New Scientist

WEEKLY March 18-24, 2017

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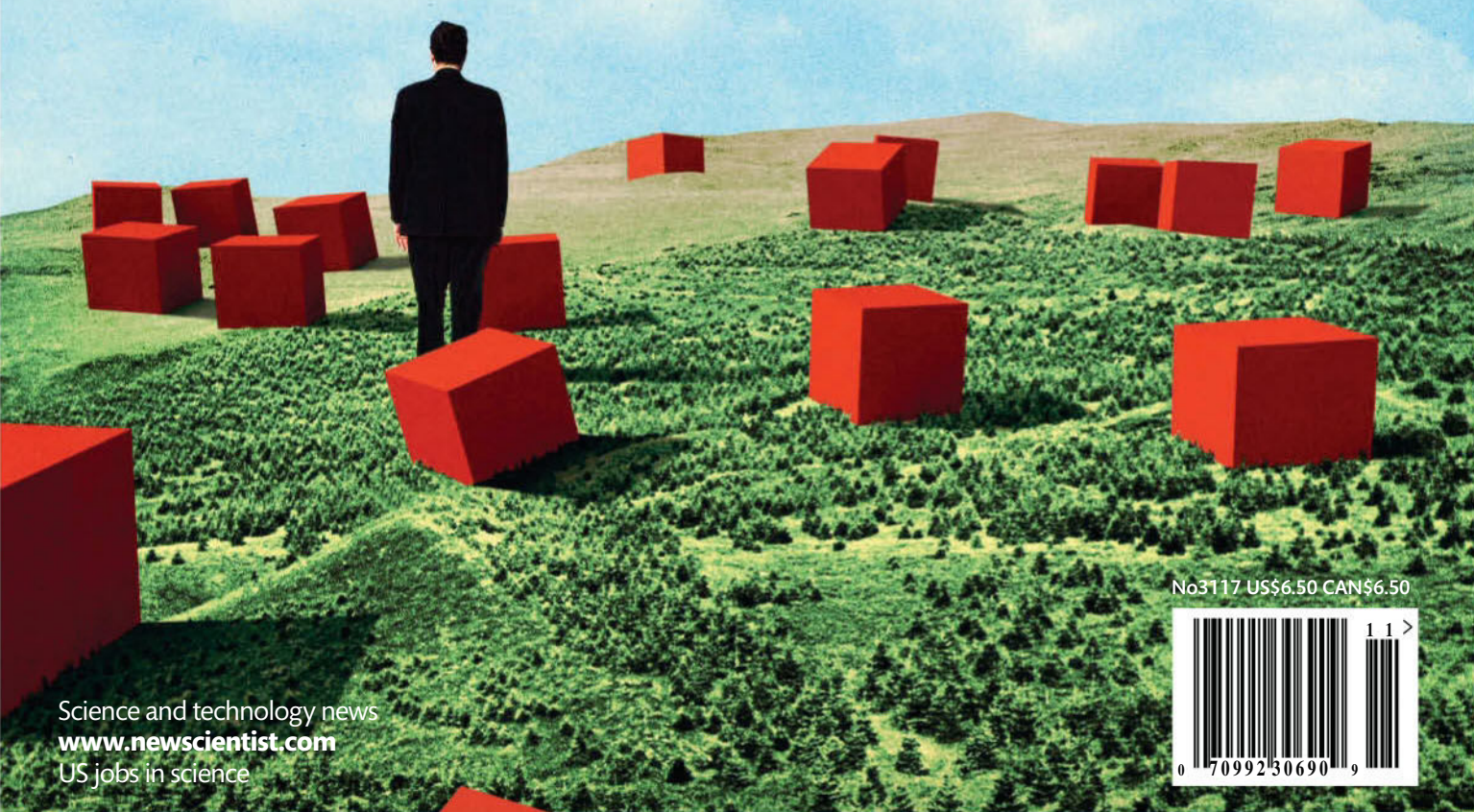

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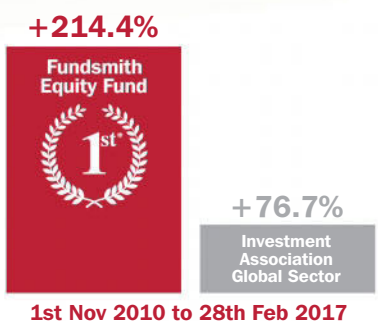

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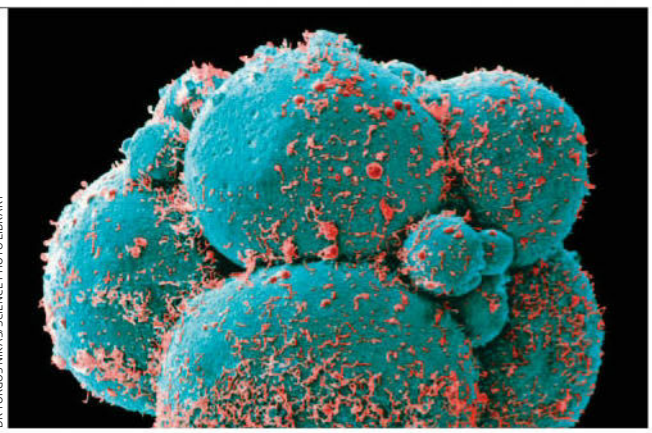

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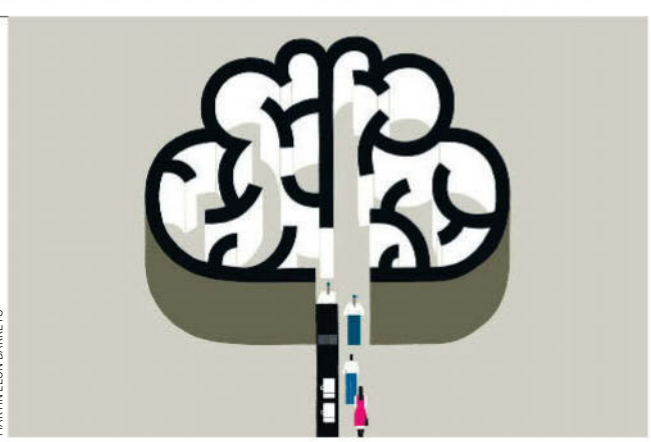

MARTIN LEON BARRETO

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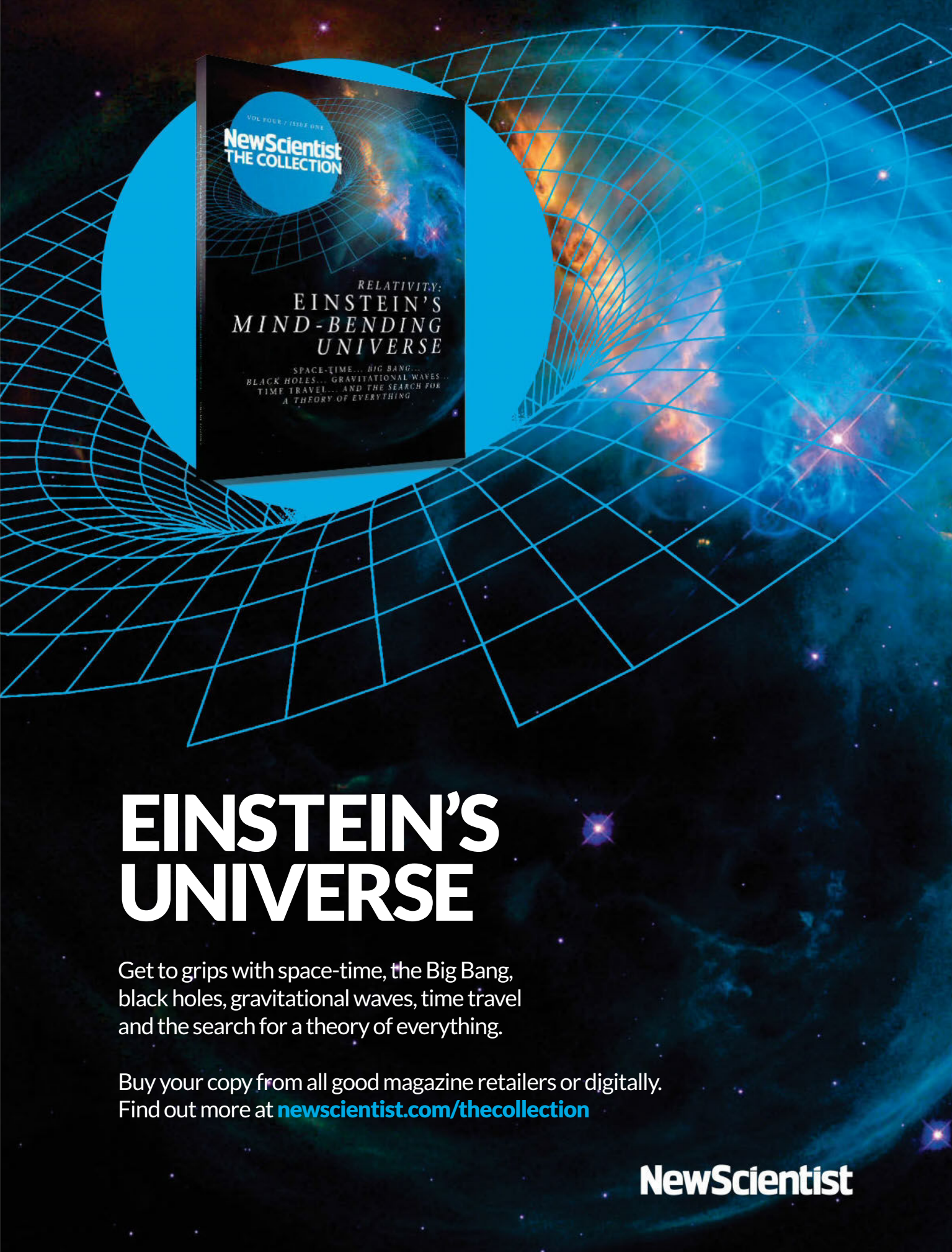

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KRISTOPHER GRUNERT/CORBIS/VCG/GETTY

Biohazards in the shed

Backyard gene editing is a headache for regulators

THE gene-editing revolution continues to gather pace, but it is also throwing us curveballs. One is an unexpected technical hitch (see page 8). Another concerns oversight and ownership: who gets to use it, and for what?

In January, David Ishee, a dog breeder from Mississippi, told the US Food and Drug Administration that he planned to use CRISPR gene editing to fix a mutation that makes Dalmatians prone to kidney disease (see page 38).

