the forearm, or in some cases the hand as well.

To compare the prevalence of this persistent blood channel, Lucas and colleagues Maciej Henneberg and Jaliya Kumaratilake from the University of Adelaide examined 80 limbs from cadavers, all donated by Australians of European descent.

The donors ranged from 51 to 101 on passing, which means they were nearly all born in the first half of the 20th century.

Noting down how often they found a chunky median artery capable of carrying a good supply of blood, they compared the figures with records dug out of a literature search, taking into account tallies that could over-represent the vessel's appearance.

The fact the artery seems to be three times as common in adults



today as it was more than a century ago is a startling find that suggests natural selection is favouring those who hold onto this extra bit of bloody supply.

"This increase could have resulted from mutations of genes involved in median artery development or health problems in mothers during pregnancy, or both actually," [says](#) Lucas.

We might imagine having a persistent median artery could give dextrous fingers or strong forearms a dependable boost of blood long after we're born. [Yet having one](#) also puts us at a greater risk of [carpal tunnel syndrome](#), an uncomfortable condition that makes us less able to use our hands.

Nailing down the kinds of factors that play a major role in the processes selecting for a persistent median artery will require a lot more sleuthing. Whatever they might be, it's likely we'll continue to see more of these vessels in coming years.

"If this trend continues, a majority of people will have median artery of the forearm by 2100," [says](#) Lucas.

This rapid rise of the median artery in adults isn't [unlike the reappearance](#) of a knee bone called the fabella, which is also three times more common today than it was a century ago.

As small as these differences are, tiny microevolutionary changes add up to large-scale variations that come to define a species.

Together they create new pressures themselves, putting us on new paths of health and disease that right now we might find hard to imagine today.

This research was published in the [Journal of Anatomy](#).

<https://bbc.in/30Q1Qty>

### **Covid: South Asian hospital patients 'at greater risk of dying'**

*Black and South Asian patients are more severely affected by Covid-19 than white patients at different stages of the disease, research suggests.*

In a study of 1,800 hospital patients, black people had a higher risk of being admitted to hospital with Covid-19 while South Asian people were more likely to die from it.

Biology was a more important factor than poverty or underlying health.

Different ethnic groups may need to be treated differently, researchers say.

It was already known that people from ethnic minority backgrounds are disproportionately affected by serious illness from Covid-19, but the exact reasons why were unclear.

#### **Varied effects**

This study, carried out at King's College Hospital in south-east London, suggests that the way the body functions is a more important factor than poverty or existing health problems.

Out of 1,827 adults admitted to the hospital between March and June with confirmed Covid-19, 872 were inner-city residents who gave their ethnicity.

Of these, 48% were black, 33% were white, 12% were mixed ethnicity and 5.6% were of Asian ethnicity - most from South Asia. They were compared with more than 3,000 people living nearby.

The analysis shows that black and mixed ethnicity patients were three times more likely to end up in hospital with Covid-19 compared with white people living in the same area.

However, their chances of survival were not very different from white patients in hospital.

By contrast, South Asian patients did not have a higher risk of being admitted to hospital with Covid-19 than white patients, but they were at higher risk of dying in hospital and needing intensive care.

The researchers found that ethnic minority patients were 10 to 15 years younger than white patients and had more underlying health conditions, particularly type 2 diabetes.

**Offering best treatments**

Prof Ajay Shah, consultant cardiologist at King's College Hospital, said the findings were "striking". "We may need different treatment strategies for different ethnic groups.

*Table. COVID-19 and Underlying Conditions*

"For black patients, the issue may be how to prevent mild infection progressing to severe, whereas for Asian patients it may be how to treat life-threatening complications."

Age in years	Proportion of confirmed or probable COVID-19 cases
20 – 29	23%
30 – 39	28%
40 – 49	36%
50 – 59	44%

Source: CDC Case Surveillance Task Force data.

Commenting on the study, England's chief medical officer, Chris Whitty, said the evidence was now clear that "people from black and minority ethnic groups are more severely affected by Covid-19".

He said the research provided "important information to help healthcare professionals offer the best possible treatment to minority ethnic patients".

