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## 'Deepfakes' ranked as most serious AI crime threat

*Fake audio or video content has been ranked by experts as the most worrying use of artificial intelligence in terms of its potential applications for crime or terrorism, according to a new UCL report.*

The study, [published in Crime Science](#) and funded by the Dawes Centre for Future Crime at UCL (and available as a policy briefing), identified 20 ways AI could be used to facilitate crime over the next 15 years. These were ranked in order of concern - based on the harm they could cause, the potential for criminal profit or gain, how easy they would be to carry out and how difficult they would be to stop.

Authors said fake content would be difficult to detect and stop, and that it could have a variety of aims - from discrediting a public figure to extracting funds by impersonating a couple's son or daughter in a video call. Such content, they said, may lead to a widespread distrust of audio and visual evidence, which itself would be a societal harm.

Aside from fake content, five other AI-enabled crimes were judged to be of high concern. These were using driverless vehicles as weapons, helping to craft more tailored phishing messages (spear phishing), disrupting AI-controlled systems, harvesting online information for the purposes of large-scale blackmail, and AI-authored fake news.

Senior author Professor Lewis Griffin (UCL Computer Science) said: "As the capabilities of AI-based technologies expand, so too has their potential for criminal exploitation. To adequately prepare for possible AI threats, we need to identify what these threats might be, and how they may impact our lives."

Researchers compiled the 20 AI-enabled crimes from academic papers, news and current affairs reports, and fiction and popular

culture. They then gathered 31 people with an expertise in AI for two days of discussions to rank the severity of the potential crimes. The participants were drawn from academia, the private sector, the police, the government and state security agencies.

Crimes that were of medium concern included the sale of items and services fraudulently labelled as "AI", such as security screening and targeted advertising. These would be easy to achieve, with potentially large profits.

Crimes of low concern included burglar bots - small robots used to gain entry into properties through access points such as letterboxes or cat flaps - which were judged to be easy to defeat, for instance through letterbox cages, and AI-assisted stalking, which, although extremely damaging to individuals, could not operate at scale.

First author Dr Matthew Caldwell (UCL Computer Science) said: "People now conduct large parts of their lives online and their online activity can make and break reputations. Such an online environment, where data is property and information power, is ideally suited for exploitation by AI-based criminal activity.

"Unlike many traditional crimes, crimes in the digital realm can be easily shared, repeated, and even sold, allowing criminal techniques to be marketed and for crime to be provided as a service. This means criminals may be able to outsource the more challenging aspects of their AI-based crime."

Professor Shane Johnson, Director of the Dawes Centre for Future Crimes at UCL, which funded the study, said: "We live in an ever changing world which creates new opportunities - good and bad. As such, it is imperative that we anticipate future crime threats so that policy makers and other stakeholders with the competency to act can do so before new 'crime harvests' occur. This report is the first in a series that will identify the future crime threats associated with new and emerging technologies and what we might do about them."

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## **We Have Ploonets. We Have Moonmoons. Now Hold Onto Your Hats For... Blanets**

*The team led by Keiichi Wada of Kagoshima University in Japan  
has given a new name to these black hole planets blanets*

[Michelle Starr](#)

It's easy to think of [black holes](#) as voracious destruction machines, slurping up everything in their immediate vicinity. But that's not always the case. The environments around active supermassive black holes are complex, and last year, a team of astronomers showed that there's a safe zone around each supermassive black hole in which [thousands of planets could be orbiting](#).

Now, the team led by Keiichi Wada of Kagoshima University in Japan has given a new name to these black hole planets - "blanets", which is just delightful - and worked out how these blanets might form from the grains of dust swirling around the black hole.

"Here, we investigate the dust coagulation processes and physical conditions of the blanet formation," they wrote in a paper currently submitted to [The Astrophysical Journal](#) for peer review, and [uploaded to the pre-print service arXiv](#).

"Our results suggest that blanets could be formed around relatively low-luminosity active galactic nuclei during their lifetime."

We know that stars can be captured in orbit around supermassive black holes - astronomers have been observing the [complex dance of stars around Sagittarius A\\*](#), the supermassive black hole at the heart of the Milky Way, for decades.

It's also been hypothesised that exoplanets - both [orbiting those captured stars, or rogue](#) - can be captured by black holes, too.

But Wada's team proposes a new class of exoplanets, those that form directly around active supermassive black holes at the hearts of galaxies. Such an active black hole is surrounded by an accretion

disc, a huge torus of dust and gas swirling around, its inner rim feeding into the black hole.

This is a lot like how planets form around stars. A clump in a gas cloud gravitationally collapses in on itself, spinning; this is the protostar. As it spins, material from the surrounding cloud forms a disc that feeds into it, while a little farther away from the star, where the material is orbiting more stably, planets can form.

In the planetary formation process, the grains of dust that make up the disc start to cling together due to electrostatic forces. These larger pieces then start to collide with each other, gradually accumulating more and more grains until the object is massive enough for gravitational forces to take over. If nothing disrupts the process, after a few million years or so, you have a planet.

In their paper last year, Wada and his team found that, at sufficient distances from the black hole, blanet formation may be even more efficient than around stars, because the orbital velocity of the accretion disc is fast enough to keep the objects from escaping orbit and drifting towards the black hole.

But there were some problems with their calculations. Firstly, it's possible that, if the collisional velocity of the gas clumps is high enough, the initial dust aggregates could smash each other apart, instead of sticking together. Secondly, the clumps could grow very rapidly at the collisional stage, which does not fit a more natural dust density model.

With these constraints in mind, the team recalculated their blanet formation model outside the 'snowline', the distance from the central body at which volatile compounds can condense into ice. And they found that, if our planetary formation model is correct, there should indeed be conditions under which blanets can form.

If the viscosity of the disc is below a certain threshold, that will prevent the aggregates from destroying each other on collision. And,

because the formation of blanets is not subject to the same limitations as planets, they can be absolute chonkers.

Around a supermassive black hole clocking in at 1 million solar masses, blanets at the snow line could form in 70-80 million years. The farther they are from the black hole, the bigger they grow. According to the team's new calculations, at around 13 light-years from the black hole, blanets could range between 20 and 3,000 Earth masses, which is right at the upper limit for planetary mass as we know it.

For a black hole at 10 million solar masses, this mass can easily tip over into brown dwarf territory: bodies that are between gas giants and stars, fusing deuterium in their cores, but not quite massive enough for hydrogen fusion.

Of course, we can't actually detect these objects, which means they have to remain purely hypothetical for now. But they have joined a growing coterie of adorably named hypothetical cosmic objects, which includes [moonmoons](#) (moons of moons) and [ploonets](#) (the moons of large exoplanets that get kicked out of planetary orbit into stellar orbit, like a planet).

And, the researchers note, blanets open up interesting avenues for exploring the extreme space around supermassive black holes.

"Our results suggest that blanets could be formed around relatively low-luminosity active galactic nuclei during their lifetime (100 million years)," [they wrote in their paper](#).

"The gaseous envelope of a blanet should be negligibly small compared with the blanet mass. Therefore, the system of blanets are extraordinarily different from the standard Earth-type planets in the exoplanet systems. The dynamical stability of such a system around a supermassive black hole may be an interesting subject for future studies."

The research has been submitted to *The Astrophysical Journal* and is available on [arXiv](#).

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## Doctors diagnose advanced cancer—in a dinosaur

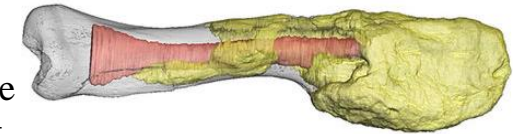
*This deformed bone is the first clear example of a malignant tumor diagnosed in a dinosaur.*

By [Gretchen Vogel](#)

The partial fibula—a bone from the lower leg—belonged to a horned, plant-eating *Centrosaurus* that lived roughly 76 million years ago in what is now Dinosaur Park in southern Alberta in Canada.

Paleontologists initially thought the bone's strange shape was due to a fracture that hadn't healed cleanly. But a new study, published today in *The Lancet Oncology*, compares the internal structure of the fossil (above) with a bone tumor from a human patient to seek a diagnosis.

The conclusion: [The dinosaur suffered from osteosarcoma](#), a cancer that, in humans, primarily attacks teens and young adults. The disease causes tumors of immature bone tissue, frequently in the long bones of the leg.



© Royal Ontario Museum/McMaster University

This isn't the first time cancer has been found in fossil remains. Scientists have identified [benign tumors in \*Tyrannosaurus rex\* fossils](#) and [arthritis in duck-billed hadrosaurs](#), as well as an [osteosarcoma in a 240-million-year-old turtle](#). But the researchers say their study is the first to confirm a dinosaur cancer diagnosis at the cellular level.

Scientists, including paleontologists, pathologists, a surgeon, and a radiologist, examined the full fossil with high-resolution computerized tomography scans and examined thin sections under the microscope to evaluate the structure of the cells. They found that the tumor was advanced enough that it had probably plagued

the animal for some time. A similar case in a human, left untreated, would likely be fatal, they write. However, because the fossil was found in a bone bed with lots of other *Centrosaurus* specimens, the dinosaur likely died in a flood with the rest of its herd and not from the cancer.

The researchers say their diagnosis shows a more careful look at unusual fossil malformations using modern imaging and diagnostic techniques can pay off, leading to new insights about the evolutionary origins of diseases.

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## Harvard Scientist Says We Need More Cheap, 'Crappy' Tests For COVID-19. Here's Why

*Call to authorize the sale of rapid tests which can be done out at home using a strip of paper that changes color in a quarter of an hour to give a result*

The aphorism "perfect is the enemy of good enough" has been played out to tragic effect in the US's inadequate testing for the [coronavirus](#), according to researchers calling for quick tests that cost only about a dollar each, and which may not be as accurate but can be carried out several times a week by the whole population.

Michael Mina, assistant professor of epidemiology at Harvard University, [has for weeks](#) been pushing for what he calls "crappy" tests.

His idea is to move away from the current high-precision molecular tests, known as PCR tests, which are still scarce in large swathes of the country and which people often have to wait hours to get done, and then have to wait days - or up to a week - for the results.

He has called for the Food and Drug Administration (FDA) to authorize the sale of rapid tests which can be done out at home using a strip of paper that changes color in a quarter of an hour to give a result, similar to a pregnancy test.

These tests have a low sensitivity, which means they miss a lot of positive results, and hence give a lot of "false negatives."

But for Mina and other experts, such a strategy would be more effective in terms of public health because across the whole population, the number of cases identified would be higher than under the current system.

The quick tests tend to be good at detecting people who emit a large amount of [virus](#), which is when they are more contagious, right at the beginning, while the PCR tests are very sensitive and can detect even small concentrations of the virus, when people are no longer as contagious.

"We're so focused on high-end expensive tests that we're not testing anyone," said Mina in the podcast "This Week in Virology."

"Maybe we only need a really crappy test," he said.

"If it's cheap enough to use it very frequently, then if it doesn't detect less than five percent of people when they're transmitting, maybe it detects 85 percent of people when they're transmitting. And that's a huge win over what we have right now."

The head of Harvard's Global Health Institute, Ashish Jha, touched on the subject on Monday.

"They're not actually crappy tests," he told reporters. "In certain circumstances they are not so sensitive when you have very low amounts of virus, and you're not doing much spreading. But when you're actually really infectious, you have large amounts of virus in your throat elsewhere and the test becomes much, much better," he said.

"From an epidemiologic point of view, that's when you want to capture people. You want to get them when they're infectious," he said. Even if rapid tests miss half the cases, it is likely that with two tests a week, they will end up detecting them.

It must also be noted that the current system is thought to be missing nine cases out of ten because so few people are being tested,

according to estimates by the Centers for Disease Control and Prevention.

The FDA has still not authorized the sale of any of the paper strip tests, which would cost between one and five dollars.

"I'm worried that our federal government is still stuck in a mental model that doesn't make sense for this [pandemic](#)," said Jha.

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### **Does the Common Cold Protect You from COVID-19?**

*There are emerging signs that some people might have heightened protection against SARS-CoV-2, perhaps thanks to recent infection by other coronaviruses.*

[Chris Baraniuk](#)

In labs all over the world lately, scientists working on COVID-19 have stumbled on an intriguing sort of finding again and again. They've found that blood samples from healthy people who were never exposed to the SARS-CoV-2 coronavirus contain reactive immune cells and targeted antibodies that could, perhaps, help stave off COVID-19.

These people may—it is still just a hypothesis—possess some degree of pre-existing immunity. If correct, it's even possible that this immunity has saved thousands from the worst manifestations of this terrible disease.