The FDA responded by telling Ishee that he could experiment, but not sell or even give away his modified dogs. The law was recently amended so that gene-edited animals require approval before they can be sold.

But the FDA also said it would reconsider if presented with evidence that certain types of gene editing pose "minimal risk". How that will be defined or decided is not clear, but it means we could soon see a cottage industry of gene-edited animals created in biohackers' sheds.

At first glance, that seems an amazingly laissez-faire attitude towards a technology that the US director of national intelligence last year flagged as a threat to national security. Any tinkering with genes raises the spectre of bioterrorism.

In reality, the FDA is walking a fine line, trying to keep abreast of a fast-moving field without stifling innovation. It cannot allow the biohacker tail to wag the CRISPR dog. But the problem requires a more sophisticated response than retrofitting old laws to new problems.

Most biohackers are motivated by curiosity or altruism. But clearly this is not enough of a safeguard. Quite apart from the prospect of bad actors, US intelligence has also warned of "unintentional misuse".

The risk can't be contained by restricting uses of CRISPR, just as nobody can stop people making bombs out of fertiliser. But the technical simplicity that makes CRISPR such an exciting technology also risks creating an unruly beast that the authorities must find a way to tame. ■

A matter of some gravity

ASK most cosmologists and they'll tell you the majority of matter in the universe is invisible. The case for dark matter is considered so overwhelming that all that is left to do is find it. Hence the multibillion-dollar industry to build ever more sensitive particle detectors, which in 30 years have turned up... nothing.

Could the search be an expensive wild goose chase, sustained more by heavy intellectual and financial investment than scientific merit?

It is too soon to write dark matter off. But there is a sense that we are at a tipping point, where heretical ideas about how gravity works are receiving a more considered hearing (see pages 28 and 32). That is a welcome development. The standard model of cosmology has been extremely successful but, like all grand scientific theories, it needs shaking up from time to time to prevent it from solidifying into dogma.

Proposals for how to rethink gravity are sketchy and may lead nowhere. But they represent exactly the sort of exciting new avenues physicists dream of exploring. So why not divert more of the billions we spend on dark matter to the hunt for its nemesis? If dark matter exists, it will withstand the challenge and emerge vindicated. Win, win. ■

TOM WILLIAMS/CQ ROLL CALL

No substitute for Obamacare

Unaffordable care

SIMPLY avoid getting sick. That might be the only strategy left for the 24 million Americans expected to lose healthcare insurance if the Trump administration pushes through a law to replace the existing Affordable Care Act, aka Obamacare (see page 24).

An assessment by the politically impartial Congressional Budget Office (CBO) has concluded that by next year, 14 million more Americans will be uninsured than under Obamacare, rising to 24 million by 2026. When combined with the 28 million who already lack insurance, this will leave a sixth of all Americans – 52 million people – without medical cover.

Trump's allies have dismissed the findings. "If you're looking to the CBO for accuracy, you're looking in the wrong place," White House spokesman Sean Spicer told reporters last week. But many fellow Republicans are also concerned about the impact of the act, not least because some govern states where Obamacare has greatly improved the health of adults on a low income.

Trump's law will scrap enhanced funding for Medicaid, which helps low-income families, and instead awards states a fixed amount per person enrolled, effectively devolving the responsibility – and cost – of citizens' welfare to individual states.

The CBO also concluded that premiums could be 15 to 20 per cent higher, at least until 2020. Insurers will be able to charge older people five times as much as young people, compared with three times at present, as they are more likely to get sick.

No jab, no play

UNVACCINATED children will be barred from attending preschools and daycare centres, the Australian prime minister has announced.

Currently, 93 per cent of Australian children receive the standard childhood vaccinations, including those for measles, mumps and rubella, but the government wants to lift this to 95 per cent. This is the level required to stop the spread of infectious disease and to protect children who are too young to be immunised or cannot be vaccinated for medical reasons.

"The policy will require all children to be fully immunised, unless they have a medical exemption"

Childcare subsidies have been unavailable to the families of unvaccinated children since January 2016, and a version of the new "no jab, no play" policy is already in place in Victoria, New South Wales and Queensland. Other states and territories only exclude unvaccinated children from preschools during infectious disease outbreaks.

The proposed policy is based on Victoria's model, which is the strictest. It requires all children attending childcare to be fully immunised, unless they have a medical exemption, such as a vaccine allergy.

But punitive measures may galvanise the anti-vaccination movement, warns Julie Leask at the University of Sydney. It may also further marginalise children from disadvantaged families, who are less likely to be vaccinated.

Our tangled web

TIM BERNERS-LEE, creator of the World Wide Web, has spoken out about fake news, political advertising and the misuse of personal data. These issues need to be solved, he says, for the web to "fulfil its true potential as a tool which serves all of humanity".

In an open letter to mark the web's 28th birthday on 12 March, Berners-Lee writes that it is too easy for misinformation to spread, because most people get their news from a few social media sites and search engines that prioritise content based on what people are likely to click on.

He also questions the ethics of online political campaigning, which can exploit vast amounts of data to target various audiences. "Targeted advertising allows a campaign to say completely different, possibly conflicting things to different groups," he writes. "Is that democratic?"

He also says that we are losing control of our personal data, which we often divulge to sign up to free services. The Web Foundation, founded by Berners-Lee, plans to work on these issues.

HEMIS / ALAMY STOCK PHOTO

Cheeky alternative to cigarettes

Tobacco endgame

SWEDEN is lighting up the way to a cigarette-free world.

The proportion of Swedish men aged between 30 and 44 who smoke fell to just 5 per cent in 2016, government figures reveal.

This makes the country the first to hit a notional tobacco "endgame" target proposed by global health bodies and some governments, to lower the prevalence of smoking to 5 per cent or below by an agreed date.

Part of the reason for this success is that many Swedes have switched their cigarettes for Snus – teabag-like pouches of tobacco, which slowly release nicotine when tucked under the cheek. Some 18 per cent of Swedish men now use Snus.

"If the Swedish success with Snus was repeated in the UK, it would reduce lung cancer rates by more than 50 per cent," says Gerry Stimson, chairman of European consumer group the New Nicotine Alliance.

Sweden also has far lower rates of oral and pancreatic cancers than anywhere else in Europe.

Space budget

NASA's new budget is all about that space. The agency's orders omit any mention of studying our own world.

On 7 March, the US Congress passed the NASA Transition Authorization Act of 2017, giving NASA a budget of $19.5 billion for the fiscal year ahead and some instructions on how to spend it.

The bill requires NASA to start working on a "human exploration roadmap", including a plan for human missions to Mars in the 2030s.

It encourages NASA to keep working on its Space Launch System rocket and Orion capsule. It also supports a planned new Mars rover to launch in 2020 and eventual robotic exploration of Jupiter's moon Europa, and it requests a strategy for NASA's study of extrasolar planets.

For the first time, Congress also added "the search for life's origin, evolution, distribution, and future in the universe" to NASA's official purpose.

Notably absent from the bill is any information about NASA's Earth science activities, for which the agency requested just over $2 billion this year. The budget now awaits negotiation in the Appropriations Committee and presidential approval.

EPA's carbon denial

THE new chief of the US Environmental Protection Agency has said he does not believe that carbon dioxide is a primary contributor to global warming.

EPA administrator Scott Pruitt told CNBC's Squawk Box that measuring the effect of human activity on the climate is "very challenging" and that "there's tremendous disagreement about the degree of impact" of carbon dioxide and other greenhouse gases. "So, no, I would not agree that [carbon dioxide] is a primary contributor to the global warming that we see," Pruitt said.

Pruitt's view is at odds with mainstream climate science, including by NASA and the US National Oceanic and Atmospheric Administration. The two agencies reported in January that Earth's 2016 temperatures were the warmest on record.

The planet's average surface temperature has risen by about 1.1°C since the late 19th century, "a change driven largely by increased carbon dioxide and other human-made emissions into the atmosphere", the agencies said in January, before Pruitt's comments.

Climate dipole behind drought

AN INDIAN Ocean phenomenon is partly to blame for the severe drought in East Africa, affecting maize and sorghum harvests and sending food prices soaring. A famine has been declared in South Sudan, while Kenya and Somalia have announced national emergencies.

According to the UN Environment Programme, 17 million people in the region now face hunger. "We are currently facing potentially the worst humanitarian disaster the world has known since 1945," says Mike Noyes at the charity ActionAid.

Long-term weather forecasts suggest it won't rain for several months. The drought is partly caused by the Indian Ocean dipole, which is similar to El Niño in the Pacific: sea surface temperatures in the east of the ocean cycle between cold and warm relative to the western ocean.

The dipole was particularly strong in 2016, with warm temperatures in the east creating more moisture in the atmosphere. This cools the air in the east, leading to winds blowing eastward from Africa across the ocean. They push away the moist air that normally brings rain to East Africa from October to December.

The famine is the third to hit the region in 25 years. The last, in 2011, resulted in 260,000 deaths.

The dipole has been getting more pronounced in recent years, and extreme climate events such as drought are projected to become more common as the world warms.

DAN KITWOOD/GETTY IMAGES

Millions face starvation – again

60 SECONDS

Social media spying ban

Facebook is banning the use of its data to spy on its users. The move comes after privacy advocates discovered companies exploiting Facebook data to help police monitor protesters. Facebook and Instagram policies now explicitly state that developers may not use their data to provide surveillance tools.

Breast cancer drug

Up to a fifth of women with breast cancer may benefit from drugs called PARP inhibitors, currently only used for cancers caused by faulty *BRCA* genes. The discovery was made by analysing 560 samples of breast cancer tissue (*Nature Medicine*, doi.org/b37d). PARP inhibitors specifically target cancer cells, so have fewer side effects.

Mangrove die-off

Last year's El Niño didn't just devastate corals in Australia's Great Barrier Reef, it also took a heavy toll on mangroves. Some 7400 hectares were lost along a 1000-kilometre stretch in north-east Australia, probably as a result of drought and heat (*Marine and Freshwater Research*, doi.org/b37h).

Fluorescent frog

A tree frog is the first amphibian known to be naturally fluorescent. A compound in its lymph and skin glands gives it a glow that may make it easier for the frogs to see and communicate with each other at twilight (*PNAS*, doi.org/b364).

Parenthood plus

Why become a parent? For one thing, it may add a few years to your life. A study of 1.4 million people in Sweden found that by the age of 60, women who had children could expect to live 1.5 years longer than childless peers. Fathers could expect nearly 2 years added to their lifespan compared with men with no children (*Journal of Epidemiology and Community Health*, DOI: 10.1136/jech-2016-207857).