Dr Sonya Babu-Narayan, associate medical director at the British Heart Foundation, said people from BAME backgrounds were more likely to have heart and circulation problems, such as high blood pressure and type 2 diabetes, which would put them more at risk.

They are also more likely to be from low-income families - but this study suggests poverty is not the major factor in severe Covid.

"Research is now needed to assess how other structural and behavioural factors may contribute, including occupation, access to health messaging and healthcare, and differences in the patient journey once people reach hospital," Dr Babu-Narayan said.

"As we see Covid-19 cases rise again in the UK, we must address these disparities with urgency."

<https://wb.md/2GXDBSZ>

**COVID Could Add 20K+ Americans a Day to 'Preexisting Conditions'**

*Daily 20,000 Americans between 20 and 60 years of age could be classified as living with a "preexisting condition" because of COVID-19*

**Damian McNamara**

Every day, another 20,000 Americans between 20 and 60 years of age could be classified as living with a "preexisting condition" because of COVID-19, researchers from the Commonwealth Fund report.

Overall, the pandemic could cause almost 3.5 million Americans to be added to this category, a fact that has important implications approximately 1 month before the US Supreme Court is expected to weigh in on the constitutionality of the Affordable Care Act (ACA), the investigators note.

"Polls show that most Americans are worried that preexisting conditions could lead to loss of insurance coverage. People are surprised and even more worried when they realize that the pandemic is adding to this problem," lead author Eric Schneider, MD, PhD, told *Medscape Medical News*.

The study was [posted online](#) in a blog post on October 8 by the Commonwealth Fund.

Schneider, senior vice president for policy and research at the Commonwealth Fund, and research associate Arnav Shah based their calculations on approximately 7.5 million cases of COVID-19 reported in the United States as of October 7, 2020. The figures include an estimated 45,000 new cases reported daily.

Using data from the Centers for Disease Control and Prevention and Johns Hopkins University, they found that 32% of 1,502,309 people with laboratory-confirmed or probable COVID-19 already had an underlying condition. This proportion varied by age.

If almost one third already have an underlying condition, this means that for the remaining 68%, insurers might consider COVID-19 their first preexisting condition.

### More Than 3 Million Could Be Affected

"We estimated that just over 3.4 million nonelderly adults had COVID-19 as a new pre-existing condition," the authors write.

"If the Supreme Court overturns the preexisting condition provision of the ACA, private insurers will be able to deny insurance coverage to adults under age 65 who are unable to get insurance through an employer or large group," Schneider said. "Testing positive for COVID-19 could be treated as a preexisting condition." Furthermore, he added, "Because the long-term health effects of COVID-19 are unknown, these adults could be uninsurable until they reach age 65 and qualify for Medicare."

There could also be economic consequences, Schneider said. "If COVID-19 causes long-term health problems the way [hepatitis C](#) or [HIV](#) do, these costs could be high for patients, providers, and government programs."

Removing preexisting condition protections could also act as a disincentive. "Healthy people might avoid COVID-19 testing altogether. And that is exactly the opposite of what must happen if we are ever to get this pandemic under control," the researchers note.

### "A Frightening Picture"

"These findings highlight the importance of preexisting protection," Nathalie Huguet, PhD, who was not affiliated with the study, told *Medscape Medical News*.

The results "provide a frightening picture of what the future could be if such protection would be dismissed, with millions of Americans of all ages unable to obtain or afford health insurance," said Huguet, research assistant professor of family medicine at Oregon Health and Sciences University School of Medicine in

Portland, Oregon, and lead author of a [study](#) that compared the prevalence of preexisting conditions among community health center patients before and after passage of the ACA.

Considering COVID-19 a preexisting condition might not affect all communities the same, she said. "Even more distressing is that COVID infection is more common in people suffering from health disparities, for example, race and ethnic minorities and low-income patients. As such, the burden of naming COVID as a preexisting condition will likely exacerbate health disparities."

Even if COVID-19 is not classified as a preexisting condition in the future, some of its long-term adverse health effects — such as neurologic complications or cardiovascular damage — could be, Huguet said.