Some of the first hints of pre-existing immunity came via T cells, the white blood cells that destroy infected cells in the body or help other parts of the immune system target an invading pathogen. In one study originally published as a preprint on [medRxiv](#) April 22, a group of scientists in Germany reported an intriguing result.

Out of 68 healthy donors who had been tested for prior exposure to SARS-CoV-2 and who were found to be negative, 24 of them had a small number of T cells in their blood that reacted when exposed to the SARS-CoV-2 spike (S) protein—a complex structure protruding from the virus's exterior surface. The study, which was

later published in [Nature](#) July 29, explains that the cells in question produced proteins on their surfaces, an indication of an immune response.

If that is indeed what's going on here, one possible explanation would be that the healthy donors had been infected by another coronavirus relatively recently, perhaps one that causes a common cold, says coauthor [Andreas Thiel](#), an immunologist at the Charité hospital, part of Universitätsmedizin Berlin. Besides more serious diseases such as COVID-19 and SARS, [human coronaviruses](#) have been known for decades to cause what are usually much milder infections. The specific viruses that cause these illnesses are found all around the world.

Immunity to common cold viruses is not thought to be very long-lasting for people, regardless of age, so it is debatable how durable a protective effect would be. "Although these viruses are not very similar [to SARS-CoV-2], the low degree of similarity is of course sufficient that the immune system, at least partly, is cross-reacting, which is a very normal thing," he says.

An earlier study in [Nature](#) on July 15 from Singapore reported that 23 patients who had caught the original SARS virus 17 years ago and a further 37 individuals who had never been found to have had SARS or COVID-19 possessed CD4+ helper T cells and CD8+ killer T cells that reacted to the SARS-CoV-2 nucleocapsid (N) protein.

Lead author [Nina Le Bert](#), an immunologist at Duke-NUS Medical School in Singapore, says that her paper chimes with Thiel's work and [a few other studies](#) that have also found SARS-CoV-2 reactive T cells in blood from people who never had COVID-19, or who were sampled before the pandemic.

A study in [Science](#) today (August 4) also found SARS-CoV-2 reactive T cells in pre-pandemic blood samples from 25 healthy individuals. In this case, the authors also mapped 142 specific

points on the SARS-CoV-2 virus called epitopes associated with this activity.

This allowed them to show, in subsequent experiments, that the T cells also reacted when exposed to epitopes on common cold coronaviruses that were similar to SARS-CoV-2 epitopes, supporting the idea that previous exposure to these common viruses might leave our immune systems primed to respond to the novel coronavirus. Determining whether the T cell activity really is protective against COVID-19 is tricky, Le Bert says. “You would need to study people before and after getting infected.”

Le Bert adds that having some degree of immunity also does not mean that people definitely won’t get infected in the first place. They may still experience mild symptoms, for example, as their immune system fends off the virus.

Thiel points out that reactive T cells could even produce the opposite result—a detrimental immune response that ultimately harms the patient, for example, when someone experiences excessive inflammation or an inability to clear the virus. “Maybe particularly in the old people, having such cross-reactive T cells could be bad,” he suggests.

Pre-existing immunity might not be limited to T cells. A preprint published on [medRxiv](#) July 23 reports that SARS-CoV-2–reactive antibodies were found in blood samples taken from people in the UK between 2018 and early 2020, before COVID-19 became widespread in the country.

Not only did the authors find that 15 out of 262 people who never had COVID-19 have IgG antibodies reactive with certain SARS-CoV-2 proteins, but further tests showed that these antibodies had a neutralizing effect on the SARS-CoV-2 spike protein, which suggests that they might be able to restrict infection by the virus.

One of the most striking findings was that these antibodies were far more prevalent in children between the ages of 1 and 16 years old.

In fact, 60 percent of children had neutralizing IgG antibodies—an order of magnitude greater than the proportion of adults who were found to have the same antibodies. Coauthor [Rupert Beale](#), an immunologist at the Francis Crick Institute in London, remarked on [Twitter](#) that this particular result was completely unexpected—“a kind of bombshell,” as he put it.

In their preprint, the authors write that kids are generally more frequently exposed to other coronaviruses, such as those that cause common colds. This could explain the prevalence of those IgG antibodies in their blood.

It is notable that, while Beale’s team detected IgG neutralizing antibodies in some of their subjects, none of the healthy donors in the study by Thiel and his colleagues were found to have reactive IgG antibodies, though they did have reactive T cells.

The presence of neutralizing antibodies does not guarantee that these children are immune to COVID-19 but it does offer one possible explanation as to why children, generally speaking, experience milder symptoms when they catch the disease.

The findings are “really interesting,” says [Sheena Cruickshank](#), an immunologist at the University of Manchester in the UK, via email. She notes that, in the study, a different type of antibody that is protective against SARS-CoV-2, IgA, was not detected in the healthy individuals unexposed to the new coronavirus. That might mean any pre-existing immunity is limited. The other big caveat is that immunity to common cold viruses is not thought to be very long-lasting for people, regardless of age, so it is debatable how durable a protective effect would be, she adds.

In Le Bert’s study, patients appeared to have retained reactive T cells for nearly two decades. She and her colleagues write in their report that this has potentially significant implications: that immunity acquired through, say, a vaccine could last for many months or years.

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## The Coronavirus Is Never Going Away

*No matter what happens now, the virus will continue to circulate around the world.*

Sarah Zhang

The coronavirus that causes COVID-19 has sickened more than 16.5 million people across six continents. It is raging in countries that never contained the virus. It is [resurging in many of the ones that did](#). If there was ever a time when this coronavirus could be contained, it has probably passed. One outcome is now looking almost certain: This virus is never going away.

The coronavirus is simply too widespread and too transmissible. The most likely scenario, experts say, is that the pandemic ends at some point—because enough people have been either infected or vaccinated—but the virus continues to circulate in lower levels around the globe. Cases will wax and wane over time. Outbreaks will pop up here and there. Even when a much-anticipated vaccine arrives, it is likely to only [suppress but never completely eradicate the virus](#). (For context, consider that vaccines exist for more than a dozen human viruses but only one, smallpox, has ever been eradicated from the planet, and that took 15 years of [immense global coordination](#).) We will probably be living with this virus for the rest of our lives.

Back in the winter, public-health officials were more hopeful about SARS-CoV-2, the coronavirus that causes COVID-19. SARS, a closely related coronavirus, emerged in late 2002 and infected more than 8,000 people but was snuffed out through intense isolation, contact tracing, and quarantine. The virus was gone from [humans by 2004](#). SARS and SARS-CoV-2 differ in a crucial way, though: The new virus spreads more easily—and in many cases asymptotically. The strategies that succeeded with SARS are less effective when some of the people who transmit COVID-19

don't even know they are infected. "It's very unlikely we're going to be able to declare the kind of victory we did over SARS," says [Stephen Morse](#), an epidemiologist at Columbia University.

If not, then what does the future of COVID-19 look like? That will depend, says [Yonatan Grad](#), on the strength and duration of immunity against the virus. Grad, an infectious-disease researcher at Harvard, and his colleagues have [modeled a few possible trajectories](#). If immunity lasts only a few months, there could be a big pandemic followed by smaller outbreaks every year. If immunity lasts closer to two years, COVID-19 could peak every other year.

At this point, how long immunity to COVID-19 will last is unclear; the virus simply hasn't been infecting humans long enough for us to know. But related coronaviruses are reasonable points of comparison: In SARS, antibodies—[which are one component of immunity—wane after two years](#). Antibodies to a handful of other coronaviruses that cause common colds [fade in just a year](#). "The faster protection goes away, the more difficult for any project to try to move toward eradication," Grad told me.

This has implications for a vaccine, too. Rather than a onetime deal, a COVID-19 vaccine, when it arrives, could require booster shots to maintain immunity over time. You might get it every year or every other year, much like a flu shot.

Even if the virus were somehow eliminated from the human population, it could keep circulating in animals—and spread to humans again. SARS-CoV-2 likely originated as a bat virus, with a [still-unidentified animal perhaps serving as an intermediate host](#), which could continue to be a reservoir for the virus. ([SARS also originated](#) in bats, with catlike palm civets serving as an intermediate host—which led officials to order the [culling of thousands of civets](#).) [Timothy Sheahan](#), a virologist at the University of North Carolina at Chapel Hill, wonders if, with

SARS-CoV-2 so widespread across the globe, humans might be infecting new species and creating new animal reservoirs. “How do you begin to know the extent of virus spread outside of the human population and in wild and domestic animals?” he says. So far, [tigers at the Bronx Zoo](#) and [minks on Dutch farms](#) seem to have caught COVID-19 from humans and, in the case of the minks, passed the virus back to humans who work on the farm.

The existence of animal reservoirs that can keep reinfecting humans is also why scientists don’t speak of “eradication” for these viruses. The Ebola virus, for example, [probably comes from bats](#). Even though human-to-human transmission of Ebola eventually ended in the West African epidemic in 2016, the virus was still somewhere on Earth and could still infect humans if it found the right host. And indeed, [in 2018](#), Ebola broke out again in the Democratic Republic of the Congo. Ebola can be contained through contact tracing, isolation, and a new vaccine, but it cannot be “[eradicated](#).” No one is quite sure why SARS has never reemerged from an animal reservoir, but this coronavirus could well follow a different pattern.

In the best-case scenario, a vaccine and better treatments blunt COVID-19’s severity, making it a much less dangerous and less disruptive disease. Over time, SARS-CoV-2 becomes just another seasonal respiratory virus, like the four other coronaviruses that cause a sizable proportion of common colds: 229E, OC43, NL63, and HKU1. These cold coronaviruses are so common that we have likely all had them at some point, maybe even multiple times. They can cause serious outbreaks, [especially in the elderly](#), but are usually mild enough to fly under the radar. One endgame is that SARS-CoV-2 becomes the fifth coronavirus that regularly circulates among humans.

In fact, virologists have wondered whether the common-cold coronaviruses also got their start as a pandemic, before settling in as routine viruses. In 2005, biologists in Belgium studied mutations in

the cold coronavirus OC43, which likely evolved from a closely related coronavirus that infects cows. Because genetic mutations accumulate at a somewhat regular rate, the researchers were able to date the spillover from cows into humans to the late 1800s. Around this time, a highly infectious respiratory disease was killing cows, and even more curiously, in 1889, a human pandemic began killing people around the world. The older people were, the more susceptible they were. This illness, which produced “[malaise, fever, and pronounced central nervous system symptoms](#),” was linked to influenza based on the antibodies found in survivors half a century later. But the cause was never definitively proved from tissue samples.

Could it have been a coronavirus that jumped from cows to humans? This is all speculative, and the possible links between the other three cold coronaviruses and past pandemics are even less clear, says [Burtram Fielding](#), a coronavirus researcher at the University of the Western Cape. “But,” he says, “I wouldn’t be surprised.” It would also be good news, in a way, because it would suggest that COVID-19 could become less deadly over time, making that transition from pandemic to common cold.

With a virus, there is a general trade-off between how contagious it is and how deadly it is. SARS and SARS-CoV-2 are illustrative points of comparison: The earlier virus killed a much higher proportion of patients, but it also did not spread as easily. And what a virus ultimately wants to do is keep spreading, which is much easier to do from a live, walking host than a dead one. “In the grand scheme of things, you know, a dead host doesn’t help the virus,” says [Vineet Menachery](#), a coronavirus researcher at the University of Texas Medical Branch. The other four coronaviruses may also be less deadly because we have all encountered them as children, and even if our immunity does not prevent us from getting them again, it may still prevent severe disease. All of this, along with immunity



from vaccines, means that COVID-19 is likely to become far less disruptive down the line.

Influenza might be another useful point of comparison. The “flu” is not one virus but actually several different strains that circulate seasonally. After pandemics like 2009’s H1N1 flu, also known as swine flu, the pandemic strain does not simply disappear. Instead, it turns into a [seasonal flu strain](#) that circulates all year but peaks during the winter. A descendent of the 2009 H1N1 pandemic strain is [still the seasonal flu today](#). The seasonal peaks never quite reach pandemic heights because of building immunity in the population. Eventually, a new strain, against which people have no immunity, comes along and sparks a new pandemic, and then it becomes the new dominant seasonal strain.

In this way, the long-term outlook for COVID-19 might offer some hope for a return to normal. “I think this virus is with us to the future,” [Ruth Karron](#), a vaccine researcher at Johns Hopkins, told me. “But so is influenza with us, and for the most part, flu doesn’t shut down our societies. We manage it.”