Gene editing tried in embryos

First results in viable human embryos reveal CRISPR's biggest problem

Michael Le Page

FIXING faulty DNA to create children free from inherited genetic disease is one step closer. The first published results from efforts to use gene editing in viable human embryos have found the technique works better than we thought – but they have also confirmed a major problem.

Previous tests found that the CRISPR technique wasn't very effective at making genetic changes in human embryos, but the latest work fixed mutations in half of the six embryos it was tried on. "It is encouraging," says Robin Lovell-Badge of the Francis Crick Institute in London, though he warns the numbers are too small to draw strong conclusions.

The idea of changing genes to prevent hereditary diseases has been around for a while, but until recently, we didn't have the tools to do it. Then came CRISPR – a revolutionary method that makes it possible to cheaply and easily edit DNA inside cells.

There have been two big obstacles to using the technique, however. The first is safety. As well as correcting a bad mutation, the CRISPR machinery can also make unwanted changes elsewhere in an embryo's genome, which may lead to cancer. However, this is becoming less of a concern. The technique has been refined to make such "off-target" changes extremely rare, plus there are ways to check embryos for unwanted changes before implanting them into the womb.

The second obstacle has been efficiency – the proportion of embryos fixed. While CRISPR is very efficient at disabling genes, it is less good at repairing faulty ones – a more useful application when it comes to embryos. IVF produces only a few embryos, and even fewer live births, so CRISPR must work most of the time if it is to be used during IVF.

The first two attempts to fix genes in human embryos repaired very few embryos. However, these embryos were genetically abnormal, formed from the fertilisation of an egg with two sperm, and would never have been able to give rise to a child.

Now a team at the Third Affiliated Hospital of Guangzhou Medical University in China has tried the technique in viable embryos (*Molecular Genetics and Genomics*, doi.org/b35x). They used unwanted immature eggs donated by people undergoing IVF, matured them, and fertilised them with sperm from men with genetic diseases.

"Mosaicism would need to be solved before embryos can be gene edited to correct a disease"

Mosaic of cells

The team managed to correct mutations in three out of six embryos, suggesting CRISPR repair is more efficient in viable embryos. "It does look more promising than previous papers," says Fredrik Lanner of the Karolinska Institute in Sweden.

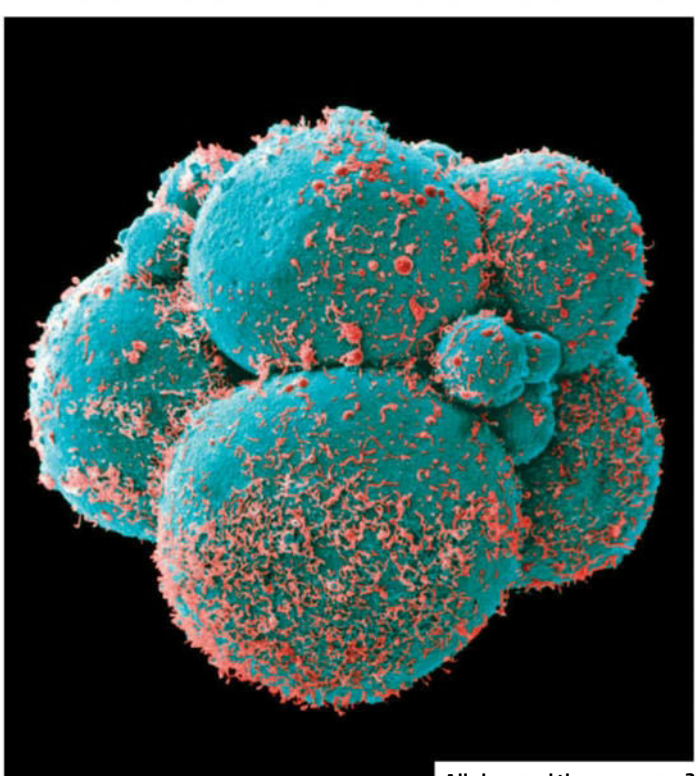
However, the study highlights a further roadblock to using gene editing to create healthy babies. Two of the edited embryos were mosaics – mixtures of edited and unedited cells. The team injected the CRISPR machinery when the embryos were just single cells, but it seems that, in these two embryos, it didn't make repairs until after they had replicated their DNA. So when they divided, some cells inherited unrepaired DNA.

This is a big problem, as it means a child could still develop the disease that gene editing was supposed to prevent. Testing wouldn't be able to tell for sure whether the mutation has been fixed. Similarly, tests to ensure there aren't any unwanted or dangerous mutations elsewhere in the genome wouldn't be reliable. "This would need to be solved before the methods could be used clinically to correct a disease," says Lovell-Badge.

There are possible solutions. Injecting the CRISPR machinery into an embryo as soon as possible after fertilisation and then destroying it a few hours later should ensure repairs only take place before the DNA replicates. This approach has already reduced mosaicism in monkey embryos. An alternative would be to fix the DNA inside stem cells from would-be parents, and then use these to generate egg or sperm cells with repaired DNA.

Even if it does become possible to safely edit our children's DNA, that doesn't necessarily mean we should. A recent report by the US National Academy of Sciences concluded that trials of this kind of gene editing should be allowed only if they meet a number of criteria, the first being "the absence of reasonable alternatives".

Yet almost all inherited diseases can be prevented for most couples by existing forms of screening, such as testing IVF embryos before implantation, without any need for CRISPR. ■



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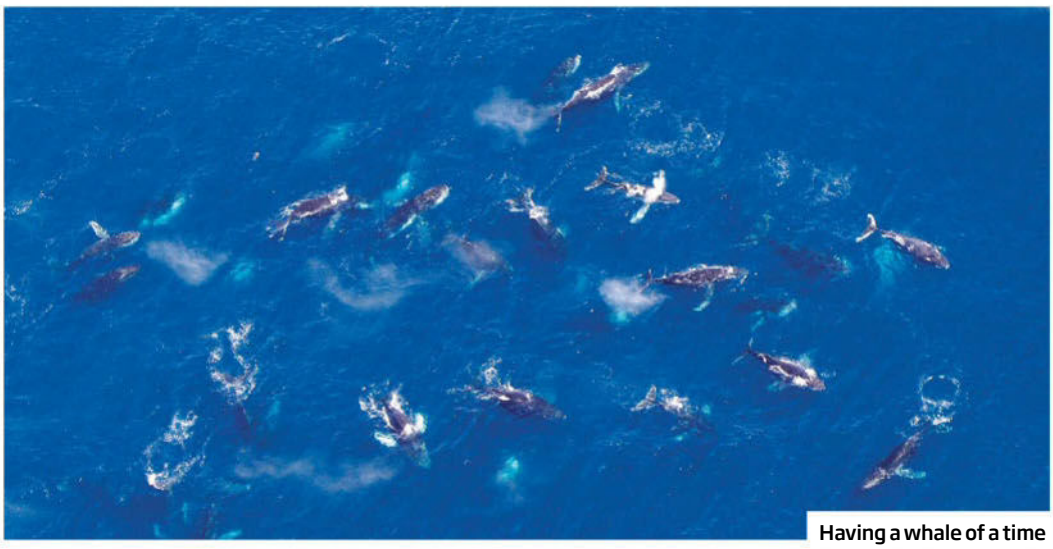
All changed the same way?



KEN FINDLAY

Having a whale of a time

Humpback whales gather in mystery super-groups

IN A baffling change to their behaviour, humpback whales are forming massive groups of up to 200 animals. No one is quite sure why yet, but it could be their long-lost natural behaviour when population levels are high.

Humpbacks aren't normally considered to be terribly social. They are mostly found alone, in pairs, or sometimes in small groups that disband quickly.

But research crews have spotted strange behaviour on three separate cruises in 2011, 2014 and 2015, as well as a handful of public observations from aircraft. These super-groups of up to 200 were spotted feeding intensively off the south-west coast of South Africa, thousands of kilometres further north than their typical feeding grounds in the polar waters of the Antarctic (*PLoS ONE*, doi.org/b33z).

"It's quite unusual to see them in such large groups," says Gísli Vikingsson at the Marine and Freshwater Research Institute in Iceland. Humpbacks mostly spend the summer in the Antarctic where they chow down on krill and build up fat stores. And winters are spent in the warmer waters of tropical and subtropical latitudes, where female whales give birth and nurse new calves.

We don't know what has led to this drastic change in behaviour. "I've never seen anything like this," says Ken Findlay at the Cape Peninsula University of Technology in South Africa, who led the study.

The whales could be shifting their behaviour in response to changes in available prey. Humpbacks occasionally eat things they happen to come across while they travel, but these feeding frenzies seem to be more than just chance opportunity. "These migrating animals might have caught on to the fact that there is food available in this highly productive upwelling system," says Findlay.

Humpbacks were spotted feeding off the south-west Cape coast once before, nearly a century ago, but since then whaling has reduced their numbers by around 90 per cent. "It's possible that the behaviour was occurring but just not where it was visible," says Findlay. "Because there were so few of them, we may not have seen it."

Or, perhaps, as population levels rise humpbacks are reverting to what was once a natural pattern of behaviour.

Another idea is that the whales are being pushed to seek out different prey in new waters due to an unexplained population resurgence in recent years. "For the last few decades, suddenly they seem to have overcome some threshold and have begun to increase very fast," says Vikingsson. He also wonders if these super-groups might help us to understand the species' surprising comeback. "These behaviours could be part of the key to explain their success in recent decades," he says.

To find out more, Findlay hopes to get a better look at what the whales are actually eating in these waters and track their migration paths to and from this new gathering spot. **Mallory Locklear** ■

Helmet vision could avert bike crashes

A HELMET display promises to give bikers eyes in the back of their head.

John Hale, founder of the UK-based start-up Zona, decided to make a better rear-view system for motorcyclists after too many close calls on his own bike. Mirrors didn't show enough detail behind him, and distracted him from looking ahead.

"The inherent problem with motorcycles is that the mirrors just do not work very well," Hale says. "You've got a very small reflected image and, because of the position of the mirrors, mostly you're seeing your elbows."

Zona puts a small backlit display into motorcycle helmets. An adjustable arm lets riders place it just below or above their eye, in their peripheral vision. A rear-facing camera on the back of the bike streams live video via Wi-Fi to a helmet-mounted receiver. The images are processed and stabilised before they reach the display, with accelerometers and gyroscopes to accommodate for the bike's movements.