*Schneider and Huguet report no relevant financial relationships.*

<https://bit.ly/30T0H13>

## Groundbreaking new coronavirus vaccine candidate also kills the flu

*The vaccine would be administered as a nasal spray rather than a traditional injection so it can generate a response at the typical entry site for these infectious pathogens.*

By [Chris Smith](#)

At least one coronavirus vaccine [might be approved for emergency use by the end](#) of the year in [America and the EU](#), at which point at-risk categories of people could be immunized. More ample vaccination campaigns will start worldwide in the first months of 2021, as more vaccines become available. That's assuming some of the experimental drugs that reach the final stage of testing prove to be safe and effective, as [not all drugs will be successful after Phase 3 trials](#) no matter how promising they seemed.

What's great about the current state of COVID-19 vaccine research is that there are hundreds of ideas in development, of which only about a dozen have reached Phase 3. The more vaccine approaches

available, the more likely we are to find one that works. And scientists have come up with what might be one of the most exciting COVID-19 vaccine concepts so far — not just because it's can be administered via a spray rather than a regular shot, but because it should offer protection from both COVID-19 and the flu, as it targets both at once.

Researchers from Hong Kong have developed an influenza and COVID-19 vaccine that will enter Phase 1 trials in Hong Kong in November. This means the drug won't seek regulatory approval for several months, and that's assuming it clears all the three stages of the trial with proven success. But the pandemic won't be over until late 2021 at the soonest. Some estimates say that the pandemic is here to stay until 2022. Even after that, the virus might circulate in communities, and scientists don't believe COVID-19 will be eradicated. Vaccines will be required to manage the illness, alongside other therapeutics and preventive measures.

Phase 1 of the trial will start in Hong Kong, enrolling some 100 adults, Yuen Kwok-Yung [told Bloomberg](#). Yuen is the chair of infectious disease in the University of Hong Kong's microbiology department, which is involved in the study.

The vaccine concept is similar to flu vaccines that are delivered via sprays. The idea behind these drugs is to start working where the respiratory viruses enter the body, the nose. But the scientists went one step further and created a dual vaccine that would offer coronavirus protection.

"Our idea is that we want both influenza and Covid-19 protection at the same time," Yuen said. The vaccine uses a weakened flu virus that only grows in the upper airway. The attenuated virus can't replicate and has been genetically engineered to include the coronavirus spike protein.

If successful, the vaccine would trigger an immune response against the coronavirus. The immune system should produce neutralizing

antibodies to that spike protein, blocking the real coronavirus. The immune system would also generate a response to the flu virus that was used in the vaccine. That said, other influenza viruses could still be infectious.

"We showed very well that it works in animals," Yuen said. "I do think that a mucosal vaccine is a great idea," he added, referring to spray vaccines that target the nasal mucus membrane.

The vaccine has received funding from the Coalition for Epidemic Preparedness Innovations (CEPI) and the Hong Kong government. CEPI will also analyze successful vaccines and compare all the research results to identify [the best approaches for preventing infection](#). The Hong Kong trial will have Phase 1 results a few months after its November start date. China started a different Phase 1 trial a few weeks ago of a vaccine candidate that comes in spray form. Separately, other researchers are studying COVID-19 drugs that can be administered via [sprays or nebulizers](#).

<https://bit.ly/3dp1zTC>

## **Poisonous Caterpillars That Look Like Bad Wigs Are Popping Up All Over Virginia**

*No matter how cute and fuzzy this critter looks, don't touch it.*

Aylin Woodward

This toupée-like insect is one of the most poisonous caterpillars in the US. Named the furry puss caterpillar - perhaps for its resemblance to less venomous house cats - people who brush up against its hairy coat have a painful reaction. And according to the Virginia Department of Forestry (VDof), there have been reports of the puss caterpillar in a few eastern Virginia counties.

"#SocialDistance away from this caterpillar!" the VDof [wrote on its Facebook](#) Tuesday.



(IrinaK/Shutterstock)

## The caterpillars can fall from trees and lodge in people's clothes

The insect's fuzzy veneer hides venomous spines. As the caterpillars grow in size, before they change into equally fuzzy southern flannel moths, their venom becomes more toxic.

Their painful sting is followed by swelling and redness, but those who get stung may also experience symptoms like headaches, [fever](#), nausea, vomiting, low blood pressure, rapid heartbeat, seizure, and in rare cases, abdominal pain, according to a [2005 paper](#) published in the *American Journal of Tropical Medicine and Hygiene*.