We want to hear what you think about this article. [Submit a letter](#) to the editor or write to [letters@theatlantic.com](mailto:letters@theatlantic.com).

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

## **Dozens of pesticides linked with mammary gland tumors in animal studies**

### ***Findings have implications for how federal agencies assess pesticides for breast cancer risk***

In an analysis of how regulators review pesticides for their potential to cause cancer, researchers at Silent Spring Institute identified more than two dozen registered pesticides that were linked with mammary gland tumors in animal studies. The new findings raise concerns about how the US Environmental Protection Agency (EPA) approves pesticides for use and the role of certain pesticides in the development of breast cancer.

Several years ago, a resident on Cape Cod in Massachusetts contacted researchers at Silent Spring looking for information on an herbicide called triclopyr. Utility companies were looking to spray the chemical below power lines on the Cape to control vegetation.

"We know pesticides like DDT increase breast cancer risk, so we decided to look into it," says co-author Ruthann Rudel, an environmental toxicologist and director of research at Silent Spring. "After examining pesticide registration documents from EPA, we found two separate studies in which rodents developed mammary gland tumors after being exposed to triclopyr, yet for some reason regulators dismissed the information in their decision not to treat it as a carcinogen."

When manufacturers apply to register a pesticide, EPA reviews existing studies and based on those studies assigns the chemical a cancer classification--for instance, how likely or unlikely the chemical is to cause cancer. After reviewing triclopyr, Silent Spring researchers wondered if evidence of mammary tumors was being ignored for other pesticides as well.

[Reporting in the journal \*Molecular and Cellular Endocrinology\*](#), Rudel and Silent Spring scientist Bethsaida Cardona reviewed more than 400 EPA pesticide documents summarizing the health effects of each registered pesticide. They found a total of 28 pesticides linked with mammary gland tumors, yet EPA acknowledged only nine of them as causing mammary tumors and dismissed the evidence entirely for the remaining 19.

Rudel and Cardona also found that many of the pesticides in their analysis behaved like endocrine disruptors, for instance, by interfering with estrogen and progesterone. "Breast cancer is highly influenced by reproductive hormones, which stimulate the proliferation of cells within the breast, making it more susceptible to tumors," says Rudel. "So, it's important that regulators consider

this kind of evidence. If they don't, they risk exposing people to pesticides that are breast carcinogens."

Traditionally, toxicologists focus on whether a chemical causes DNA damage when determining its potential to cause cancer. But recent findings in cancer biology show there are [many ways chemicals can trigger the development of cancer](#). For example, chemicals can suppress the immune system, cause chronic inflammation, or disrupt the body's system of hormones, all of which can lead to the growth of breast tumors and other types of tumors as well.

"In light of our findings, we hope EPA updates its guidelines for assessing mammary gland tumors by considering evidence that more completely captures the biology of breast cancer, such as the effects of endocrine disruptors," says Cardona.

Rudel and Cardona recommend that EPA re-evaluate five pesticides in particular--IPBC, triclopyr, malathion, atrazine and propylene oxide--due to their widespread use and the evidence uncovered in the new analysis. IPBC is a preservative in cosmetics; triclopyr is an agricultural herbicide that is also used to control vegetation growth along rights-of-way; malathion is a common residential and agricultural pesticide and is used in some lice treatments; atrazine is one of the most commonly-used herbicides in agriculture; and propylene oxide is used to preserve food, cosmetics, and pharmaceuticals, and has many similarities with ethylene oxide, a known human carcinogen.

The project is part of Silent Spring Institute's [Safer Chemicals Program](#) which is developing new cost-effective ways of screening chemicals for their effects on the breast. Knowledge generated by this effort will help government agencies regulate chemicals more effectively and assist companies in developing safer products.

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**Reference:**

*Cardona, B. and R.A. Rudel. 2020. US EPA's regulatory pesticide evaluations need clearer guidelines for considering mammary gland tumors and other mammary gland effects. Molecular and Cellular Endocrinology. DOI: 10.1016/j.mce.2020.110927*

<https://wb.md/30zNLRj>

## **CDC Anticipates 2020 Outbreak of Acute Flaccid Myelitis**

***Officials at the Centers for Disease Control and Prevention (CDC) anticipate another peak year for acute flaccid myelitis (AFM) and encourage clinicians to be prepared to recognize AFM and immediately hospitalize patients.***

**Megan Brooks**

AFM is a medical emergency, CDC Director Robert R. Redfield, MD, said today during a press briefing.

"We are pushing AFM information out far and wide to educate all clinicians, especially frontline providers, to alert and prepare them for increased cases this year," he said.

AFM is a rare but serious neurologic condition primarily affecting children. It is characterized by the sudden onset of arm or leg weakness that can progress quickly; patients can become paralyzed over the course of hours or days and require a ventilator to help them breathe. Some patients will be permanently disabled.

### **Details of 2018 Outbreak**

Since 2014, cases of AFM in the United States have spiked every 2 years between August and November. The largest outbreak occurred 2 years ago, in 2018, with 238 confirmed cases in 42 states.

[Enteroviruses](#), particularly enterovirus-D68, are likely responsible for these peaks in cases, insofar as these viruses tend to circulate every 2 years, Redfield said.

"This means that it will be circulating at the same time as [influenza](#) and other infectious disease, including COVID-19, and could be another outbreak for clinicians, parents, and children to deal with," Redfield said.

In a *Vital Signs* report [released today](#), CDC researchers describe the characteristics of the 238 AFM cases from 2018. Most cases were in young children (mean age, 5.3 years); 58% of the patients were male; and 86% of the cases occurred during August and November. Most children who developed AFM had fever or respiratory illness about 6 days before the onset of limb weakness. Once limb weakness developed, it was common for them also to have difficulty walking, as well as neck or [back pain](#), limb pain, and fever.

"Clinicians should suspect AFM in patients with sudden limb weakness, especially during the months of August through November. Recent respiratory illness or fever in the presence of neck or back pain or any neurological symptom should heighten the clinicians concern," Redfield said.

In the 2018 outbreak, approximately three quarters of patients were brought to medical care within 1 day. Most went to the emergency department. Overall, 98% of patients with AFM were hospitalized; 54% were admitted to intensive care units; and 23% required [mechanical ventilation](#).

Speaking at the briefing, Thomas Clark, MD, MPH, pediatrician and deputy director of the Division of Viral Diseases at CDC, noted that although most patients were hospitalized within 1 day of developing limb weakness, 10% were not hospitalized until 4 or more days after developing limb weakness.

"This could indicate delays in recognition of AFM and present an opportunity for improvement in patient outcomes," Clark said.

He noted that enterovirus-D68 was the most common virus identified among specimens tested from patients. Poliovirus, a

vaccine-preventable cause of paralysis, was not detected in any of the cases. Patients who tested positive for enterovirus-D68 typically had more severe AFM and were more likely to require intensive care and ventilation.

"All clinicians should remain vigilant for AFM and promptly evaluate patients," Clark said in a statement. "During the COVID-19 pandemic, this may require adjusting practices to perform clinical evaluations of patients by phone or telemedicine. However, clinicians should not delay hospitalizing patients when they suspect AFM," he added.

*Morb Mortal Wkly Rep. Published online August 4, 2020. [Full text](#)*

<https://bit.ly/2XQe6ZN>

### **Blood-thinner with no bleeding side-effects is here**

*In a study led by EPFL, scientists have developed a synthetic blood-thinner that, unlike all others, doesn't cause bleeding side-effects.*

Patients who suffer from thrombosis, pulmonary embolism or stroke are usually put on drugs that help their blood flow more smoothly through their body. Occupying a large section of the drug market, anticoagulants, or "blood thinners" as they are popularly known, can keep blood clots from forming or getting bigger, and can therefore help with recover from heart defects or prevent further complications.

But there is a catch: blood thinners work by blocking enzymes that help to stop bleeding after an injury. Because of this, virtually every blood thinner available today can lead to serious, and even life-threatening bleeding following an injury.

The problem remained unsolved until a few years ago, when a study was carried out on mice that had been genetically modified to be deficient in an enzyme that normally helps blood clot. The enzyme is called "coagulation factor XII" (FXII), and the mice without the enzyme had a very reduced risk of thrombosis without

having bleeding side-effects. The discovery triggered a race for FXII inhibitors.

### **Finally, a synthetic inhibitor**

Participating in the race, the Laboratory of Therapeutic Proteins and Peptides of Professor Christian Heinis at EPFL has developed the first synthetic inhibitor of FXII. The inhibitor has high potency, high selectivity, and is highly stable, with a plasma half-life of over 120 hours. [Published in \*Nature Communications\*](#), the study is the result of an extensive collaboration with three other labs in Switzerland and the US.

"The FXII inhibitor is a variation of a cyclic peptide that we identified in a pool of more than a billion different peptides, using a technique named phage display," says Heinis. The researchers then improved the inhibitor by painstakingly replacing several of its natural amino acids with synthetic ones. "This wasn't a quick task; it took over six year and two generations of PhD students and post-docs to complete."

With a potent FXII inhibitor in hand, Heinis's group wanted to evaluate it in actual disease models. To do this, they teamed up with experts in blood and disease-modeling at the University Hospital of Bern (Inselspital) and the University of Bern.

Working with the group of Professor Anne Angellillo-Scherrer (Inselspital), they showed that the inhibitor efficiently blocks coagulation in a thrombosis model without increasing the bleeding risk. Then they assessed the inhibitor's pharmacokinetic properties with the group of Professor Robert Rieben (University of Bern). "Our collaboration found that it is possible to achieve bleeding-free anti-coagulation with a synthetic inhibitor," says Heinis.

### **Artificial lungs**

"The new FXII inhibitor is a promising candidate for safe thromboprotection in artificial lungs, which are used to bridge the time between lung failure and lung transplantation," says Heinis.

"In these devices, contact of blood proteins with artificial surfaces such as the membrane of the oxygenator or tubing can cause blood clotting." Known as 'contact activation', this can lead to severe complications or even death and limits the use of artificial lungs for longer than a few days or weeks.

To test the effectiveness of the FXII inhibitor in artificial lungs, Heinis's group turned to Professor Keith Cook at Carnegie Mellon University (US), an expert for artificial lung system engineering. Cook's group tested the inhibitor in an artificial lung model, and found that it efficiently reduced blood clotting, all without any bleeding side-effects.

The only problem is that the inhibitor has a relatively short retention time in the body: it's too small and the kidneys would filter it out. In the context of artificial lungs, this would mean constant infusion, since suppressing blood clotting for several days, weeks or months requires a long circulation time.

But Heinis is optimistic: "We're fixing this; we're currently engineering variants of the FXII inhibitor with a longer retention time."

<https://bit.ly/3a8tIN3>

### **When mammals ate dinosaurs**

*The cervical rib of a long-necked dinosaur from northwest China provides the oldest known evidence to date that early mammals fed on dinosaur meat around 160 million years ago.*

A research team led by Professor Hans-Ulrich Pfretzschner from the Department of Geosciences at the University of Tübingen discovered bite marks of a mammal the size of a modern shrew on a bone fragment of a sauropod that was approximately 20 meters long and weighed several tons. The researchers say the mammals were probably eating a dinosaur's carcass; this was the only way for such a small animal to eat a large one. This discovery, which provides

information on the life and environment of the early mammals, has been published in the journal *The Science of Nature*.

"The [early mammals](#) lived in the shadow of the [dinosaurs](#) for more than 160 million years. On average they reached a weight of about one hundred grams," says Felix Augustin from the research team, the first author of the new study. "However, we now know that they nevertheless developed an astonishing biodiversity and occupied a large number of ecological niches." Alongside the numerous insect-eating ground-dwellers, there were also semi-aquatic, tree-dwelling, digging, and even gliding mammals. This diversity is reflected in their different diets, which researchers can determine indirectly by examining the shape of teeth and jaws. "Direct evidence such as [bite marks](#) on bones or stomach contents is very rare," says Augustin. "Furthermore, all the evidence we have to date dates back to the Cretaceous period at the earliest and is at most about 100 million years old. That's why our discovery from about 160 million years ago is so special."

#### **Rich fossil site**

In 2000, researchers of a Chinese-German expedition excavated numerous fossils of vertebrates such as turtles and crocodiles, dinosaurs and mammals from the Jurassic period, the time about 160 million years BCE, from what is now the Junggar Basin in the province of Xinjiang in northwest China. While re-examining the fossil bones, the team noticed tiny gnaw marks on a fragment of [bone](#), which on closer examination turned out to be bite marks made by early mammals. The researchers working in vertebrate paleontology compared the notches with a large number of similar marks on fossilized and unfossilized bones. "The gnaw marks were very similar to those of today's insect-eating mammals, such as shrews," says Augustin.