One glance at the display gives the rider a wide view of the road behind them. Although the screen is just next to the user's face, Zona's optics fool the eye into focusing at a more comfortable distance of around 3 metres. The system also stores the footage so it can be examined after an accident, for example.

Hale says the display, which was unveiled at a motorcycle show last month, has garnered interest from a broad range of motorcyclists, including scooter riders and touring bikers.

"The screen is next to the user's face, but the optics allow the eye to focus at a comfortable distance"

A device that fits into any helmet is likely to appeal to motorcyclists more than previous attempts at smart helmets, says Alex Stedmon at Coventry University, UK. But he says Zona-using bikers should still check their mirrors and do the "lifesaver" check, turning their heads to check blind spots. "I'd see this as perhaps an additional resource," he says. Victoria Turk ■

We'll never get to absolute zero

Leah Crane

IT'S an absolute. Mathematics has put speed limits on cooling, finally proving a century-old law – that unless you have infinite time and resources, you can't get to the absolute zero of temperature.

In 1906, German chemist Walther Nernst formulated the heat theorem, which states that as a perfect crystal approaches the absolute zero point of 0 kelvin (–273.15°C), the system's entropy also goes to zero. This work earned him the 1920 Nobel prize in chemistry.

The rule was controversial, with heavyweights like Albert Einstein and Max Planck debating it and introducing their own formulations. In 1912, Nernst defended his version by adding another clause, the unattainability principle, which states that absolute zero is physically unreachable.

Taken together, these two rules make up the modern third law of thermodynamics.

But because earlier arguments focused only on specific mechanisms or were crippled by questionable assumptions, some physicists have always remained unconvinced of its validity.

Now Jonathan Oppenheim and Lluís Masanes at University College London have mathematically derived the unattainability principle and placed limits on how fast a system can cool, creating a general proof of the third law.

"In computer science, people ask this question all the time: how long does it take to perform a computation?" says Oppenheim. "Just as a computing machine performs a computation, a cooling machine cools a system." So, he and Masanes asked how long it takes to get cold.

Cooling can be thought of as a series of steps: heat is removed from the system and dumped into the surrounding environment again and again, and each time the system gets colder. How cold depends on how much work can be done to remove the heat and the size of the reservoir for dumping it.

By applying mathematical techniques from quantum information theory, they proved that no real system will ever reach 0 kelvin: it would take an infinite number of steps (*Nature Communications*, doi.org/b374).

Getting close to absolute zero is possible, though, and Masanes and Oppenheim quantified the steps of cooling, setting speed limits for how cold a given system can get in finite time.

As quantum computing advances, the need to quantify cooling becomes more pressing. To store data, the particles in a quantum computer are put into particular energy states; extra energy and the warmth that it brings push particles out of those states, degrading or destroying the stored data.

"It's not just removing the energy of the system," Masanes says. "It's also about removing uncertainty."

The limits set by this research are far less stringent than the technological limitations for now: nobody has reached temperatures or cooling speeds near what Masanes and Oppenheim found are the bounds. As technology improves, they hope that these bounds will start to become practically relevant.

"The work is important – the third law is one of the fundamental issues of contemporary physics," says Ronnie Kosloff at the Hebrew University of Jerusalem. "It relates thermodynamics, quantum mechanics, information theory – it's a meeting point of many things." ■

BERND SCHUMACHER/ PLAIN PICTURE

It's unattainable

Tiny particles spread effect of brain injuries

MICROPARTICLES secreted after head injuries could help explain how inflammation spreads and ultimately boosts the risk of dementia.

Head injuries are increasingly being linked to cognitive problems and degenerative brain disease in later life, and inflammation is a prime suspect. Mysterious particles a micrometre in diameter have previously been found in the spinal fluid of people with traumatic brain injuries, but their function was unclear.

To investigate, Alan Faden at the University of Maryland School of Medicine in Baltimore and his colleagues injured the brains of sedated mice. They saw that activated immune cells called microglia secrete such microparticles in response to brain injury. The particles also activated resting microglia, enabling them to trigger inflammation beyond the site of damage.

Indeed, when Faden injected microparticles from injured animals into the brains of uninjured animals, they caused inflammation. This didn't happen when microparticles from uninjured mice were injected (*Journal of Neuroinflammation*, DOI: 10.1186/s12974-017-0819).

The particles have receptors that latch onto cells and are packed with inflammatory chemicals, plus RNA fragments that can switch genes on or off. "The effect of these particles in driving inflammation even in animals without traumatic brain injury is convincing," says Marie-Ève Tremblay of Laval University in Quebec, Canada, who last year discovered microglia that could be linked with dementia.

"The particles activate resting microglia, enabling them to trigger further inflammation"

One of Faden's co-authors, Stephen Thom, has already developed an agent that might work against these microparticles. Called PEG-TB, it neutralises the ability of the particles to trigger inflammation by causing them to fall apart. Experiments in pigs have shown that PEG-TB lessens the inflammation resulting from traumatic brain injuries. Andy Coghlan ■

Virtual world's end probes human nature

EVER wondered how you would behave at the end of the world? Players' actions in a video game that was set up to terminate at a certain time could provide some clues.

The players were taking part in a beta test of the online role-playing game ArcheAge. The whole thing was to last about 11 weeks, so researchers could study their actions as the end drew near. Players knew that once the test was over, that would be it for their character: all of their progress would be deleted.

Made by XL Games in South Korea, ArcheAge lends itself well to behavioural analysis because it is a wide-open, "sandbox" game. Players have the freedom to build houses, throw parties, learn a trade, spend money, advance through the ranks of complex guilds, even kill people.

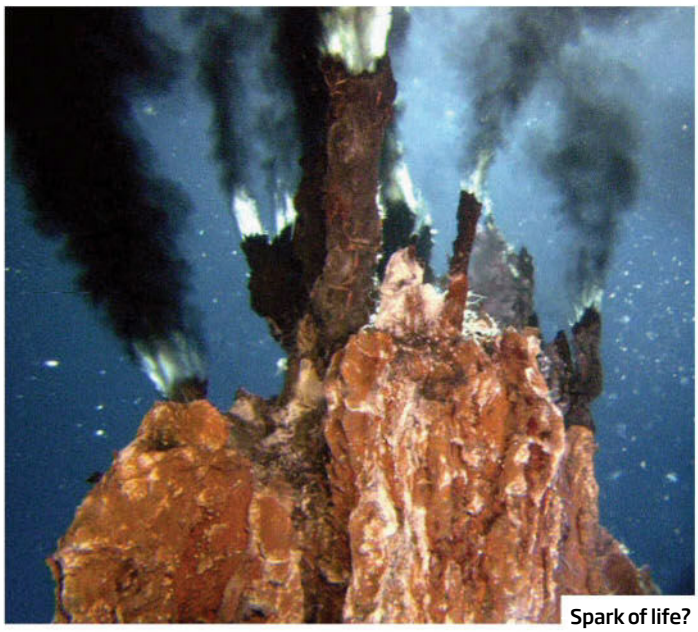
When the team analysed 270 million records of players' actions, they found that apart from a few outliers who became more murderous, most players didn't resort to killing sprees or antisocial behaviour as the game progressed. In fact, they tended to become more social, and abandoned trying to advance their characters or complete quests (arxiv.org/abs/1703.01500).

"People don't really go off the deep end – they just stop worrying about the future," says team member Jeremy Blackburn at Telefonica Research in Barcelona, Spain.

That makes sense, says Dmitri Williams at the University of Southern California in Los Angeles. "There's a big difference between planting an apple tree, even when you know you're going to die, because then your kids can enjoy it, to 'the world is going to end and there will be no apple tree for anybody'," he says.

Behaviour in virtual worlds doesn't always mirror real life, but the video game environment offers one big advantage: researchers have no real-life Armageddon to study.

Anna Nowogrodzki ■



AF ARCHIVE / ALAMY STOCK PHOTO

Spark of life?

Metabolic reactions may have existed before life

WE MAY be a step closer to solving the mystery of how life began some 4 billion years ago.

A primeval version of the metabolic processes that all life uses to survive could have arisen spontaneously on Earth in the absence of enzymes, according to a new study. This suggests that key parts of metabolism are older than life itself and could have emerged easily on early Earth.

Metabolism describes the complex network of chemical reactions that enable organisms to generate energy and the molecules they need to survive, grow and reproduce. The Krebs cycle is at the heart of this network. It is a circular chain of reactions that generates precursors of amino acids and lipids used to build proteins and membranes, and molecules that help the cell to produce its energy.

But how such a complex cycle developed in the first place has remained a mystery.

Now Markus Ralser at the Francis Crick Institute in London and his colleagues have demonstrated that the Krebs cycle could have existed from the outset. The early life forms then simply adopted it, developing enzymes to make it more efficient, the team thinks.

Modern enzymes that catalyse the Krebs cycle all use very different mechanisms to do so. For the process to have occurred spontaneously, a simple, inorganic molecule must have existed naturally in Earth's early oceans that could catalyse such a diverse set of reactions.

"The simplicity of it is super exciting because it gives you a plausible sense of how life may have started"

Ralser's team looked at chemical intermediates of the Krebs cycle, which we know were around 4 billion years ago. Some have been identified on meteorites and others in laboratory recreations of Earth's early oceans. The team then exposed these chemicals to peroxydisulphate, a source of highly reactive agents called sulphate radicals, which would have been present in early ocean sediments. This triggered a sequence of 24 chemical reactions that were very similar to those seen in the Krebs cycle today (*Nature Ecology & Evolution*: DOI: 10.1038/s41559-017-0083).

This backs Ralser's earlier findings that metal ions can drive two other parts of cells' metabolism. "The most surprising thing is that, again, a single molecule acts as the catalyst for all of the reactions we discovered," says Ralser. "The simplicity of it is super-exciting because it gives you a plausible feeling about how it could have all started."

Sulphate radicals would been found in abundance near hydrothermal vents, where life might have started. Ralser thinks that these hardwired chemical reactions provided a template upon which the evolutionary machinery could build once it came into being.

"With the metabolic pathway alone, you have a very good starting point for life, but it is not life, just a chemical-reaction network," says Ralser. "You also need things like membranes to contain the reactions, and the genetic machinery that enables inheritance."

The big challenge now is to understand how these elements were brought together and made to work in a living cell for the first time, he says.

Not everyone agrees with the team's conclusions, though.

"This is a neat paper and the findings are striking and careful," says Nick Lane, an evolutionary biochemist at University College London. But he says the team only shows the oxidative Krebs cycle, which is not as ancient as the reductive cycle, which fixes CO_2 using hydrogen and is still found in some ancient bacteria. Until they show that, says Lane, it's not certain that metabolism did precede life.