Some people may even experience multiple stings because the caterpillars can fall from trees and become lodged in clothes, "particularly shirt collars," the authors wrote. The Florida Poison Information Centre (FPIC) recommends [treating puss caterpillar stings](#) by placing scotch tape over the sting, then peeling it off to remove the spines.



*Southern flannel moth after the caterpillar stage.* ([Patrick Coin/Wikimedia Commons, CC BY-SA 2.5](#))

## Its sting can send people to the hospital

In 2018, a puss caterpillar dropped from a tree onto 5-year-old Adrie Chambers who was playing in the yard of her Texas Daycare. Chambers's arm went numb, and she was rushed to the hospital where she was treated with steroids and made a full recovery.

[A Florida teenager](#) also got stung in 2018 and was hospitalized shortly after. His mother published a Facebook post about her son's sting, which was [reportedly shared hundreds of thousands of times](#). And in May 2017, a mum recalled hearing a "blood-curdling scream" after her [5-year-old son stepped on one of the caterpillars](#).

Outbreaks of puss caterpillar stings even prompted public school closures in Texas in 1923 and 1951.

The [puss caterpillar is found on the East Coast](#) between Florida and

New Jersey, but its habitat extends as far west as Arkansas and Texas, according to a [guide published by the University of Florida's Entomology Department](#).

According to the VDOF, the caterpillars eat oak and elm leaves but can be found in parks or near structures.

Ren Oliver's family spotted one of the furry insects on a deck when eating dinner in Tappahannock in early September.

"My 5-year-old son saw it and said, 'Don't anyone touch it! It's probably poisonous!'" Oliver told Business Insider.

After reading about how venomous they are, Oliver's father picked up the caterpillar with paper towels and flung it in a nearby river.

"Thankfully we escaped it but it was the wildest looking thing and so appropriate for 2020. Just bizarre," Oliver said.

*Caroline Praderio contributed reporting to this story.*

<https://bit.ly/2SMBnc1>

## Japanese politician slammed as 'dinosaur' after claiming LGBT education would mean 'no children'

*Masateru Shiraishi, 78, warned that if local schools were required to teach students about same-sex families, the ward 'will have no residents'*

[Julian Rvall](#)

Politicians have been urged to attend a town hall meeting with LGBT residents to hear their grievances and proposals about ways to make the community more harmonious. Photo: Xinhua

A local politician representing a district in central Tokyo has refused to retract or apologise for claiming the ward would cease to exist if a local ordinance was passed requiring schools to teach students about [same-sex families](#).

Masateru Shiraishi, chairman of the Adachi Ward welfare committee, made his remarks at a meeting last month and they have since provoked widespread condemnation online, with posters criticising him as a "dinosaur" guilty of hate speech.

“If L [lesbians] and G [gays] spread to Adachi Ward completely we will have no residents because it means there will be no children,” he said during a debate about the proposed law.

Shiraishi, 78, later told the *Mainichi* newspaper: “If LGBT people get focused on in an excessive manner, then children will lose their sense of the need for having and raising more children in the future. Schools must teach the importance of normal marriage and having and raising children.”

Shiraishi, a member of the Liberal Democratic Party, was issued a warning by the assembly speaker and his own party but remained defiant. “The way that people receive my comments will be different,” he said. “I, for example, am offended by all the opinions that I hear from the Japanese Communist Party. If the parties involved are unhappy, then that is fine.”

The backlash on social media was swift, with Shiraishi’s comments condemned as [hate speech that should disqualify him from public office](#).

One Twitter user suggested local politicians should be obliged to attend a town hall meeting with LGBT residents to hear their grievances and proposals about ways to make the community more harmonious.

Another Twitter user wrote: “*Homophobic lawmakers should perish.*” Another called for Shiraishi to “[attend] re-education or he will only repeat the same thing”.

On the Japan Today website, one poster wrote: “*This dinosaur should just disappear. He is one of the reasons why [Japan](#) is stuck in the past.*”

Other messages noted that Japan has a [population crisis and a falling birth rate](#) that pose serious challenges, but posters insisted homosexuality was not the cause.