Due to the extreme difference in size, the researchers assume that the mammals ate the remains of one animal only. "The marks

provide valuable insights into the biology of these early mammals from China, which according to the reconstructions were very small insectivorous or omnivorous animals. We were able to prove for the first time that they were not above eating carrion," says Hans-Ulrich Pfretzschner. This behavior is also seen in modern insectivores and other small mammals such as rodents. The surrounding rock in the Junggar Basin provided additional information about the environmental conditions at the time and suggest that the northwest of China had rivers and floodplains and a dry, warm climate when these dinosaurs were alive.

*More information:* Felix J. Augustin et al. *The smallest eating the largest: the oldest mammalian feeding traces on dinosaur bone from the Late Jurassic of the Junggar Basin (northwestern China)*, *The Science of Nature* (2020). DOI: [10.1007/s00114-020-01688-9](https://doi.org/10.1007/s00114-020-01688-9)

<https://nyti.ms/3a8f7kN>

## **Scientists Uncover Biological Signatures of the Worst Covid-19 Cases**

*Studies of patients with severe cases of Covid-19 show the immune system lacks its usual coordinated response.*

By [Katherine J. Wu](#)

Scientists are beginning to untangle one of the most complex biological mysteries of the coronavirus pandemic: Why do some people get severely sick, whereas others quickly recover?

In certain patients, according to a [flurry of recent studies](#), the virus appears to make the immune system go haywire.

Unable to marshal the right cells and molecules to fight off the invader, the bodies of the infected instead launch an entire arsenal of weapons — a misguided barrage that can wreak havoc on healthy tissues, experts said.

"We are seeing some crazy things coming up at various stages of infection," said Akiko Iwasaki, an immunologist at Yale University who led one of the new studies.

Researchers studying these unusual responses are finding patterns that distinguish patients on the path to recovery from those who fare far worse. Insights gleaned from the data might [help tailor treatments to individuals](#), easing symptoms or perhaps even vanquishing the virus before it has a chance to [push the immune system too far](#).

“A lot of these data are telling us that we need to be acting pretty early in this process,” said John Wherry, an immunologist at the University of Pennsylvania who recently published a study of these telltale immune signatures. As more findings come out, researchers may be able to begin testing the idea that “we can change the trajectory of disease,” he said.

When a more familiar respiratory infection, like a flu virus, tries to gain a foothold in the body, the immune response launches a defense in two orchestrated acts. First, a cavalry of fast-acting fighters flocks to the site of infection and tries to corral the invader, buying the rest of the immune system time to mount a more tailored attack.

Much of the early response depends on [signaling molecules called cytokines](#) that are produced in response to a virus. Like microscopic alarms, cytokines can mobilize reinforcements from elsewhere in the body, triggering a round of inflammation.

Eventually, these cells and molecules leading the initial charge will stand down, making way for [antibodies and T cells](#) — specialized assassins built to home in on the virus and the cells it has infected.

But this coordinated handoff seems to break down in people with severe Covid-19.

Rather than bowing out gracefully, the cytokines that drive the first surge never stop sounding the alarm, even after antibodies and T cells arrive on the scene. That means the wildfire response of inflammation may never get snuffed out, even when it’s no longer needed.

“It’s normal to develop inflammation during a viral infection,” said Catherine Blish, a viral immunologist at Stanford University. “The problem comes when you can’t resolve it.”

This sustained signaling may result in part from the body’s inability to keep the virus in check, Dr. Iwasaki said. Many who struggle to recover from their illness seem to harbor the pathogen long after other patients have purged it, perhaps goading the immune system into prolonging its frantic inflammatory siege.

[Plenty of other viruses](#), including those that cause AIDS and herpes, have evolved tricks to elude the immune system. Recent evidence hints that the new coronavirus might have a way of delaying or muffling interferon, one of the [earliest cytokine defenses](#) the body mounts.

The failure of this first line of defense may dupe the immune system into sounding its alarm bells even louder, dragging out the response into something destructive. “It’s an enigma,” said Avery August, an immunologist at Cornell University. “You have this raging immune response, but the virus continues to replicate.”

And the quality of these cytokines may matter as much as the quantity. In a [paper published last week in Nature](#), Dr. Iwasaki and her colleagues showed that patients with severe Covid-19 appear to be churning out signals that are better suited to subduing pathogens that aren’t viruses.

Although the delineations aren’t always clear-cut, the immune system’s responses to pathogens can be [roughly grouped into three categories](#): type 1, which is directed against viruses and certain bacteria that infiltrate our cells; type 2, which fights parasites like worms that don’t invade cells; and type 3, which goes after fungi and bacteria that can survive outside of cells. Each branch uses different cytokines to rouse different subsets of molecular fighters.

People with moderate cases of Covid-19 take what seems like the most sensible approach, concentrating on type 1 responses, Dr.

Iwasaki's team found. Patients struggling to recover, on the other hand, seem to be pouring an unusual number of resources into type 2 and type 3 responses, which is kind of "wacky," Dr. Iwasaki said. "As far as we know, there is no parasite involved."

It's almost as if the immune system is struggling to "pick a lane," Dr. Wherry said.

This disorientation also seems to extend into the realm of B cells and T cells — two types of immune fighters that usually need to stay in conversation to coordinate their attacks. Certain types of T cells, for instance, are crucial for coaxing B cells into manufacturing disease-fighting antibodies.

Last month, Dr. Wherry and his colleagues [published a paper in Science](#) finding that, in many patients with severe Covid-19, the virus had somehow driven a wedge between these two close-knit cellular communities. It's too soon to tell for sure, but perhaps something about the coronavirus is preventing B and T cells from "talking to each other," he said.

These studies suggest that treating bad cases of Covid-19 might require an immunological reset — drugs that could, in theory, restore the balance in the body and resurrect lines of communication between bamboozled cells. Such therapies could even be focused on specific subsets of patients whose bodies are responding bizarrely to the virus, Dr. Blish said: "the ones who have deranged cytokines from the beginning."

But that's easier said than done. "The challenge here is trying to blunt the response, without completely suppressing it, and getting the right types of responses," Dr. August said. "It's hard to fine-tune that."

[Timing](#) is also crucial. Dose a patient too early with a drug that tempers immune signaling, and they may not respond strongly enough; give it too late, and the worst of the damage may have already been done. The same goes for treatments intended to shore

up the initial immune response against the coronavirus, like [interferon-based therapies](#), Dr. Blish said. These could stamp out the pathogen if [given shortly after infection](#) — or run roughshod over the body if [administered after too long of a delay](#).

So far, treatments that block the effects of [one cytokine at a time](#) have yielded mixed or lackluster results — perhaps because researchers haven't yet identified the right combinations of signals that drive disease, said Donna Farber, an immunologist at Columbia University.

Steroids like dexamethasone, on the other hand, are like "big hammers" that can curb the activity of multiple cytokines at once, Dr. Farber said. Early clinical trials have hinted at dexamethasone's benefits against severe cases of the coronavirus, and more are underway. Such [broad-acting treatments have their downsides](#). But, she added, "it seems that's a good strategy, until we know more."

<https://bit.ly/2XIZJq5>

**New research suggests racism could be a genetic trait**  
*Beliefs that some groups are superior to others are deeply influenced by genetics.*

Black Lives Matter (BLM) is an anti-racist movement in the United States, founded as a reaction to many incidents of racism and brutal police violence against black people. The movement got widespread international support in 2020 after the police murder on the Afro-American George Floyd. The murder set off a chain of demonstrations all over the world.

The death of U.S. citizen George Floyd caused demonstrations all over the world. Still, police violence against [vulnerable groups](#) and minorities is nothing new. How can racist attitudes and practices have survived so many generations?

According to a study published in the journal *Proceedings of the National Academy of Sciences (PNAS)*, beliefs that some groups are superior to others are deeply influenced by genetics.

## Racist Genes

The researchers looked into why some attitudes tend to appear simultaneously. This may for example apply to the perception of some groups being better than others, or to the perception that certain ethnic and cultural groups are more capable of making decisions in society.

Previous research has suggested that such opinions often appear together, and that the environment only rarely shapes them.

Could it be that we are born with predispositions to certain political opinions? According to the findings, the answer is yes.

"People who share the same sets of attitudes also appear to share the same genes," said Thomas Haarklau Kleppestø, Ph.D. fellow at the Department of Psychology, University of Oslo.

## Political Attitudes

Around 2,000 adult Norwegian twins, identical and non-identical, answered a questionnaire to measure their social dominance orientation (SDO), a personality trait where a high score indicates a preference of a societal hierarchy.

Former research has linked this trait to political attitudes. A high score increases the possibility for support to items such as "Some groups of people must be kept in their place," and "Some groups of people are inferior to other groups."

The participants were to state their opinions on eight political proposals, such as strict immigration control and deportation of Romani people. Former research has found these proposals to correlate with SDO.

The researchers reasoned as follows: If the political opinions of [identical twins](#) were more alike than among non-identical, the reason would be genetic. Identical twins share 100 percent of their genes, while non-identical share 50 percent.

Therese Lillefosse (41), the identical twin sister of Kathrine Lillefosse, does believe that identical twins often share a common mind-set.

"In upper secondary school we made the same mistakes on our tests. On one occasion, the essays we wrote were identical to the extent of our teacher suspecting we had cheated," she said.

The sisters did not participate in Kleppestø's study, but how would they answer if asked the same questions in separate interviews?

First, eighteen questions about social dominance orientation reveal that Kathrine and Therese share similar thoughts on this matter.

None of them favors a hierarchical society.

## Fellowship of Genes

As expected, the researchers found a link between SDO and political attitudes. e.g., those who favored a hierarchical structure in society often wished for stricter immigration control and reduced foreign aid.

However, the findings also revealed that peoples' SDO had a genetic connection to all the eight measured political attitudes.

According to Kleppestø, this could partly explain the link between the political attitudes.

"We do not believe that our genome directly controls our political attitudes. However, we speculate that we are born with a predisposition that is strengthened over time, for example when we find friends with similar preferences," Kleppestø said.

The researchers believe that you may be born with a personality trait that could lead you into environments where it is enforced. So-called active gene-environment-correlation is well-known phenomenon in behavioral genetics.

## When Their Paths Parted

Kathrine and Therese Lillefosse had similar lives until they started comprehensive school. They chose different specializations and got new friends. Today, they share many friends, but not all.



Their lives are quite different now. They used to work in the same company, but Therese got a chronic illness and chose [family life](#) over her work. Today she has a husband, two kids, and no job. Kathrine is single and runs her own styling business.

When asked about their political opinions, they agree on several points. For instance, both wish for more foreign aid. However, on two questions their opinions are far apart: Kathrine would like a stricter immigration policy, while Therese would not. Kathrine would also support deportation of Romani people. Therese disagrees.

### Not Entirely Alike

Thomas Haarklau Kleppestø is not surprised.

"It is quite common that identical twins on average are more similar than non-identical. However, that does not mean that all identical twins are completely alike," he explained. "If identical twins were completely alike and non-identical fifty percent alike, genetics would explain all variation. Mystery solved. It is not like that."

As early as in childhood one can see differences between identical twins, in spite of common genes and environment. According to Kleppestø, this has to do with the brain. Our most complex organ contains around 88 billion neurons. Each one connects to thousands of other neurons. Some connections disappear, others become stronger.

"The genes provide some rules for these connections. However, coincidences will always occur. It is like baking a cake; even if you use the same recipe, the cakes are never 100 percent the same."

Experiences and environments also affect all humans. "Particularly systematic experiences will affect you, for instance whether you have been married or single for 20 years," Kleppestø said.

### Becoming a Mum Softens Up

Kathrine and Therese Lillefosse are not surprised that their mindsets today differ a bit. At certain points, though, they are

astounded. "Therese is more engaged than I thought she would be. I didn't think she cared so much about Romani people and immigration policies," Kathrine Lillefosse said.

She believes her part time job as a bartender to be the root of her own skepticism. She had some experiences that did not boost her support for immigration.

Therese, on the other hand, believes that becoming a mother made her softer. "Earlier, Kathrine was the more flexible one. Now, when I raise my kids, I want them to treat others as they wish others to treat them. It is important to be inclusive," Therese Lillefosse said.

Through her children, she also met immigrants. She asked herself why they should not be there, while others can.