Linda Geddes ■

How to manipulate the mind's eye

Alice Klein

JEAN-PIERRE MOONEY knows the colour of his girlfriend's hair and eyes, but he just can't picture her face. "If she went missing and the police asked me to draw a sketch, I wouldn't know how," he says. "That's like sorcery for me."

The 34-year-old from Brisbane, Australia, is unable to "see" anything in his mind's eye. The condition, aphantasia, affects about 2 per cent of people but remains largely mysterious.

Now there is a glimmer of progress in understanding it, with the discovery that electrically stimulating the brain can alter the strength of our mental imagery. Although it's not yet clear if this can benefit those with aphantasia, it could potentially be used to boost memory, navigational ability and creativity in people who don't have the condition. It might also tone down mental imagery in those haunted by dark, intrusive thoughts.

Joel Pearson at the University of New South Wales in Sydney and his colleagues wanted to understand whether the ability to form mental images was completely absent in people with aphantasia, or if the conscious brain was somehow blocking access to those images.

The team exploited a phenomenon called binocular rivalry, in which people perceive only one image despite their left and right eye being shown different images at the same time. The effect can be usually influenced by priming: for instance, asking someone to imagine the colour red before showing a red image to one eye and a green image to the other.

Pearson found that people who don't have aphantasia perceived the primed red image 60 to 95 per cent of the time. But when they tested 15 people with aphantasia, they perceived the red image about half the time – no more than predicted by chance. This hints that those with aphantasia are unable to form mental images in the first place, he says (osf.io/preprints/psyarxiv/pdjb9).

Next, they scanned the brains of 31 people without aphantasia. This revealed that those who self-reported greater visual imagery had higher activity in the prefrontal cortex, which exerts control over other brain areas, and lower activity in the visual cortex.

Pearson thinks the visual cortex is a little like a sketch pad: "If there's too much activity and noise, it's like sketching on a dirty piece of paper: it's hard to see the drawing." Conversely, because the prefrontal cortex controls the visual cortex, higher activity in the former may enhance visualisation, he says.

"The technique could potentially be used to boost memory, navigation skills and creativity"

Pearson's team also showed that it is possible to alter the strength of people's mental imagery by electrically stimulating the visual and prefrontal cortices using a technique called transcranial direct current stimulation, or tDCS (bioRxiv, doi.org/b36b).

Pearson is now planning to test whether tDCS can help people with aphantasia to see with their mind's eye. He also plans to explore whether manipulating it has wider benefits, such as boosting memory or reducing visual hallucinations in people with psychotic disorders.

However, Sergio Della Sala at the University of Edinburgh, UK, warns that clinical benefits may take time to emerge. "It may be hard to target a single cognitive process or modify an individual behaviour by applying a generalised current [using tDCS]," he says. ■

IMAGE SOURCE/GETTY

Drawing a blank

Dogs deceive humans to get what they want

OUR best friends can be sneaky and manipulative when they want to maximise the number of tasty treats they get to eat.

Marianne Heberlein at the University of Zurich in Switzerland wanted to test dogs' ability to use deception to get what they want from humans. The idea came to her as she watched her own dogs – one of them occasionally pretends to see something interesting in the backyard to trick the other into giving up the prime sleeping spot. "This sort of thing happens quite often, but it is not well studied," she says.

To see if dogs would deceive humans too, Heberlein's team paired various pooches with two human partners – one who always gave the dog treats and another who always withheld them.

After the dogs learned which of the pair would give them the food, the pets were given the opportunity to lead each person to one of three boxes containing either a juicy sausage, a less-appetising dry dog biscuit or nothing. The humans didn't know what was in the boxes, and the dogs got a chance after the trial to get a treat from a box. This gave them an incentive to deceive the humans who withheld the treats by taking them to the empty box before claiming the tasty treat. And that's just what they did over the two days of testing (*Animal Cognition*, doi.org/b35v).

Heberlein was surprised how quickly some dogs figured out the optimal behaviour to get most of their preferred treats. A few of them led the selfish human to the empty box from the very first trial, and always managed to get the most treats.

The question now is whether dogs are flexible enough to deceive us in other contexts, says Daphna Buchsbaum, who studies dog cognition at the University of Toronto, Canada. "If they can, I'd say it was evidence of very sophisticated social reasoning," she says. Brian Owens ■

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Robot expresses pain like a patient

Timothy Revell

CAN you tell when someone is unwell just by studying their face? Understanding expressions can help doctors improve their diagnoses, but it is a difficult skill to practise. Step forward a robot that can express pain.

Many doctors already use robotic patient simulators in their training to practise procedures and test their diagnostic abilities. "These robots can bleed, breathe and react to medication," says Laurel Riek at the University of California, San Diego. "They are incredible, but there is a major design flaw – their face."

Patient simulators usually have static faces, often with an open mouth so doctors can practise checking airways. Unlike a real patient, they show no emotion.

In an attempt at emulating realistic patient feedback, Riek and her team have given a robotic face the ability to simulate expressions of pain, disgust and anger. Alongside the robot, the team also created a virtual avatar as an alternative tool. "Interpreting a patient's facial expressions can help determine if they are having a stroke, are in pain or are having a reaction to medication, so doctors need to be able to do this from day one," Riek says.

To make the robot and avatar, the researchers collected videos of people expressing pain, disgust and anger, and used face-tracking software to convert their expressions into a series of moving points. They then mapped these onto the robot and avatar faces. The robot used was Hanson Robotics' Philip K. Dick, a humanoid modelled on the science fiction writer that has realistic rubber skin and can move its facial features.

To test how well people could perceive emotions from the simulated facial expressions, videos of the robot and avatar were shown to 102 volunteers, who had to judge which emotion matched which expression. Half the volunteers were clinicians, such as doctors, nurses and pharmacists, and half had no medical background.

The clinicians turned out to be less accurate than the non-clinicians at recognising both pain and anger. In the starkest difference, the clinicians correctly identified pain expressed by the virtual avatar only 54 per cent of the time, compared with 83 per cent for the non-clinicians.

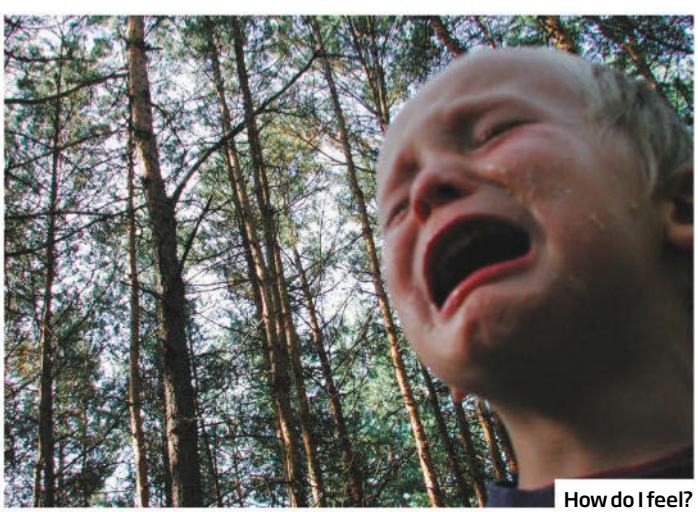
This follows previous research that suggests doctors are worse at interpreting pain in humans than laypeople and tend to underestimate the severity. This could partly be a result of medical training decreasing levels of empathy.

The researchers think the expressive robots and avatars could help train doctors to better interpret pain. They presented their research last week at the Conference on Human-Robot Interaction in Vienna, Austria.

"This work could be used very soon to better train our medical professionals and improve patient outcomes," says Priscilla Briggs, a software engineer at Google with a background in human-robot interaction. But further work will be required to show that a robot's expressions can improve clinicians' performance, she says.

Later this year, Riek and her colleagues plan to carry out a trial of the technology. Student doctors at the University of California, San Diego, will use the robot in simulated scenarios such as a person recovering from a stroke.

"We will explore how realism and expressiveness affects student learning outcomes, their sense of immersion and how well they assess patient cues in order to accurately and safely intervene," says Riek. ■



KATHRIN SCHMIEDER/PLAINPICTURE

How do I feel?

Life could hop between newly found planets

THE newly discovered planets of the TRAPPIST-1 system could be a playground for rock-riding microbes.

Three of the small, dim star's seven planets orbit firmly within its habitable zone – the region with the right temperature to retain liquid water, thought to be a requisite for life. They keep close to each other, only a few times the distance between Earth and the moon, looming large in one another's sky.

At such short distances, when a meteorite hits the surface of one of the planets, the resulting debris could make its way between all three.

If bacteria or other forms of life stowed away on a piece of debris, they could hitch-hike between worlds in a process called panspermia. Some scientists believe life on Earth may have started this way, as microbial stowaways from Mars.

Now, Manasvi Lingam and Avi Loeb at Harvard University have determined that this sort of transfer is 1000 times more likely to occur between the TRAPPIST-1 planets than between Earth and Mars.

"Because these distances are so close, a lot more different kinds of species, microbial or otherwise, could migrate from one planet to another," says Lingam.

This means that if there is life on one planet, there is probably life on all three. They compared the TRAPPIST system to a series of islands, using mathematical methods from island ecology to describe migration and extinction between them (arxiv.org/abs/1703.00878).

"Different kinds of species, microbial or otherwise, could migrate from one planet to another"

But some biologists reject this metaphor. "This work is interesting, but no, planets are not islands, even if they are close," says Valeria Souza at the National Autonomous University of Mexico.

Even on Earth, she says, it is difficult for species to migrate between islands, and evolution would take them all down a different route once they arrived. Leah Crane ■

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Artificial life comes a huge step closer

Bob Holmes

WHERE we had one, we now have six. The team that built the first synthetic yeast chromosome has added five more chromosomes to their repertoire, totalling roughly a third of the organism's genome. It's a dramatic scaling-up of our capabilities, opening the door to large-scale genomic engineering.

It was in 2014 that Jef Boeke, now at New York University Langone Medical Center in New York City, and his colleagues constructed a single yeast chromosome. They then replaced one of a living yeast cell's natural chromosomes with it – the first time this had been done in more complex cells with a nucleus.

Boeke's team has since edited the yeast's entire genome – streamlining it and adding molecular labels to ease future work – before farming out the synthesis of the 16 rewritten chromosomes to an international consortium of geneticists and yeast biologists.

Each of the additional five chromosomes announced last week was assembled from pieces of 30,000 to 60,000 DNA letters. This allowed researchers to "debug" each section as they went, correcting for errors that had crept in during the editing process.