“It is because of the horrific memories Japanese people have about their childhoods in this society,” one poster wrote. “Once you are

past 12 years old, you have no more fun and you become another robot for the rest of your life. That, and the low salaries and long, unpaid overtime hours.”

Another poster wrote: “Perhaps looking for a way to improve the lives of your constituents rather than being a homophobe would be a good idea as a politician.”

Yuichi Kamiya, secretary general of the Japan Alliance for LGBT Legislation, also refuted the connection. “Anti-discrimination laws for LGBT people have been enacted in European Union member countries, but this has not lowered their birth rates,” he said.

“The LGBT population does not increase or decrease because of education,” he said, adding Shiraishi should either leave office or retract his remarks.

<https://bit.ly/2SL0WdC>

## **Protective antibodies persist for months in survivors of serious COVID-19 infections**

*The study also demonstrates that measuring antibodies can be an accurate tool for tracking the spread of the virus in the community.*

Boston - People who survive serious COVID-19 infections have long-lasting immune responses against the virus, according to a new study led by researchers at Massachusetts General Hospital (MGH). The study, published in [Science Immunology](#), offers hope that people infected with the virus will develop lasting protection against reinfection. The study also demonstrates that measuring antibodies can be an accurate tool for tracking the spread of the virus in the community.

The immune system produces proteins called antibodies in response to SARS-CoV-2, the virus that causes COVID-19. “But there is a big knowledge gap in terms of how long these antibody responses last,” says Richelle Charles, MD, an investigator in the Division of Infectious Diseases at MGH and a senior author of the paper. To

find out, she and her colleagues obtained blood samples from 343 patients with COVID-19, most of whom had severe cases. The blood samples were taken up to four months after a patient's symptoms emerged. The blood's plasma was isolated and applied to laboratory plates coated with the receptor-binding domain (RBD) of the virus's "spike" protein, which attaches to cells, leading to infection. The team studied how different types of antibodies in the plasma bound to RBD. The results were compared to blood samples obtained from more than 1,500 individuals prior to the pandemic.

The researchers found that measuring an antibody called immunoglobulin G (IgG) was highly accurate in identifying infected patients who had symptoms for at least 14 days. Since the standard PCR (nasal swab) test for SARS-CoV-2 loses sensitivity over time, augmenting it with a test for antibodies in patients who have had symptoms for at least eight days (at which time 50 percent are producing antibodies) will help identify some positive cases that might otherwise be missed, says Charles.

The researchers found that IgG levels remained elevated in these patients for four months, and were associated with the presence of protective neutralizing antibodies, which also demonstrated little decrease in activity over time. "That means that people are very likely protected for that period of time," says Charles. "We showed that key antibody responses to COVID-19 do persist."

In another finding, Charles and her colleagues showed that people infected with SARS-CoV-2 had immunoglobulin A (IgA) and immunoglobulin M (IgM) responses that were relatively short-lived, declining to low levels within about two and a half months or less, on average. "We can say now that if a patient has IgA and IgM responses, they were likely infected with the virus within the last two months," says Charles.

Knowing the duration of the immune response by IgA and IgM will help scientists obtain more accurate data about the spread of SARS-

CoV-2, explains Jason Harris, MD, a pediatric infectious disease specialist at MGH and co-senior author of the study. "There are a lot of infections in the community that we do not pick up through PCR testing during acute infection, and this is especially true in areas where access to testing is limited," he says. "Knowing how long antibody responses last is essential before we can use antibody testing to track the spread of COVID-19 and identify 'hot spots' of the disease."

*Richelle Charles, MD, is an assistant professor of Medicine at Harvard Medical School (HMS). Jason Harris, MD, is an associate professor of Pediatrics at HMS. Lead authors of the paper are Anita Iyer, PhD, a post-doctoral fellow at MGH; and Forrest K. Jones, a doctoral student in infectious disease epidemiology at the Johns Hopkins Bloomberg School of Public Health.*

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

## **A new species of Darwin wasp from Mexico named in observance of the 2020 quarantine period**

*The new species goes by the official scientific name **Stethantyx covida***

Scientists at the [Autonomous University of Tamaulipas \(UAT\)](#) in Mexico recently discovered five new species of parasitoid wasps in Mexico, but the name of one of them sounds a bit weird: covida. Why this name?