### Political Personality

Leif Edward Ottesen Kennair, a psychology professor at the Norwegian University of Science and Technology (NTNU), believes that Kleppestø's study confirms former studies.

"We have long known that there is a genetic base for attitudes or political orientation. Studies like this make us able to call it a general finding," Kennair said.

He added that the researchers also provided new knowledge.

"For example, they have found that our genes may provide us with a political personality. However, this is on a group level. We also develop in interaction with the environment."

The study suggests that upbringing and family relations have a minor effect on attitudes. If you feel that you and your family are alike, genetics are the most important reason, according to Kennair.

"The environment affects us. However, it affects us mainly by making us less—not more—similar to our family," Kennair said.

*More information:* Thomas Haarklau Kleppestø et al. *Correlations between social dominance orientation and political attitudes reflect common genetic underpinnings*, *Proceedings of the National Academy of Sciences* (2019). [DOI: 10.1073/pnas.1818711116](https://doi.org/10.1073/pnas.1818711116)

*Journal information:* [Proceedings of the National Academy of Sciences](#)

<https://bit.ly/30Go7KD>

## These dogs are trained to sniff out the coronavirus.

Most have a 100% success rate

*What does a pandemic smell like? If dogs could talk, they might be able to tell us.*

Susan Hazel \* Anne-Lise Chaber \*\*

We're part of an international research team, [led by](#) Dominique Grandjean at France's National Veterinary School of Alfort, that has been training detector dogs to sniff out traces of the novel coronavirus (SARS-CoV-2) since March.

These detector dogs are trained using sweat samples from people infected with [COVID-19](#). When introduced to a line of sweat samples, most dogs can detect a positive one from a line of negative ones with 100% accuracy.

Across the globe, coronavirus detector dogs are being trained in the United Arab Emirates (UAE), Chile, Argentina, Brazil and Belgium. In the UAE, detector dogs – stationed at various airports – have already started [helping efforts](#) to control COVID-19's spread. This is something we hope will soon be available in Australia too.

### A keen nose

Our international colleagues found detector dogs were able to detect SARS-CoV-2 in infected people when they were still [asymptomatic](#) before later testing positive. When it comes to SARS-CoV-2 detection, we don't know for sure what the dogs are smelling.

The volatile organic compounds (VOCs) given off in the sweat samples are a complex mix. So it's likely the dogs are detecting a particular profile rather than individual compounds.

Sweat is used for tests as it's [not considered infectious](#) for COVID-19. This means it presents less risk when handling samples.

### COVID-19 sniffing dogs in Australia

Here in Australia, we're currently working with professional trainers of detector dogs in South Australia, Victoria and New

South Wales. The most common breed used for this work so far has been the German shepherd, with various other breeds also involved. We are also negotiating with health authorities to collect sweat samples from people who have tested positive to the virus, and from those who are negative. We hope to start collecting these within the next few months.

We will need to collect thousands of negative samples to make sure the dogs aren't detecting other viral infection, such as the common cold or influenza. In other countries, they've passed this test with flying colours.

Once operational, detector dogs in Australia could be hugely valuable in many scenarios, such as screening people at airports and state borders, or monitoring staff working in aged care facilities and hospitals daily (so they don't need repeat testing).

To properly train a dog to detect SARS-CoV-2, it takes:

- *6-8 weeks for a dog that is already trained to detect other scents,*
- or*
- *3-6 months for a dog that has never been trained.*

### Could the dogs spread the virus further?

Dogs in experimental studies have not been shown to be able to [replicate the virus](#) (within their body). Simply, they themselves are not a source of infection.

Currently, there are two case reports in the world of dogs being potentially contaminated with the COVID-19 virus by their owners. Those dogs didn't become sick.

To further reduce any potential risk of transmission to both people and dogs, the apparatus used to train the dogs doesn't allow any direct contact between the dog's nose and the sweat sample.

The dog's nose goes into a stainless steel cone, with the sweat sample in a receptacle behind. This allows free access to the volatile olfactory compounds but no physical contact.

Furthermore, all the dogs trained to detect COVID-19 are regularly checked by nasal swab tests, rectal swab tests and blood tests to identify antibodies. So far, none of the detector dogs has been found to be infected.

### Hurdles to jump

Now and in the future, it will be important for us to identify any instances where detector dogs may present false positives (signalling a sample is positive when it's negative) or false negatives (signalling the sample is negative when it's positive).

We're also hoping our work can reveal exactly which volatile olfactory compound(s) is/are specific to COVID-19 infection.

This knowledge might help us understand the disease process resulting from COVID-19 infection – and in detecting other diseases using detector dogs.

This pandemic has been a huge challenge for everyone. Being able to find [asymptomatic people](#) infected with the coronavirus would be a game-changer – and that's what we need right now.

### A friend to us (and science)

Perhaps we shouldn't be surprised about dogs' ability to detect COVID-19, as we already know their noses are amazing.

Dogs can help detect [hypoglycaemia in diabetics](#), warn people who are about to have an [epileptic seizure](#) and have been used to [sniff out some cancers](#).

Their great potential in dealing with the current pandemic is just one of myriad examples of how dogs enrich our lives.

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### Disclosure statement

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<https://bit.ly/3it2Rhp>

## Dinosaur relative's genome linked to mammals

### *A rare reptile whose ancestors once roamed the earth with dinosaurs*

Scientists from the University of Adelaide and South Australian Museum have collaborated with Otago University, New Zealand and a global team to sequence the genome of the tuatara - a rare reptile whose ancestors once roamed the earth with dinosaurs.

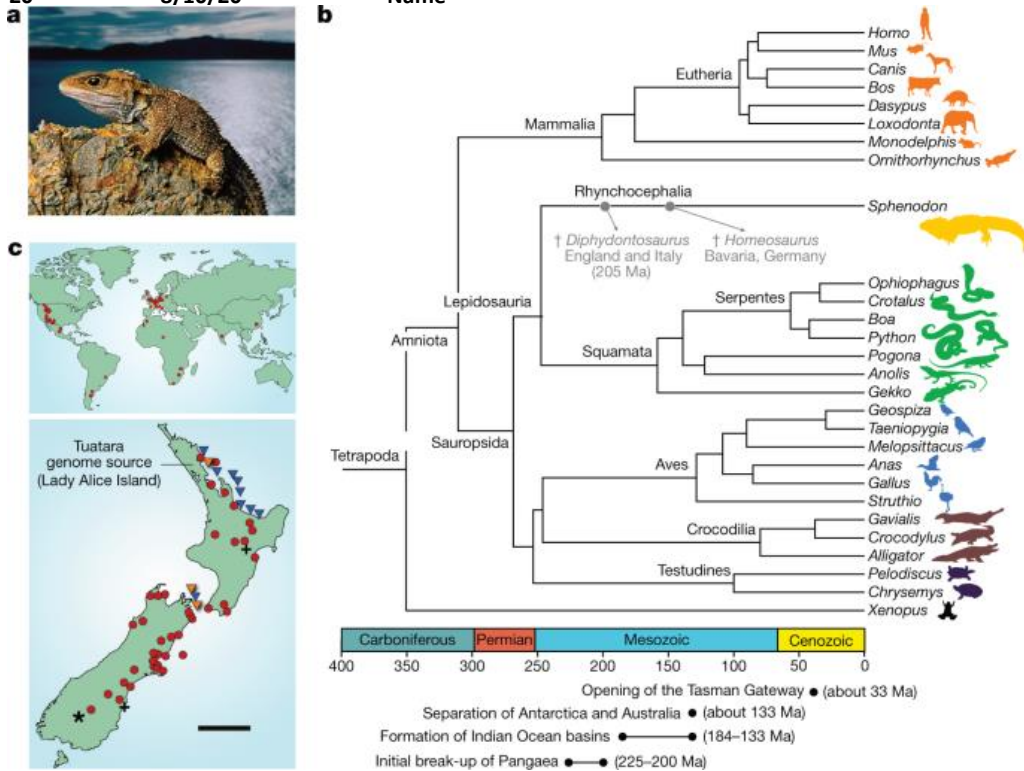
The findings on this remarkable living, single species reptile, which originated in the Triassic period around 250 million years ago and is only found in New Zealand, have been [published in Nature](#).

Professor David Adelson's lab of the University of Adelaide's Department of Molecular and Biomedical Science and Dr Terry Bertozzi of the South Australian Museum carried out key analysis of the tuatara genome that revealed an unusual architecture, half-way between mammal and reptile.

"The tuatara is the last surviving species of a reptile group that roamed the earth with the dinosaurs and remarkably, its genome shares features with those of mammals such as the platypus and echidna," said Professor Adelson.

The key contribution of Professor Adelson's lab and Dr Bertozzi was to demonstrate that some sequences of DNA that move or jump location, referred to as 'jumping genes', found in the tuatara are most similar to those found in platypus while others are more similar to those in lizards.

"The tuatara genome contained about 4% jumping genes that are common in reptiles, about 10% common in monotremes (platypus and echidna) and less than 1% common in placental mammals such as humans," said Professor Adelson.



"This was a highly unusual observation and indicated that the tuatara genome is an odd combination of both mammalian and reptilian components." "The unusual sharing of both monotreme and reptile-like repetitive elements is a clear indication of shared ancestry albeit a long time ago," said Dr Bertozzi.

With no close relatives, the position of tuatara on the tree of life has long been contentious. The research places tuatara firmly in the branch shared with lizards and snakes, but they appear to have split off and been their own species for around 250 million years - an enormous amount of time given primates only originated around 65 million years ago, and hominids, from which humans descend, originated approximately six million years ago.

"It has been a privilege to be part of this project, which has been a true, historic collaboration with local iwi (Māori indigenous tribe)

Ngātiwai. While this is largely fundamental science, I expect it to yield new ways of thinking about our own genome structure that may have relevance to our health," said Professor Adelson.

*a, The tuatara, (S. punctatus) is the sole survivor of the order Rhynchocephalia. b, c, The rhynchocephalians appear to have originated in the early Mesozoic period (about 250–240 million years ago (Ma)) and were common, speciose and globally distributed for much of that era. The geographical range of the rhynchocephalians progressively contracted after the Early Jurassic epoch (about 200–175 Ma); the most recent fossil record outside of New Zealand is from Argentina in the Late Cretaceous epoch (about 70 Ma). c, The last bastions of the rhynchocephalians are 32 islands off the coast of New Zealand, which have recently been augmented by the establishment of about 10 new island or mainland sanctuary populations using translocations. The current global population is estimated to be around 100,000 individuals. Rhynchocephalian and tuatara fossil localities are redrawn and adapted from ref. <sup>1</sup> with permission, and incorporate data from ref. <sup>2</sup>. In the global distribution map (c, top); triangle = Triassic; square = Jurassic; circle = Cretaceous; and diamond = Palaeocene. In the map of the New Zealand distribution (c, bottom); asterisk = Miocene; cross = Pleistocene; circle = Holocene; blue triangle = extant population; and orange triangle = population investigated in this study. Scale bar, 200 km. Photograph credit, F. Lanting.*

<https://bit.ly/3fJYH3c>

## Lava tubes on Mars and the Moon are so wide they can host planetary bases

*Martian and lunar tubes are respectively 100 and 1,000 times wider than those on Earth*

The [international journal Earth-Science Reviews](#) published a paper offering an overview of the lava tubes (pyroducts) on Earth, eventually providing an estimate of the (greater) size of their lunar and Martian counterparts.

This study involved the Universities of Bologna and Padua and its coordinators are Francesco Sauro and Riccardo Pozzobon. Francesco Sauro is a speleologist and head of the ESA programmes

CAVES and PANGAEA, he is also a professor at the Department of Biological, Geological, and Environmental Sciences at the University of Bologna. Riccardo Pozzobon is a planetary geologist at the Department of Geosciences of the University of Padua.

"We can find lava tubes on planet Earth, but also on the subsurface of the Moon and Mars according to the high-resolution pictures of lava tubes' skylights taken by interplanetary probes.

Evidence of lava tubes was often inferred by observing linear cavities and sinuous collapse chains where the galleries cracked", explains Francesco Sauro. "These collapse chains represent ideal

gateways or windows for subsurface exploration. The morphological surface expression of lava tubes on Mars and the Moon is similar to their terrestrial counterpart. Speleologists thoroughly studied lava tubes on Earth in Hawaii, Canary Islands, Australia and Iceland".