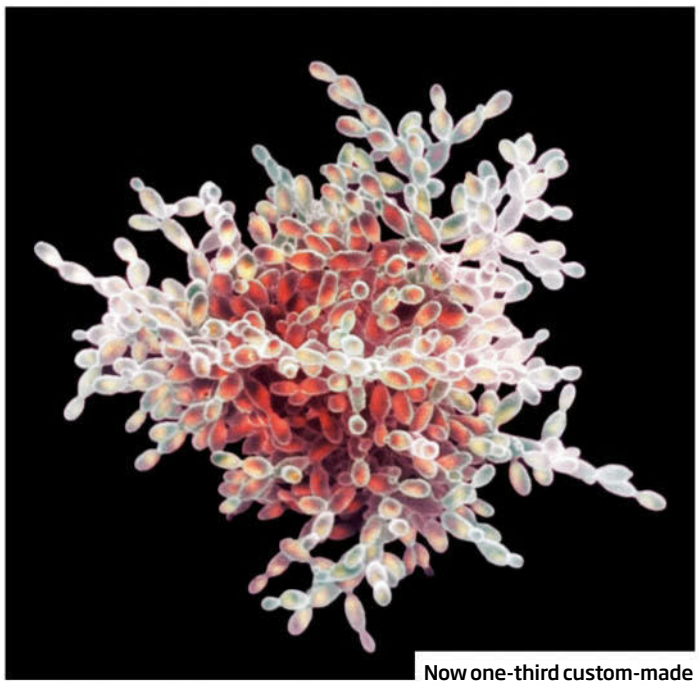
As a result of this careful debugging, yeast cells with the synthetic chromosomes grow just as quickly in the lab as normal, wild yeast, despite the wholesale alterations (*Science*, DOI: 10.1126/science.aaf4557).

Other researchers say the health of the modified yeast is remarkable. "It now sets the stage for the ultimate, which is putting all 16 synthetic chromosomes into one cell," says Dan Gibson at Synthetic Genomics, a biotech company in La Jolla, California. "I now have more confidence that they'll be able to achieve that."

If and when they do, researchers hope to learn a huge amount. "If you take a bicycle and break it down to its smallest parts in your basement, and reassemble it again, you know a hell of a lot more about your bicycle than you did before," says Boeke. Similarly, taking apart a genome and rebuilding it should yield new understanding of life and its processes.

A synthetic genome will also give bioengineers unprecedented control over yeast metabolism, which we already exploit to make chemicals such as drugs and perfumes. That would allow them to expand the range of molecules yeast can produce.

Researchers could also try inserting human versions of genes into yeasts, something they already do for a few genes at once. Synthetic chromosomes would allow them to go a great deal further – a big plus when it comes to testing new drugs. But the biggest payoffs may be ones that no one foresees, says geneticist George Church at Harvard University. ■



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Gender bias in films laid bare by software

MACHINE learning is taking on Hollywood's gender bias. Technology that automatically detects how long men and women appear on screen reveals that in recent popular films, men have had almost twice as much screen time as women.

The software uses algorithms for face and voice recognition that have been trained on annotated video to identify whether a character is male or female, and can measure how long they are on screen to a fraction of a second. It was developed by Shri Narayanan at the University of Southern California, Los Angeles, in partnership with the Geena Davis Institute on Gender in Media and Google.org, the search engine's charitable arm.

In an analysis of the 100 highest-grossing live-action films from each of the past three years, the software found that women appear on average for just 36 per cent of the total time that characters are on screen. Oscar-winning films are even less representative, with women in those getting just 32 per cent of screen time and 27 per cent of speaking time.

Narayanan's program can analyse a feature film in less than 15 minutes and also distinguish when one person is on screen but someone else is talking. "Often when women are speaking, it's actually the men that are shown on screen," he says.

Of the 300 films, the only genre in which women appeared for longer than men was horror, with 53 per cent of screen time. In dramas, women only appeared for an average of a third of the film, and in crime films less than a quarter. The system did not account for non-binary characters.

Calculating how often women appear on screen is one way to measure gender bias, says Ginette Vincendeau at King's College London, but it's also important to consider who they are portraying and what their characters are talking about. "It's not just a question of quantity," she says.

"Of the 300 films, the only genre in which women appeared for longer than men was horror"

Narayanan is also using machine-learning algorithms to analyse scripts, which could reveal further insights about the roles of female characters, such as the content of their dialogue.

But the real agents of change in the film world are the production companies, says Vincendeau, and it's up to them to make sure that more women are placed in leading roles – both behind and in front of the camera. Matt Reynolds ■

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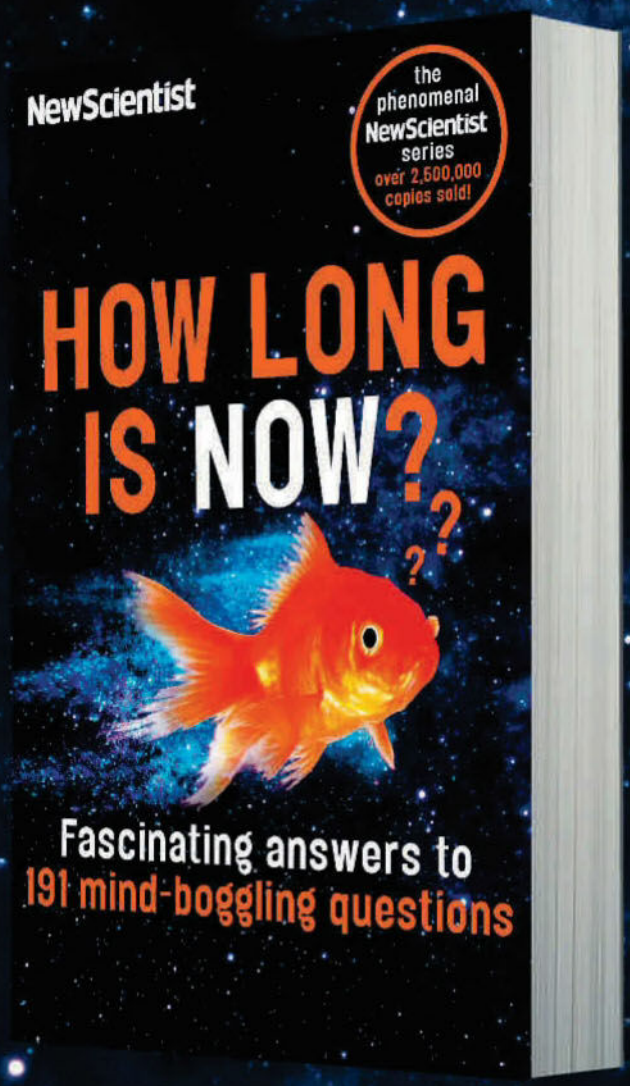

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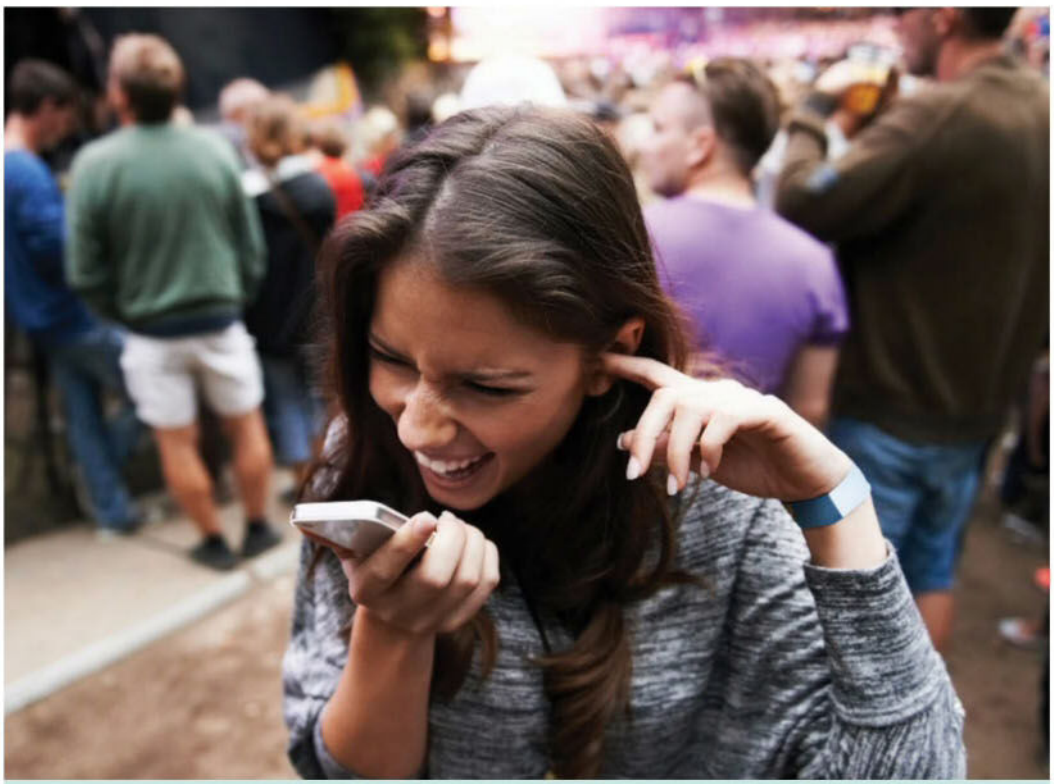


PEOPLEIMAGES/GETTY

Brain fills gaps in hearing without you realising

WHEN it's noisy, you can still keep track of a conversation amid the sound of revving motorcycles or a raucous cocktail party. Your brain helps by predicting what's coming next and filling in any blanks.

We have known since the 1970s that the brain can "fill in" inaudible sections of speech, but we don't know exactly how. To investigate, Matthew Leonard at the University of California, San Francisco, and his team played volunteers words that were partially obscured or inaudible to see how their brains responded.

The experiment involved people who already had electrodes implanted into their brains to monitor epilepsy. These electrodes detect seizures, but can also record other types of brain activity.

The volunteers were played recordings of a word that could either be "faster" or "factor", with the middle sound replaced by noise. Data from the electrodes showed that their brains responded as if they had actually heard the missing "s" or "c" sound.

This seems to be because the inferior frontal cortex region of the brain predicts what word someone is likely to hear, two-tenths of a second before the superior temporal gyrus starts processing the sounds a person has heard (*Nature Communications*, doi.org/bzx8).

"The brain has evolved a way to overcome interruptions that happen in the real world," says Leonard.