In fact, the reason is quite simple. The thing is that the team of Andrey Khalaim (also a researcher at the [Zoological Institute of Russian Academy of Sciences](#) in Saint Petersburg, Russia) and Enrique Ruíz Cancino discovered the new to science species during the 2020 global quarantine period, imposed due to the COVID-19 pandemic. Their findings are described in a newly published research [article](#), in the peer-reviewed, open-access scientific journal [ZooKeys](#).

"We thought that it was a good idea to remember this extraordinary year through the name of one remarkable species of Darwin wasp found in seven Mexican States (including Tamaulipas, where the

UAT campus is located) and also Guatemala," explain the scientists. The new species, which goes by the official scientific name *Stethantyx covida*, belongs to the Darwin wasp family Ichneumonidae, one of the most species-rich insect families, which comprises more than 25,000 species worldwide.

"Darwin wasps are abundant and well-known almost everywhere in the world because of their beauty, gracility, and because they are used in biological control of insect pests in orchards and forests.

Many Darwin wasp species attack the larvae or pupae of butterflies and moths. Yet, some species are particularly interesting, as their larvae feed on spider eggs and others, even more bizarre, develop on living spiders!" further explain the authors of the new study.



*Holotype specimen of the newly described species of parasitic Darwin wasp Stethantyx covida. Andrey I. Khalaim*

*Stethantyx covida* is a small wasp that measures merely 3.5 mm in length. It is predominantly dark in colour, whereas parts of its body and legs are yellow or brown. It is highly polished and shining, and the ovipositor of the female is very long and slender.

Along with *Stethantyx covida*, the authors also described four other Mexican species of Darwin wasps from three different genera (*Stethantyx*, *Meggoleus*, *Phradis*), all belonging to the subfamily Tersilochinae.

Some tersilochines are common on flowers in springtime. While the majority of them are parasitoids of larvae of various beetles, some Mexican species attack sawflies, inhabiting the forests.

**Original source:** Khalaim AI, Ruíz-Cancino E (2020) Contribution to the taxonomy of Mexican Tersilochinae (Hymenoptera, Ichneumonidae), with descriptions of five new species. *ZooKeys* 974: 1-21. <https://doi.org/10.3897/zookeys.974.54536>

<https://bit.ly/3dhm111>

## Neanderthal DNA May Be COVID Risk

*A stretch of Neanderthal DNA has been associated with some cases of severe COVID-19, but it's unclear how much a risk it poses.*

**Christopher Intagliata reports.**

The risk factors for COVID-19 are many: old age; obesity; heart conditions. But early genetic studies have identified another trait that some people who develop severe COVID-19 seem to share: a cluster of genetic variations on their third chromosome.

And that DNA sequence likely derives from Neanderthals, says Hugo Zeberg of the Max Planck Institute. "It's quite striking this variant has lingered on for 50,000 years."

Fifty-thousand years ago is the approximate time humans and Neanderthals interbred. And over the millennia, these Neanderthal variants have become more common in some *Homo sapiens* populations than others.

For example, 16 percent of people of European descent carry at least one copy of the Neanderthal stretch; half of South Asians do—and nearly TWO-thirds of Bangladeshis.

"And it's fascinating it is so high. Points to the fact that it must have been beneficial in the past. It's much higher than we expect. And then it's totally expunged in East Asia and China. So something has happened, driving the frequency up in certain places, and removing it totally in other places."

The details are in the journal *Nature*. [Zeberg, H. et al. [The major genetic risk factor for severe COVID-19 is inherited from Neanderthals](#)]

Zeberg and his colleague write that perhaps the Neanderthal DNA happens to boost the risk of developing severe COVID-19—and they point to the fact that in the U.K., people of Bangladeshi descent have twice the risk of dying of COVID-19 than the general



population.

But as epidemiologist Keith Neal of the University of Nottingham pointed out via email, people of African descent in the U.K. are also being hurt more by the virus—despite having hardly any Neanderthal genes.

Instead, it's social factors—like crowded, multigenerational households or working front line jobs—that are more likely to be driving the trends seen in the U.K. That's according to Andrew Hayward, director of the Institute of Epidemiology and Health Care at University College London.