*The morphological surface expression of lava tubes on Mars and the Moon is similar to their terrestrial counterpart.* ESA / Luca Ricci

"We measured the size and gathered the morphology of lunar and Martian collapse chains (collapsed lava tubes), using digital terrain models (DTMs), which we obtained through satellite stereoscopic images and laser altimetry taken by interplanetary probes", reminds Riccardo Pozzobon. "We then compared these data to topographic studies about similar collapse chains on the Earth's surface and to laser scans of the inside of lava tubes in Lanzarote and the Galapagos. These data allowed to establish a restriction to the relationship between collapse chains and subsurface cavities that are still intact".

Researchers found that Martian and lunar tubes are respectively 100 and 1,000 times wider than those on Earth, which typically have a diameter of 10 to 30 meters. Lower gravity and its effect on volcanism explain these outstanding dimensions (with total volumes exceeding 1 billion of cubic meters on the Moon).

Riccardo Pozzobon adds: "Tubes as wide as these can be longer than 40 kilometres, making the Moon an extraordinary target for subsurface exploration and potential settlement in the wide protected and stable environments of lava tubes. The latter are so big they can contain Padua's entire city centre".

"What is most important is that, despite the impressive dimension of the lunar tubes, they remain well within the roof stability threshold because of a lower gravitational attraction", explains Matteo Massironi, who is professor of Structural and Planetary Geology at the Department of Geosciences of the University of Padua. "This means that the majority of lava tubes underneath the maria smooth plains are intact. The collapse chains we observed might have been caused by asteroids piercing the tube walls. This is what the collapse chains in Marius Hills seem to suggest. From the latter, we can get access to these huge underground cavities".

Francesco Sauro concludes: "Lava tubes could provide stable shields from cosmic and solar radiation and micrometeorite impacts which are often happening on the surfaces of planetary bodies. Moreover, they have great potential for providing an environment in which temperatures do not vary from day- to night-time. Space agencies are now interested in planetary caves and lava tubes, as they represent a first step towards future explorations of the lunar surface (see also NASA's project Artemis) and towards finding life (past or present) in Mars subsurface".

Researchers also point out how this study opens up to a completely new perspective in planetary exploration, which is increasingly focusing on the subsurface of Mars and the Moon.

"In autumn 2019, ESA called up universities and industries with a campaign seeking ideas for developing technologies for lunar caves exploration. They are specifically looking for systems that would land on the lunar surface to operate missions exploring lunar tubes", clarifies Unibo professor Jo De Waele, who is one of the authors of the study and a speleologist. "Since 2012, in collaboration with some European universities including Bologna and Padua, ESA has been carrying out two training programmes for astronauts focusing on the exploration of underground systems (CAVES) and planetary geology (PANGAEA). These programmes include lava tubes on the island of Lanzarote. So far, 36 astronauts from five space agencies have received training in cave hiking; moreover, six astronauts and four mission and operation specialists have received geological field training".

*The title of this study is "Lava tubes on Earth, Moon and Mars: A review on their size and morphology revealed by comparative planetology" and it was published in the journal Earth-Science Reviews. The authors are: Francesco Sauro, Jo De Waele and Pierluigi De Berardinis (Department of Biological, Geological and Environmental Sciences of the University of Bologna); Riccardo Pozzobon and Matteo Massironi (Department of Geosciences of the University of Padua); Tommaso Santagata (VIGEA - Virtual Geographic Agency in Reggio Emilia).*

<https://bit.ly/2XK0dfj>

## **Electric cooker an easy, efficient way to sanitize N95 masks, study finds**

***One 50-minute, 212 F cooking cycle in a dry electric multicooker decontaminates an N95 respirator without chemicals and without compromising the filtration or fit.***

CHAMPAIGN, Ill.

-- Owners of electric multicookers may be able to add another use to its list of functions, a new study suggests: sanitization of N95 respirator masks.

The University of Illinois, Urbana-Champaign study found that 50 minutes of dry heat in an electric cooker, such as a rice cooker or

Instant Pot, decontaminated N95 respirators inside and out while maintaining their filtration and fit.

This could enable wearers to safely reuse limited supplies of the respirators, originally intended to be one-time-use items.

Led by civil and environmental engineering professors Thanh "Helen" Nguyen and Vishal Verma, the researchers published their findings in the journal [\*Environmental Science and Technology Letters\*](#).

N95 respirator masks are the gold standard of personal protective equipment that protect the wearer against airborne droplets and particles, such as the coronavirus that causes COVID-19.

"A cloth mask or surgical mask protects others from droplets the wearer might expel, but a respirator mask protects the wearer by filtering out smaller particles that might carry the virus," Nguyen said.

High demand during the COVID-19 pandemic has created severe shortages for health care providers and other essential workers, prompting a search for creative approaches to sanitization.

"There are many different ways to sterilize something, but most of them will destroy the filtration or the fit of an N95 respirator," Verma said.

"Any sanitation method would need to decontaminate all surfaces of the respirator, but equally important is maintaining the filtration efficacy and the fit of the respirator to the face of the wearer.

Otherwise, it will not offer the right protection."

The researchers hypothesized that dry heat might be a method to meet all three criteria - decontamination, filtration and fit - without requiring special preparation or leaving any chemical residue.

They also wanted to find a method that would be widely accessible for people at home.

They decided to test an electric cooker, a type of device many people have in their pantries.

They verified that one cooking cycle, which maintains the contents of the cooker at around 100 degrees Celsius or 212 Fahrenheit for 50 minutes, decontaminated the masks, inside and out, from four different classes of virus, including a coronavirus - and did so more effectively than ultraviolet light.

Then, they tested the filtration and fit.

"We built a chamber in my aerosol-testing lab specifically to look at the filtration of the N95 respirators, and measured particles going through it," Verma said.

"The respirators maintained their filtration capacity of more than 95% and kept their fit, still properly seated on the wearer's face, even after 20 cycles of decontamination in the electric cooker."

The researchers created a [video](#) demonstrating the method.

They note that the heat must be dry heat - no water added to the cooker, the temperature should be maintained at 100 degrees Celsius for 50 minutes and a small towel should cover the bottom of the cooker to keep any part of the respirator from coming into direct contact with the heating element.

However, multiple masks can be stacked to fit inside the cooker at the same time, Nguyen said.

The researchers see potential for the electric-cooker method to be useful for health care workers and first responders, especially those in smaller clinics or hospitals that do not have access to large-scale heat sanitization equipment.

In addition, it may be useful for others who may have an N95 respirator at home - for example, from a pre-pandemic home-improvement project - and wish to reuse it, Nguyen said.

*The Environmental Protection Agency and the U.S. Department of Agriculture supported this work.*

*The paper "Dry heat as a decontamination method for N95 respirator reuse" is available [online](#). DOI: 10.1021/acs.estlett.0c00534*

<https://bit.ly/2XH9RiL>

## Denisovans Interbred with Mysterious Archaic Hominin: Study

*Applying a new algorithm to human, Neanderthal, and Denisovan genomes to find signatures of older proposed migration events*

In a [new study](#) published in the journal *PLoS Genetics*, researchers analyzed the genomes of two Neanderthals, a Denisovan, and two African humans; and found that 1% of the [Denisovan genome](#) was introgressed from an unknown archaic hominin ancestor; about 15% of these archaic regions were, in turn, introgressed into modern humans and continue to exist in the genomes of people alive today.

Roughly 50,000 years ago, a group of humans migrated out of Africa and interbred with Neanderthals in Eurasia. But that's not the only time that our ancient human ancestors and their relatives swapped DNA.

The sequencing of genomes from Neanderthals and Denisovans has yielded many new insights into these interbreeding events and into the movement of ancient human populations.

In the new study, Cornell University researchers Melissa Hubisz and Amy Williams and Adam Siepel of Cold Spring Harbor Laboratory developed a new algorithm for analyzing genomes that can identify segments of DNA that came from other species, even if that gene flow occurred thousands of years ago and came from an unknown source.

The scientists used the algorithm, named ARGweaver-D, to look at genomes from two Neanderthals, a Denisovan and two African humans.

They found evidence that 3% of the Neanderthal genome came from ancient humans, and estimate that the interbreeding occurred between 200,000 and 300,000 years ago.

Furthermore, 1% of the Denisovan genome likely came from an unknown and more distant relative, possibly *Homo erectus*, and about 15% of these archaic regions may have been passed down to modern humans who are alive today.

The findings confirm previously reported cases of gene flow between ancient humans and their relatives, and also point to new instances of interbreeding.

“Given the number of these events, genetic exchange was likely whenever two groups overlapped in time and space,” the authors said.



*A portrait of a juvenile female Denisovan based on a skeletal profile reconstructed from ancient DNA methylation maps.* Maayan Harel.

The ARGweaver-D algorithm solves the challenging problem of identifying tiny remnants of gene flow that occurred hundreds of thousands of years ago, when only a handful of ancient genomes are available.

This algorithm may also be useful for studying gene flow in other species where interbreeding occurred, such as in wolves and dogs.

“What I think is exciting about this work is that it demonstrates what you can learn about deep human history by jointly reconstructing the full evolutionary history of a collection of sequences from both modern humans and archaic hominins,” Dr. Siepel said.

“The ARGweaver-D algorithm is able to reach back further in time than any other computational method I’ve seen. It seems to be especially powerful for detecting ancient introgression.”

*M.J. Hubisz et al. 2020. Mapping gene flow between ancient hominins through demography-aware inference of the ancestral recombination graph. PLoS Genet 16 (8): e1008895; doi: 10.1371/journal.pgen.1008895*

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

## Research suggests viability of brain computer to improve function in paralyzed patient

*Fully implantable wireless brain-computer interface designed to improve functional independence in patients with severe paralysis*

FAIRFAX, Va. -- Researchers demonstrated the success of a fully implantable wireless medical device called a stentrode brain-computer interface designed to improve functional independence in patients with severe paralysis. The abstract was presented today at the Society of NeuroInterventional Surgery's (SNIS) 17th Annual Meeting.

The study, Motor Neuroprosthesis Implanted using Cerebral Venography Improves Activities of Daily Living in Severe Paralysis, is the first-in-human examination of the stentrode, an implantable brain-computer interface, conducted at The Royal Melbourne Hospital. The first patient to receive the device was a 75-year-old man with severe paralysis due to amyotrophic lateral sclerosis (ALS), who was totally dependent on his wife for care.

"The implantation procedure combined functional MRI coregistration with angiography to precisely place the stentrode over the motor cortex," said Professor Peter Mitchell, principal investigator and leader of the operative team.

Following implantation of the device, the patient increased independence and could perform essential activities, such as text messaging, online shopping and managing his finances.

"The results in this first human trial show promise that this device may restore voluntary motor function of personal computers and devices for patients with severe paralysis due to brain, spinal cord, peripheral nerve or muscle dysfunction," said Dr. Thomas Oxley, lead author of the study and Associate Professor in the Vascular Bionics Laboratory at the University of Melbourne. "We need to



conduct additional research to confirm our preliminary results and prove the validity of this ground-breaking technology."

The stentrode brain-computer interface translates brain activity associated with attempted movements and digitally converts thoughts into command functions of external devices. The data shows successful control of devices that improve instrumental activities of daily living, which can include texting, emailing, online shopping and banking.

<https://wb.md/31wGS2q>

## **Obesity Epidemic Threatens Effectiveness of Any COVID-19 Vaccine**

*Vaccines engineered to protect the public can be less effective in obese adults than in the general population*

Sarah Varney

For a world crippled by the coronavirus, salvation hinges on a vaccine. But in the United States, where at least 4.6 million people have been infected and nearly 155,000 have died, the promise of that vaccine is hampered by a vexing epidemic that long preceded COVID-19: obesity.

Scientists know that vaccines engineered to protect the public from influenza, hepatitis B, tetanus and rabies can be less effective in obese adults than in the general population, leaving them more vulnerable to infection and illness. There is little reason to believe, obesity researchers say, that COVID-19 vaccines will be any different.

"Will we have a COVID vaccine next year tailored to the obese? No way," said Raz Shaikh, an associate professor of nutrition at the University of North Carolina-Chapel Hill.

"Will it still work in the obese? Our prediction is no."

More than 107 million American adults are obese, and their ability to return safely to work, care for their families and resume daily life

could be curtailed if the coronavirus vaccine delivers weak immunity for them.

In March, still early in the global pandemic, [a little-noticed study from China](#) found that heavier Chinese patients afflicted with COVID-19 were more likely to die than leaner ones, suggesting a perilous future awaited the U.S., whose population is among the heaviest in the world.

And then that future arrived.

As intensive care units in New York, New Jersey and elsewhere filled with patients, the federal Centers for Disease Control and Prevention warned that obese people with a body mass index of 40 or more — known as morbid obesity or about 100 pounds overweight — were among the groups at highest risk of becoming severely ill with COVID-19. About 9% of American adults are in that category.