Tiny moon past Neptune completes set

EVERYBODY gets a moon. The discovery of a small moon orbiting the third-largest dwarf planet means all the large objects orbiting beyond Neptune have satellites.

Last April, the dwarf planet Makemake became the ninth of the 10 trans-Neptunian objects (TNOs) with diameters of 1000 kilometres known to have a moon.

So when dwarf planet 2007 OR_{10} was found to be rotating more slowly than expected, it was suspected that a moon might be the culprit. To find it, John Stansberry at the Space Telescope Science Institute in Baltimore, Maryland, and his colleagues found eight images of the world in Hubble Space Telescope archives from 2009 and 2010.

The moon appeared in every image. The team presented these results at a planetary sciences meeting in October, and now in a paper (arXiv.org/abs/1703.01407).

Studying the moon's orbit can help determine the mass and composition of 2007 OR_{10}.

TNOs are relics from the era of planet building, so they present an opportunity to peer at our solar system's history. These moons probably formed when a large rock collided with the parent body and the debris coalesced in orbit. The fact all large TNOs have a moon suggests a crowded, chaotic past.

Computer assesses surgeons' stitches

STEADY does it. A video analysis system uses motion tracking and machine learning to rate how good surgeons are at suturing wounds.

"Different surgeons all have different styles of suturing," says Aneeq Zia at the Georgia Institute of Technology in Atlanta. His team filmed 41 surgeons and nurses practising suturing and knot-tying on foam boards. A clinician then rated each person.

A machine-learning algorithm used these scores to learn which features were highly rated. It found the best clinicians moved their hands in a similar way with every stitch. Those with lower scores moved less predictably.

The system then gave the videos its own rating. It matched the clinician's assessments with an accuracy of 93.2 per cent (arxiv.org/abs/1702.07772).

Zia hopes the system could give trainee surgeons useful feedback as they hone their skills.

Asteroid clay could block radiation

FUTURE astronauts could use asteroid clay as a radiation shield.

Cosmic radiation presents a major health risk for astronauts travelling into deep space to set up colonies on the moon or Mars. But the heavy aluminium shields currently used on short missions would be too expensive to ship.

For a long-term presence on other worlds, we will need to use materials found in space, says Daniel Britt at the University of Central Florida. He says asteroids could provide that material.

Clays in asteroids are rich in hydrogen, making them up to 10 per cent better than aluminium at blocking harmful rays (*Advances in Space Research*, doi.org/b33x). But it is still not clear how they would be extracted.

Beetles evolve to look like ant prey

IT'S quite a ploy. Rove beetles blend seamlessly into army ant societies, but instead of helping out, they devour the young of their unsuspecting companions.

The deceit is so successful that it has evolved independently in at least 12 parasitic rove beetle species – a phenomenon called convergent evolution. In each case, the beetles' entire body shape has evolved to resemble the army ants they prey on, and they smell and act like the ants too. They even go marching on raids with them.

"What we found is that multiple times, the ancestors of these rove beetles adapted to life inside army ant colonies," says Joseph Parker at Columbia University in New York. "Each time, their body shape and behaviour underwent the same radical changes."

Parker says the finding challenges arguments by palaeontologist and author Stephen Jay Gould and others that different creatures would evolve if the evolutionary clock was restarted from scratch.

Instead, it suggests that evolution may take similar and predictable paths whenever a certain scenario arises. In this case, distantly related beetles first prey on army ants directly, but later evolve to sneak into the army itself (*Current Biology*, doi.org/b2vj).

TAKU SHIMADA

How to think like a memory champion

BECOMING a memory champion is easier than you think. It seems almost anyone can learn a technique used by mnemonists to memorise hundreds of words or digits in minutes – and it nearly doubles mental performance after six weeks of training.

The technique, called the method of loci, involves imagining a well-used route and associating the information to be learned with landmarks along it.

Martin Dresler at Radboud University Medical Centre in Nijmegen, the Netherlands, and his colleagues recruited 23 of the world's top 50 memory athletes, and scanned their brains to identify features that might mark them out from the general population. They found no structural differences, but recorded unusual patterns of activity in areas linked to memory and visuospatial processing.

Next, they recruited 51 volunteers who had never before tried to improve their memories and split them into three groups. One was told to practise the method of loci for 30 minutes a day for six weeks, and another merely to hold information in their heads for short periods. The third group did no training.

At the start of the study, the volunteers could remember 26 to 30 words from a list of 72. After six weeks, those who practised the method of loci could remember a further 35 words, compared to 11 for the second group and nine for those that did no training.

The brain activity of the first group also became closer to that of the elite memorisers (*Neuron*, DOI: 10.1016/j.neuron.2017.02.003).

Bone-inspired steel resists cracking

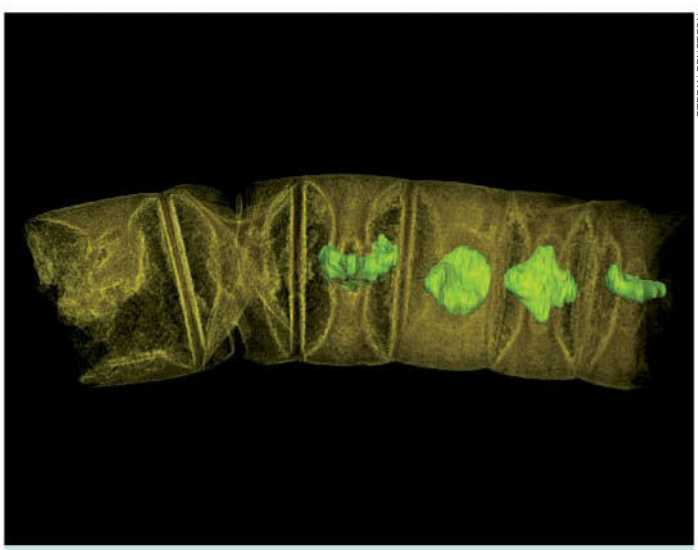
A SPECIAL kind of steel modelled on the properties of bone could be used to make safer buildings and planes.

Steel is vulnerable to scratching, which can cause microcracks that spread until the material fails. To create a more crack-resistant steel, researchers drew inspiration from the complex structure of bones.

Long bones have a thin outer layer of connective tissue covering a matrix of cortical bone. Beneath that is a layer of spongy, porous bone, on top of a hollow centre filled with bone marrow. This hierarchical structure increases the material's resistance to cracks, says Motomichi Koyama at Kyushu University in Japan.

Koyama and colleagues altered the nanostructure of two types of steel to mimic the multilayered structure of bone. When subjected to stress, the new materials showed better resistance than conventional steel. Cracks don't spread as easily, because it takes more energy to find a path through the structure (*Science*, DOI: 10.1126/science.aal2766).

Koyama says it would be possible to scale up production "with a small effort".



STEFAN BENGTSON

Oldest plant-like fossils found in rock

THE oldest plant-like fossils ever discovered suggest multicellular life began at least 1.6 billion years ago.

Fossils of red algae were found in rocks from Chitrakoot in central India embedded in mats of cyanobacteria, called stromatolites. "We have shown with great probability that plants have a history 400 million years older than previously known," says Stefan Bengtson, at the Swedish Museum of Natural History in Stockholm.

His team found distinct cellular structures inside the fossils characteristic of red algae, which are eukaryotic, meaning they have complex cells, like plants and humans. They also found platelets inside the cells, which could be early chloroplasts, the organelles where photosynthesis takes place (*Plos Biology*, DOI: 10.1371/journal.pbio.2000735).

The early development of the multicellular eukaryotic organisms is disputed due to the scarcity of fossils older than 1 billion years. If the new findings and dating of the fossils are correct, the theory of early complex life on Earth will need to be tweaked. "The tree of life has to be recalibrated," says Bengtson.

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Prepare for a visit in 18 years

A promise worth keeping?

A new law highlights the conflict between a sperm or egg donor's privacy and a child's right to know. **Alice Klein** asks what's at stake

IN 1978, Benedict Clark was a 20-year-old medical student when he heeded his lecturer's call for sperm donations. In return, he was paid A$10 and promised eternal anonymity. "It seemed like a good thing to do because there was a shortage of sperm donors," he says. "I felt comfortable knowing that it was anonymous."

Clark was one of hundreds of people – many of them medical students – who agreed to donate sperm or eggs anonymously in the state of Victoria, Australia, in the 1970s, 80s and 90s. That ended in 1998 when a law required that future donors be identifiable.

Now, in a world first, Victoria has retroactively removed the privacy of donors like Clark. Since 1 March, all donor-conceived people have had the legal right to find out their donor's name and date of birth, even if the donor was promised anonymity. Some barriers remain – including a requirement to formally seek permission before making contact, under threat of a A$7500 fine – but Clark, now a general practitioner in Wonthaggi in southern Victoria, says it is still a "broken promise". He believes many who donated would not have done so if they had known they could be tracked down later.

Meanwhile, shortages of donor sperm and eggs are being reported in some countries that have ended anonymity. Other jurisdictions will watch Victoria's experience with interest. Could it put donation in jeopardy?

When IVF began in the 1970s, the common wisdom was that it would be best for the child and their family to have nothing to do with their sperm or egg donor. Over the years, though, it became clear that knowing your genetic identity is important. In recognition of this, since 1998 all new donors in Victoria have had to consent to being identifiable when their offspring turn 18.

Then, one case in Victoria suggested that more was needed.

"Sperm and egg shortages are being reported. Could Victoria's experience put donation in jeopardy?"

Narelle Grech – conceived with anonymous sperm before the law changed – was diagnosed with advanced bowel cancer at 28. She said she cried when her doctor told her it was a heritable disease: "Not only for the fact that I was now terminally ill, but also as I most likely inherited this disease from my paternal family and my anonymous sperm donor", whom she knew only as T5.

History lessons

Donor-conceived people had long argued that anonymity should be lifted retroactively: Grech's case seemed to show why. If she had known she had a family history of early-onset bowel cancer on her donor's side, she might have been screened at a young age, and the disease caught and treated early.

Cases like hers helped change the law again, and donor-conceived people born in Victoria before 1998 can now find out any information in their donor's available health records.

However, it turns out that knowing her donor's identity might not have helped Grech after all. Her donor told *New Scientist* that he knows of no family history of bowel cancer. Neither does Grech's mother.

David Thomas at the Garvan Institute of Medical Research in Sydney, Australia, speculates that Grech's disease may have been due to a unique combination of certain recessive genes whose pairing Grech inherited from her mother and donor.