And, as both epidemiologists pointed out—it's worth remembering that you can only develop *severe* COVID-19 if you're exposed to the virus in the first place.

<https://bit.ly/2IbqT3J>

### **Unique vine 'greenhouses' found by 91-year-old nature volunteer**

*Has a "unique" way of using its leaves to curl around its fruits to envelop them in a protective microclimate*

An unusual vine discovered by a 91-year-old volunteer nature guide in Japan has a "unique" way of using its leaves to curl around its fruits to envelop them in a protective microclimate, scientists said on Wednesday.

The cucurbitaceous vine, a type found in East Asia, is an oddity because while leaves come in all shapes and sizes and perform a crucial role in photosynthesis, they are rarely associated with reproduction.

But a new study published in the journal *Proceedings of the Royal Society B: Biological Sciences* found that the vine had specialised leaves able to enclose fruit and enhance [seed production](#) in colder conditions.

The research was co-authored by Nobuyuki Nagaoka, the 91-year-old guide at Yamagata Prefectural Natural Museum Park, who first

spotted the leaf behaviour in 2008 and has observed it every autumn since.

Intrigued by the strange leaf "greenhouses", he looked online for information about the vine, said co-author of the study Shoko Sakai, a professor at the Center for Ecological Research at Kyoto University.

"Our newsletter published in 1998 had an article about this plant. He saw the article and sent me a letter in 2008," Sakai told AFP.

He said initially when he saw a picture of the leaf enclosure he thought "it was a maldeveloped or pest-infected shoot".

But "when we read subsequent observation records he sent to us, it became clear that this was an interesting phenomenon worth further investigation," he said, adding that it was only when the researchers examined the real thing that they could confirm what it was.

"When I saw it, I was excited to find out that they were indeed leaves," Sakai said.

### **Cold-weather protection**

The researchers looked at [plants](#) at different altitudes at the foot of Mount Gassan, in the southern part of the Dewa Mountains, in an area partly within Yamagata park.

They describe the vine as a slender, annual plant that often inhabits the edges of deciduous forests with disturbances like roads, rivers or mountains. It can either be hermaphrodite or male and produces small, white flowers pollinated from August to September and later develop into fruits, each with a single seed.

The study, which also included experts from Japan's Forestry and Forest Products Research Institute, reported "for the first time, a unique function of leaves that enclose immature fruits in an annual vine".

They noted some leaves on hermaphrodite plants that were undeveloped in summer "expanded and overlapped with each other" to create a sort of cocoon around immature fruit.

The study found that these specialised "enclosure leaves" are produced towards the end of the growing season and produced a microclimate of up to 4.6 degrees Celsius warmer than was recorded around fruit where the leaves had been plucked off.

Removing the leaf enclosures negatively affected the survival and growth of the vine's [fruit](#), although they were unable to identify the mechanism, said the authors. They also found that the leaves grew thicker protective layers in colder areas and said the results suggest that the [vine](#) enclosures allow the plant to produce seeds under the cold weather the plant encounters at the end of its life.

### **Scientific first**

These enclosure leaves were found to have less photosynthetic ability and were different in greenness and structure from others.

Previous research has described some functions of leaves that aid reproduction, such as the plant *Saururus chinensis*, whose leaves can temporarily turn white to attract pollinators.

But the study said such traits were likely "in conflict with traits that promote photosynthesis, the primary function of leaves".

"Plants produce many leaves in their lifetime. Size, shape, and thickness among the leaves are often very diverse within an individual," said Sakai, adding that previously this had been viewed in terms of photosynthesis. "In this study, we found that some leaves play more important roles in reproduction rather than photosynthesis."

The research was Nagaoka's first scientific paper, Sakai said, adding that he was still guiding tours at the park and observing vines. "I think he should be proud of his paper, but he is very humble," he added.

*More information:* Nobuyuki Nagaoka et al. Green greenhouse: leaf enclosure for fruit development of an androdioecious vine, *Schizopepon bryoniifolius*, *Proceedings of the Royal Society B: Biological Sciences* (2020). [DOI: 10.1098/rspb.2020.1718](https://doi.org/10.1098/rspb.2020.1718)