As weeks passed and a clearer picture of who was being hospitalized came into focus, federal health officials expanded their warning to include people with a [body mass index of 30](#) or more. That vastly expanded the ranks of those considered vulnerable to the most severe cases of infection, [to 42.4% of American adults](#).

Obesity has long been known to be a significant risk factor for death from cardiovascular disease and cancer. But scientists in the emerging field of immunometabolism are finding obesity also interferes with the body's immune response, putting obese people at greater risk of infection from pathogens such as influenza and the novel coronavirus. In the case of influenza, obesity has emerged as a factor making it more difficult to vaccinate adults against infection. The question is whether that will hold true for COVID-19. A healthy immune system turns inflammation on and off as needed, calling on white blood cells and sending out proteins to fight infection. Vaccines harness that inflammatory response. But blood tests show that obese people and people with related metabolic risk

factors such as high blood pressure and elevated blood sugar levels experience a state of chronic mild inflammation; the inflammation turns on and stays on.

Adipose tissue — or fat — in the belly, the liver and other organs is not inert; it contains specialized cells that send out molecules, like the hormone leptin, that scientists suspect induces this chronic state of inflammation. While the exact biological mechanisms are still being investigated, chronic inflammation seems to interfere with the immune response to vaccines, possibly subjecting obese people to preventable illnesses even after vaccination.

An effective vaccine fuels a controlled burn inside the body, searing into cellular memory a mock invasion that never truly happened.

Evidence that obese people have a blunted response to common vaccines was first observed in 1985 when obese hospital employees who received the hepatitis B vaccine showed a significant decline in protection 11 months later that was not observed in non-obese employees. The finding was replicated in a follow-up study that used longer needles to ensure the vaccine was injected into muscle and not fat.

Researchers found similar problems with the hepatitis A vaccine, and other studies have found significant declines in the antibody protection induced by tetanus and rabies vaccines in obese people.

"Obesity is a serious global problem, and the suboptimal vaccine-induced immune responses observed in the obese population cannot be ignored," pleaded researchers from the Mayo Clinic's Vaccine Research Group in a 2015 study published in the journal *Vaccine*.

Vaccines also are known to be less effective in older adults, which is why those 65 and older receive a supercharged annual influenza vaccine that contains far more flu virus antigens to help juice up their immune response.

By contrast, the diminished protection of the obese population — both adults and children — has been largely ignored.

"I'm not entirely sure why vaccine efficacy in this population hasn't been more well reported," said Catherine Andersen, an assistant professor of biology at Fairfield University who studies obesity and metabolic diseases. "It's a missed opportunity for greater public health intervention."

In 2017, scientists at UNC-Chapel Hill provided a critical clue about the limitations of the influenza vaccine. In a [paper published in the International Journal of Obesity](#), they showed for the first time that vaccinated obese adults were twice as likely as adults of a healthy weight to develop influenza or flu-like illness.

Curiously, they found that adults with obesity did produce a protective level of antibodies to the influenza vaccine, but they still responded poorly.

"That was the mystery," said Chad Petit, an influenza virologist at the University of Alabama.

One hypothesis, Petit said, is that obesity may trigger a metabolic dysregulation of T cells, white blood cells critical to the immune response. "It's not insurmountable," said Petit, who is researching COVID-19 in obese patients. "We can design better vaccines that might overcome this discrepancy."

Historically, people with high BMIs often have been excluded from drug trials because they frequently have related chronic conditions that might mask the results. The clinical trials underway to test the safety and efficacy of a coronavirus vaccine do not have a BMI exclusion and will include people with obesity, said Dr. Larry Corey, of the Fred Hutchinson Cancer Research Center, who is overseeing the phase 3 trials sponsored by the National Institutes of Health.

Although trial coordinators are not specifically focused on obesity as a potential complication, Corey said, participants' BMI will be documented and results evaluated.

Dr. Timothy Garvey, an endocrinologist and director of diabetes research at the University of Alabama, was among those who stressed that, despite the lingering questions, it is still safer for obese people to get vaccinated than not.

"The influenza vaccine still works in patients with obesity, but just not as well," Garvey said. "We still want them to get vaccinated."

<https://bit.ly/31CkW5Z>

**A titanate nanowire mask that can eliminate pathogens**  
*EPFL researchers developed a filter "paper" made from titanium oxide nanowires which is capable of trapping pathogens and destroying them with light.*

As part of attempts to curtail the Covid-19 pandemic, paper masks are increasingly being made mandatory. Their relative effectiveness is no longer in question, but their widespread use has a number of drawbacks. These include the environmental impact of disposable masks made from layers of non-woven polypropylene plastic microfibrils. Moreover, they merely trap pathogens instead of destroying them. "In a hospital setting, these masks are placed in special bins and handled appropriately," says László Forró, head of EPFL's Laboratory of Physics of Complex Matter. "However, their use in the wider world - where they are tossed into open waste bins and even left on the street - can turn them into new sources of contamination."

Researchers in Forró's lab are working on a promising solution to this problem: a membrane made of titanium oxide nanowires, similar in appearance to filter paper but with antibacterial and antiviral properties.



*A prototype of a personal protection mask with a titanate filter, which shows efficient to kill bacteria and viruses. Courtesy of Swoxid SA / Endre Horvath*

Their material works by using the photocatalytic properties of titanium dioxide. When exposed to ultraviolet radiation, the fibers convert resident moisture into oxidizing agents such as hydrogen peroxide, which have the ability to destroy pathogens. "Since our filter is exceptionally good at absorbing moisture, it can trap droplets that carry viruses and bacteria," says Forró. "This creates a favorable environment for the oxidation process, which is triggered by light."

The researchers' work [appears today in \*Advanced Functional Materials\*](#), and includes experiments that demonstrate the membrane's ability to destroy E. coli, the reference bacterium in biomedical research, and DNA strands in a matter of seconds. Based on these results, the researchers assert - although this remains to be demonstrated experimentally - that the process would be equally successful on a wide range of viruses, including SARS-CoV-2.

Their article also states that manufacturing such membranes would be feasible on a large scale: the laboratory's equipment alone is capable of producing up to 200 m<sup>2</sup> of filter paper per week, or enough for up to 80,000 masks per month. Moreover, the masks could be sterilized and reused up a thousand times. This would alleviate shortages and substantially reduce the amount of waste created by disposable surgical masks. Finally, the manufacturing process, which involves calcining the titanite nanowires, makes them stable and prevents the risk of nanoparticles being inhaled by the user.

A start-up named Swoxid is already preparing to move the technology out of the lab. "The membranes could also be used in air treatment applications such as ventilation and air conditioning systems as well as in personal protective equipment," says Endre Horváth, the article's lead author and co-founder of Swoxid.

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

## UK-India experts seek to stop antibiotic waste that creates more superbugs

*Waste generated by India's drug manufacturing industry could be damaging environmental bacteria and creating 'superbugs' that are resistant to antibiotics—prompting a UK-India scientific intervention.*

by Tony Moran

British and Indian researchers are joining forces to investigate the impact of waste release on microbial ecosystems—determining how much active antibiotic is released and which other potentially toxic chemicals are contained in the waste that may affect [bacteria](#).

Led by scientists at the University of Birmingham, the SELECTAR project includes experts from the University of Leeds, Aligarh Muslim University, Panjab University, CSIR-Central Drug Research Institute, in Lucknow, Indian Institute of Technology (IIT) Delhi, Jamia Millia Islamia University, in Delhi.

Most of the world's [antibiotics](#) are produced in Indian pharmaceutical factories—either by chemical synthesis or growing vast numbers of the micro-organisms which naturally produce them. Either method generates large quantities of waste, potentially containing active antibiotics and chemicals which may be toxic to bacteria and other cell types.

This waste goes through treatment plants before being released into the environment.

An estimated 58,000 babies die in India every year from superbug infections passed on from their mothers, whilst [drug](#) resistant pathogens cause between 28,000 to 38,000 extra deaths in the European Union every year.

Project lead Professor Alan McNally, from the University of Birmingham, said, "Without antibiotics we are unable to treat the majority of infectious diseases and chronic infections.

Antibiotics prevent the deaths of patients suffering from respiratory diseases such as CF and COPD, and are the corner stone of treatments for cancer and leukemia.

However, manufacturing these wonder drugs generates waste which is treated before being released into the environment, creating an enormous potential issue.

Put simply, the more we expose bacteria to antibiotics the more likely they may be to evolve resistance to the drugs meaning they can't be used to treat infections.

We desperately need to know exactly how much the release of antibiotic production waste leads to increasing antimicrobial resistance, which could ultimately plunge medicine back into the dark ages."

Supported by over £790,000 of funding from UK Research and Innovation's (UKRI) Fund for International Collaboration, the UK-Indian team of scientists will sample environments into which antibiotic production waste is released, and compare them to pristine environments.

The experts will carefully examine the waste to determine exactly how much active antibiotic is released but also which other potentially [toxic chemicals](#) it contains that may affect bacteria.

They will also test the ability of these chemicals to create resistant bacteria, as a consequence of them trying to avoid [chemical](#) killing.

Professor Iqbal Ahmed, of Aligarh Muslim University said, "Release of waste from the manufacturing process creates an enormous potential issue in India and beyond, as the more we expose bacteria to antibiotics the faster they evolve resistance to the drugs meaning they can't be used to treat infections.

Our approach will allow us to determine exactly what effect the [waste](#) has on the microbial ecosystem; does it kill all beneficial bacteria to only leave harmful resistant bacteria alive."

<https://wb.md/2F3Yk6J>

## Consensus Document Reviews Determination of Brain Death

*A group of experts representing various international professional societies has drafted a consensus statement on the determination of brain death or death by neurologic criteria (BD/DNC).*

Erik Greb

The document, a result of the World Brain Death Project, surveys the clinical aspects of this determination, such as clinical testing, apnea testing, and the number of examinations required, as well as its social and legal aspects, including documentation, qualifications for making the determination, and religious attitudes toward BD/DNC.

The recommendations are the minimum criteria for BD/DNC, and countries and professional societies may choose to adopt stricter criteria, the authors note. Seventeen supplements to the consensus statement contain detailed reports on topics the statement examines, including focuses on both adults and children.

"Perhaps the most important points of this project are, first, to show the worldwide acceptance of the concept of BD/DNC and what the minimum requirements are for BD/DNC," corresponding author Gene Sung, MD, MPH, director of the neurocritical care and [stroke](#) division at the University of Southern California in Los Angeles, told *Medscape Medical News*.

Second, "this standard is centered around a clinical determination without the need for other testing," Sung said.

The consensus document and supplements were [published online](#) August 3 in the *Journal of the American Medical Association*.

### Comprehensive Review

A lack of rigor has led to many differences in the determination of BD/DNC, said Sung.

"Some of the variance that is common are the numbers of exams and examiners that are required and whether ancillary tests are required for determination of BD/DNC. In addition, a lot of guidelines and protocols that are in use are not thorough in detailing how to do the examinations and what to do in different circumstances," he noted.

Professional societies such as the World Federation of Intensive and Critical Care recruited experts in BD/DNC to develop recommendations, which were based on relevant articles that they identified during a literature search.

"We wanted to develop a fairly comprehensive document that, along with the 17 supplements, builds a foundation to show how to determine BD/DNC — what the minimum clinical criteria needed are and what to do in special circumstances," Sung said.

Major sections of the statement include recommendations for the minimum clinical standards for the determination of BD/DNC in adults and children.

Determination must begin by establishing that the patient has sustained an irreversible brain injury that resulted in the loss of all brain function, according to the authors. Confounders such as pharmacologic paralysis and the effect of CNS depressant medications should be ruled out.

In addition, clinical evaluation must include an assessment for coma and an evaluation for brainstem areflexia. Among other criteria, the pupils should be fixed and nonresponsive to light, the face should not move in response to noxious cranial stimulation, and the gag and cough reflexes should be absent. Apnea testing is recommended to evaluate the responsiveness of respiratory centers in the medulla.

Although the definition of BD/DNC is the same in children as in adults, less evidence is available for the determination of BD/DNC

in the very young. The authors thus advise a cautious approach to the evaluation of infants and younger children.

Recommendations vary by age and often require serial examinations, including apnea testing, they note.

### **Ancillary Testing**

The consensus statement also reviews ancillary testing, which the authors recommend be required when the minimum clinical examination, including the apnea test, cannot be completed and when it is in the presence of confounding conditions that cannot be resolved.

The authors recommend digital subtraction angiography, radionuclide studies, and transcranial Doppler ultrasonography as ancillary tests based on blood flow in the brain. However, they suggest CT angiography and magnetic resonance angiography not be used.

A lack of guidance makes performing an apnea test in patients receiving extracorporeal membrane oxygenation (ECMO) challenging, according to the authors. Nevertheless, they recommend that the same principles of BD/DNC be applied to adults and children receiving ECMO.

They further recommend a period of preoxygenation before the apnea test, and the document describes in detail the method for administering this test to people receiving ECMO.

Another potentially challenging situation pointed out in the consensus document is the determination of BD/DNC in patients who have been treated with targeted temperature management. Therapeutic hypothermia, particularly if it is preceded or accompanied by sedation, can temporarily impair brainstem reflexes, thus mimicking BD/DNC.

The new document includes a flowchart and step-by-step recommendations as well as suggestions for determining BD/DNC under these circumstances.

Among document limitations acknowledged by the authors is the lack of high-quality data from randomized controlled trials on which to base their recommendations.

In addition, economic, technological, or personnel limitations may reduce the available options for ancillary testing, they add. Also, the recommendations do not incorporate contributions from patients or social or religious groups, although the authors were mindful of their concerns.

To promote the national and international harmonization of BD/DNC criteria, "medical societies and countries can evaluate their own policies in relation to this document and fix any deficiencies," Sung said.

"Many countries do not have any BD/DNC policies and can use the documents from this project to create their own. There may need to be discussions with legal, governmental, religious, and societal leaders to help understand and accept BD/DNC and to help enact policies in different communities," he added.

### **Divergent Definitions**

The determination of death is not simply a scientific question, but also a philosophical, religious, and cultural question, write Robert D. Truog, MD, director of the Harvard Center for Bioethics in Boston, Massachusetts, and colleagues in an [accompanying editorial](#).

Future research should consider cultural differences over these questions, they add.

"Most important is that there be a clear and logical consistency between the definition of death and the tests that are used to diagnose it," Truog told *Medscape Medical News*.

The concept of whole brain death was advanced as an equivalent to biological death, "such that when the brain dies, the body literally disintegrates, just as it does after cardiac arrest," but evidence

indicates that this claim is untrue, Truog said. Current tests also do not diagnose the death of the whole brain, he added.

Another hypothesis is that brainstem death represents the irreversible loss of consciousness and the capacity for spontaneous respiration.

"Instead of focusing on biology, [this definition] focuses on values and is based on the claim that when a person is in a state of irreversible apneic unconsciousness, we may consider them to be dead," said Truog. He and his co-editorialists argue that the concept of whole brain death should be replaced with that of brainstem death.

"This report should be a call for our profession, as well as for federal and state lawmakers, to reform our laws so that they are consistent with our diagnostic criteria," Truog said.

"The most straightforward way of doing this would be to change US law and adopt the British standard of brainstem death, and then refine our testing to make the diagnosis of irreversible apneic unconsciousness as reliable and safe as possible," he concluded.

*The drafting of the consensus statement was not supported by outside funding. Sung has reported no relevant financial relationships. Truog has reported receiving compensation from Sanofi and Covance for participating in data and safety monitoring boards unrelated to the consensus document.*

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<https://bit.ly/2XJ0108>

## Authors' 'invisible' words reveal blueprint for storytelling

*According to new research, small words can be found in a similar pattern across most storylines*

The "invisible" words that shaped Dickens classics also lead audiences through Spielberg dramas. And according to new research, these small words can be found in a similar pattern across most storylines, no matter the length or format.

When telling a story, common but invisible words—a, the, it—are used in certain ways and at certain moments. In a study published in *Science Advances*, researchers from The University of Texas at Austin and Lancaster University in Lancaster, United Kingdom, recorded the use of such words across thousands of fictional and nonfictional stories, mapping a universal blueprint for storytelling.

"We all have an [intuitive sense](#) of what defines a story. Until now, no one has been able to objectively see or measure a story's components," said study co-author and UT Austin psychology researcher Jamie Pennebaker.

In a computer analysis of nearly 40,000 fictional narratives, including novels and movie dialogues, the researchers tracked authors' use of pronouns (she, they), articles (a, the), and other short words, unveiling a consistent "narrative curve:"

1. Staging: Stories begin with a lot of prepositions and articles like "a" and "the." For example, "The house was next to the lake, below a cliff." These words help authors set the scene and convey the most basic information the audience needs to understand concepts and relationships throughout the story.

2. Plot progression: Once the stage is set, authors incorporate more and more interactional language, including auxiliary verbs, adverbs and pronouns. For example, "the house" becomes "her home" or "it."

3. Cognitive tension: As a story progresses toward its climax, cognitive-processing words rise—action-type words, such as "think," "believe," "understand" and "cause," that reflect a person's [thought process](#) while working through a conflict.

This combined linguistic pattern in stories may reflect how humans optimally process information, the researchers said. Prior studies have shown that young children can easily assign names to people and things; ascribing action, however, proves more difficult.

"If we want to connect with an audience, we have to appreciate what information they need, but don't yet have," said study lead author Ryan Boyd, a UT Austin alum and an assistant professor of behavioral analytics at Lancaster University. "At the most fundamental level, humans need a flood of 'logic language' at the beginning of a story to make sense of it, followed by a rising stream of 'action' information to convey the actual plot of the story."

The research team compared the established fictional story structure to more than 30,000 factual texts, including 28,664 New York Times articles, 2,226 TED Talks and 1,580 Supreme Court opinions. Though many shared striking similarities, each genre had unique structures that reflected the different relationships between the authors and their audiences.

"Take TED Talks, for example. They mostly show the same pattern, except at the end where the cognitive tension aspect of stories continues to climb with words like 'think' or 'because,'" said study co-author Kate Blackburn, a post-doctoral research fellow at UT Austin. "This makes perfect sense. The goal of the TED Talk is to inspire, and leave the audience questioning what they have just heard from the speaker. In this sense, we seem to be able to tap into the structure of other forms of storytelling, as if we can identify that story's fingerprint."

**More information:** More details on the team's analysis are available at The Arc of Narrative website: [www.arcofnarrative.com](http://www.arcofnarrative.com)

R.L. Boyd at Lancaster University in Lancaster, UK et al., "The narrative arc: Revealing core narrative structures through text analysis," *Science Advances* (2020).

[advances.sciencemag.org/lookup ... /1126/sciadv.aba2196](https://advances.sciencemag.org/lookup?.../1126/sciadv.aba2196)

<https://bit.ly/2DUg4AQ>

## Some Coronavirus Patients Are Getting Rashes, And It May Signal Underlying Issues

*Patients with severe coronavirus may experience rashes and lesions indicative of underlying blood clots, a new report suggests.*

Anna Medaris Miller, Business Insider

In the paper, published in [JAMA Dermatology](#) Wednesday, researchers described four New York City patients who were intubated with severe coronavirus and had skin complications.

All experienced "acral fixed livedo racemosa", or [discolored, sometimes broken skin](#) on the extremities, and "retiform purpura", or uneven skin lesions caused when red blood cells leak into the skin, according to the researchers from from New York-Presbyterian/Weill Cornell Medical College.

The two complications are "hallmark manifestations" of [skin blood clots](#), they wrote.

Indeed, even though all patients received therapy to help prevent blood clots when they were admitted, all developed clots in their skin and were thought to have pulmonary embolisms, or an artery blockage in the lung.

It's unclear if or when the patients were discharged.

The researchers weren't able to identify exactly when the rashes first appeared and didn't use the type of imaging they'd like in order to spare staff exposure.



JAMA Network

But the findings are a lesson to other healthcare professionals to take skin manifestations as a potential sign of abnormal underlying blood clots, which can [lead to strokes, heart attacks, pulmonary embolisms](#), and other potentially fatal complications.

**The list of coronavirus manifestations continues to grow**

The paper is far from the first to note the coronavirus may cause complications in the skin, with patients reporting "[COVID toes](#)", or [purple, swollen toes that look like they have been frostbitten](#), early on.



In a Facebook group for coronavirus patients and survivors, people have reported fluid-filled blisters, full-body rashes, hives, red and purple spots, patches of skin that burn, chicken-pox like bumps, and more.

In some of these cases, the skin changes may be due to [blood clotting](#) in the skin's small blood vessels.

The skin is just one of the organs that can populate with blood clots, a common denominator among some of the most poorly understood and dangerous coronavirus symptoms.

[In fact, blood clots were found in "almost every organ" of coronavirus patients' autopsies](#), a NYU pathologist said.

**Skin abnormalities are among the growing list of non-respiratory ways the coronavirus seems to manifest**

Doctors are increasingly understanding that the coronavirus is far from "only" a respiratory condition.

While the Centres for Disease Control and Prevention's list of potential symptoms slowly grows, including issues like hair loss and clogged ears, a [recent survey](#) of more than 1,500 patients found hundreds reporting other complications ranging from dizziness to flashes of light in vision to weight gain to nerve sensations.

The wide-ranging ways the disease appears to manifest starkly sets it apart from any other [virus](#) Dr Anthony Fauci has seen in his 40 years, the infectious disease expert [said in a webinar](#) hosted by US News & World Report last week.

"I've never seen anything that has such a broad range of manifestations from a certain percentage of people," he said, noting that up to 40% have no symptoms, many have minor symptoms, some get hospitalized, and some die.

"You go from nothing to death," he said. "It's very, very unusual."

<https://bit.ly/3irv7RL>

## Humans Have Been Making Poison Arrows For Over 70,000 Years, Study Finds

*New evidence now suggests humans have been shooting poison arrows through the last 72,000 years*

[Tessa Koumoundouros](#)

From slaying centaurs to biblical mentions, poison-tipped arrows are a staple of cultural stories in the west. But they've also proved highly effective in reality, so much so that indigenous peoples around the world are still making use of them today, to successfully feed themselves and their families.

The [Kalahari San](#) of southern Africa hunt with small bone- or iron-tipped arrows that may look quite dainty, but when coated with poison, they also prove quite lethal. The hunter-gatherers daub their weapons with larvae entrails of a beetle called [Diamphidia nigroonata](#). The larvae contain a diamphotoxin poison that is capable of [bringing down an adult giraffe](#).

Some of the earliest solid evidence of poison use is traces of the [highly toxic compound ricin](#) on 24,000-year-old wooden applicators, found in South Africa's Border cave. However, archaeologists have long suspected this hunting technique is much older, and new evidence now suggests humans have been shooting poison arrows through the last 72,000 years.

In a new study, archaeologist Marlize Lombard from the University of Johannesburg in South Africa examined the unique properties of known poison arrows, comparing them to those that don't rely on poison, by analysing 128 bone pointed arrows.

Arrows that don't use poison need to deeply pierce the bodies of prey to effectively kill or incapacitate, whereas those laced with poison just need to stab through an animal's skin to access its bloodstream.

Using a measurement called the tip cross-sectional area (the part of the arrowhead important for both cutting into prey hide and the arrow's flight dynamics) allowed Lombard to compare arrows through time. She focused her study on bone-tipped arrows because a lot of previous work looked only at stone-tipped arrows, given more of these have been preserved.

Lombard then assessed 306 Late Stone Age bone-point arrows, for these established properties.

Six of the bone-pointed arrows dated as far back as 72,000-80,000 years, from the Blombos Cave in South Africa. Three of these arrows have properties consistent with poisoned arrowheads.

"One is smaller, which if used as an un-poisoned arrowhead would have been ineffective," Lombard [wrote](#), which would make these the oldest known poison arrows in the world.

The sample size for the oldest arrows is small, and Lombard cautions that such a metric approach to weapons function can only tell us what the weapon had the potential to achieve, rather than the way they were actually used. Other clues are also required to establish probable use.

"When dealing with the human past, numbers alone can seldom reveal the nuances necessary for a deep understanding of techno-behaviours – for that a measure of qualitative assessment and interpretation is required," she [wrote](#).

Another of the bone points found at Klasies River Mouth in South Africa, older than 60,000 years, was found to have micro-cracks, which are consistent with use as an arrow. This arrow was also found to have a black residue that Lombard and other researchers suspect is either poison, glue, or even both.

In more recent times, humans have made use of poisons from a large variety of life, including plants, [poison dart frogs](#) and [even venomous lizards](#). Today, some of these poisons have the potential to be [medically useful](#).

If Lombard's findings hold true, they go to show how this ancient human technology became such an effective tool – one that has well and truly stood the test of time.

*This research was published in the [Journal of Archaeological Science: Reports](#).*