That being so, there is something that might have helped her better than knowing her donor. Whole-genome sequencing scans your genetic code and looks for mutations that signal an increased risk of cancer, diabetes, heart disease and many other conditions. We don't know every disease-causing mutation, but a whole-genome test can alert people to known ones that warrant early medical attention.

A mutation that might have

caused Grech's susceptibility is a recessive bowel cancer gene called *MUTYH*. If an individual carries two copies of it – one from each parent – they are advised to begin bowel cancer screening from the age of 25.

A whole-genome test is often more reliable than family histories. It could be even more valuable in places where donors remain largely anonymous, says Thomas. This includes France, Spain, and those born before 2005 in the UK, when the country stopped allowing anonymous donations. In addition, most sperm and egg donations in the US are anonymous, although rules vary at different clinics.

In the past, whole-genome sequencing has mainly been confined to research settings. Now companies like Illumina and Veritas Genetics are offering it. The cost is coming down to US$1000 and will continue to fall, says Thomas.

"When we start sequencing everyone's genomes, it won't really matter what your family history says"

So far the test doesn't identify every risk, but that is coming. "When we start sequencing everyone's genomes and learn more, we'll be able to identify all these mutations," Thomas says. "Then it won't really matter what your family history says."

That could be an argument for reversing the drive towards donor disclosure, which may have downsides beyond the discomfort of donors. Some believe that banning anonymous donations in Australia, the UK and New Zealand has led to sperm and egg shortages. For example, Luciano Nardo, a gynaecologist based in the UK, says British fertility clinics increasingly rely on donations imported from Denmark and Spain because it is hard to find donors willing to identify themselves. Because of this, he says the 2005 UK law change should be repealed.

A similar pattern is emerging in Canada, where de-anonymised donation has been correlated with increasing demand for sperm from the US, where no law requires identifiability.

However, that pattern may not show cause and effect. According to the UK Human Fertilisation and Embryology Authority, the number of newly registered donors has actually increased in the UK since anonymous donations were banned. The same trend has happened in Victoria, says Louise Johnson, CEO of the Victorian Assisted Reproductive Treatment Authority, as fertility clinics have become better at recruiting donors.

Another complicating factor is that sperm donors are counted only in countries where they are not anonymous. In Australia and the UK, the number has gone up since donors were made identifiable; in New Zealand, it has held steady. It could still be true that countries with anonymous donations have more donors, but without hard data it is difficult to draw conclusions.

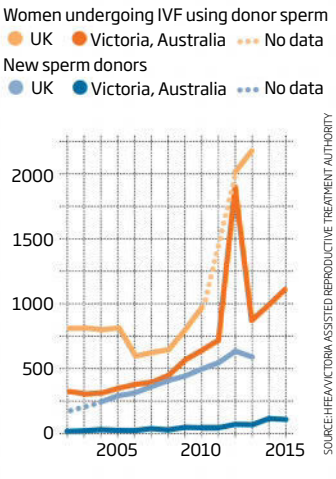
One thing is clear, though – there has been a recent spike in demand for sperm (see graph, above). One of the main reasons is that more single women and women in same-sex relationships are using donor sperm to have children, Johnson says. In the year 2014-15, 85 per cent of donor sperm in Victoria was used by single and lesbian women.

Johnson does not believe that anonymous donations are the way to boost numbers – especially as anonymity is becoming impossible anyway. In the age of genetic genealogy testing and the internet, many donor-conceived people are tracking down their donors on their own (see "DNA detectives", below).

Better options are advertising and promotion, she says. For example, a publicity push in Victoria in 2013-14 almost doubled the number of new sperm donors the following year.

Donor shortage?

Donations have not gone down – where evidence exists – but a sharp rise in IVF means demand exceeds supply



Who am I?

Furthermore, insisting on anonymity assumes that the only reason donor-conceived people look for their donors is medical history. That's not true, says Johnson. "It's also about a sense of identity and fully knowing where you come from," she says. "Many donor-conceived people feel there's a gaping hole in their life without that information and they experience absolute grief over not knowing."

Not knowing her donor's identity while growing up made Chloe Allworthy feel like a puzzle piece was missing. "I had many interests, such as music and dance, that had come out of the blue," she says. "I knew no one else in the family who was interested in these things, or who had a personality like mine."

Allworthy met her sperm donor a year ago, after a search agency helped to find him. He welcomed her into his family and they are still in touch. "Finding my donor gave me a real sense of belonging," she says. She hopes the new law will help more people like her.

Regardless of the science, the voices of donor-conceived people may inevitably be heard more clearly than those of donors like Benedict Clark. During the campaign to change Victoria's law, he says, donors could not mount a counter-campaign themselves – to do so, they would have had to identify themselves. So does loss of anonymity reduce willingness to contribute? We may never know. ■

DNA DETECTIVES

Many donor-conceived people are turning to commercial DNA ancestry tests to track down their sperm or egg donor. These tests sequence customers' DNA and compare it with that of other customers who also want to find relatives. The result is a list of genetic relatives who donor-conceived people can email in search for clues to their donor.

Chloe Allworthy took three such tests, from Family Tree DNA, AncestryDNA and 23andMe. Her mother did likewise, to rule out relatives on her side. The closest match Allworthy found was her donor's third cousin – according to their DNA – but this relative did not know the donor's identity. Nevertheless, Allworthy worked out that her donor was probably in South Australia, based on the location of most of his relatives in the database. She enlisted a search agency to help her find him, but that took two years.

Other donor-conceived people have had more luck. In 2005, a 15-year-old boy became the first to find his sperm donor using a DNA ancestry test. He matched two relatives of his donor and used their last names as clues. His mother had a record of his donor's birthday and birthplace, so the boy was able to match the name with a birth record.

Allworthy thinks stories like this are becoming more common. "So many donor-conceived friends have tracked through DNA databases to locate their donor," she says.

A grimmer outlook

Ripping up the Obamacare health system would be a blow for the well-being of millions of Americans, says **Laudan Aron**

A VOW to "repeal and replace" the US healthcare framework known as Obamacare was a key part of Donald Trump's presidential campaign.

So last week, the Republican-controlled Congress introduced a bill called the American Health Care Act in an effort to keep that promise. While the details of its potential impact were still being worked out, its broad significance was crystal clear: the new bill will dismantle much of Obamacare.

To understand what this means you need to comprehend the law it aims to undo, properly called the Affordable Care Act (ACA). According to Linda Blumberg and others at the Urban Institute, the non-partisan think tank where I work, the ACA has accomplished a lot, expanding health insurance coverage to an estimated 20 million extra people and cutting the uninsured rate among non-elderly adults by about 40 per cent. It improved access to care and reduced discrimination by health status. Predictions of unsustainable costs, both for the government and across the health sector, have not become a reality and rates of employer-sponsored insurance coverage remain high.

Republicans claim the ACA is in a death spiral – a scenario in which premiums soar as enrolment drops. That is untrue. Some areas have seen significant increases in premiums, but mostly because insurers had priced plans too low originally; these premiums are not high in absolute terms.

Initial assessments of the ACA alternative do not bode well for the health of US citizens. Most experts say it will cut coverage and raise household costs.

The bill eliminates the mandate requiring most US citizens to have health insurance, a centrepiece of

Eater beware

An opaque web of supply and processing has left faith in food fragile, says **Nicola Temple**

YOU probably know about the row over Subway's chicken by now. A broadcaster in Canada said that the DNA in samples from the fast food chain was only about 50 per cent chicken, the rest mostly soya.

I smiled when I heard. Not because I have anything against Subway. I don't, and it has denied the allegation, saying protein-based tests, arguably better for quantifying ingredients, show soya is just a tiny bit of its product.

No, I smiled because I thought of how a woman from the Canadian Food Inspection Agency had responded to a talk I gave about this kind of thing last year. I cited Canadian examples where fish had been mislabelled as more expensive species, spices padded out with cheap fillers and "locally" grown vegetables turned out to be from Mexico. She spoke up a lot to repeat her mantra: Canada's food is the safest in the world.

Of course, she is right. Its food is very safe. But authenticity and safety are often separate concerns, and we still worry a lot about the veracity of food labels.

We worry because what we eat often reaches us via an incredibly complex, largely anonymous supply network. Within this, production processes can be opaque, labels ambiguous and authenticity testing sparse. So are we destined to always dine with a side serving of scepticism?

"Food vendors have to know not just who supplies them, but who supplies those suppliers"

Maybe not. There is an armoury of analytical methods that can help. DNA tests, immunoassays, chromatography and isotope analysis can shed light on what's in food and where it comes from.

Governments do use these to monitor regulatory compliance. However, with resources tight, safety testing takes priority. As for doing it yourself, there simply isn't a market for a $20 single-use dipstick that diners can plunge

the ACA and a key provision for pooling risk in insurance markets.

Just as crucially, it undermines Medicaid, an important national safety net providing more than 70 million low-income people – often children, senior citizens or people with disabilities – with insurance and other critical benefits.

Incredibly, an ACA requirement that cover must include essential services such as emergency care, maternity and newborn services, prescription drugs, and mental health and addiction treatment has been scrapped for some plans.

For decades, US citizens across all ages and classes have died younger and had higher rates of disease and injury than citizens of other wealthy nations. Last year, US life expectancy dropped for the first time since 1993.

Many factors contribute to this, and how we organise and deliver healthcare is one of them. High-quality, affordable healthcare does not guarantee good health for a nation, but it is a critical ingredient. Many Americans may lose this if the Republican Party's ACA "replacement" passes.

The result will be a giant step backwards for the nation's health and well-being. ■

Laudan Aron is a senior fellow with the Urban Institute in Washington DC

into battered fish to see if it is cod.

In any event, this will take more than technology. Good business practices are also key. Food vendors have to know not just who supplies them, but who supplies those suppliers.

Some firms are conducting regular internal authenticity tests and working this assurance into their brand. But all this comes at a cost. Ultimately, real progress may boil down to how much we, as consumers, are willing to pay in order to trust our food. ■

Nicola Temple is a science writer and co-author of *Sorting the Beef from the Bull* (Bloomsbury)

INSIGHT Breastfeeding

CHRISTINE HENKE/PLAINPICTURE

Feed me!

When breast isn't best, or even enough

Clare Wilson

"BREAST is best". This well-known slogan is meant to convey that babies should be fed exclusively with breast milk. And there are many health benefits associated with breast milk.

But some extreme proponents insist on breastfeeding at all costs, leading to fears that following this advice in the first few days after birth could in rare cases have tragic consequences.

Christie del Castillo-Hegyi's baby son became dehydrated after she struggled to produce milk but was discouraged in hospital from supplementing with formula. She believes neonatal dehydration was a factor in his later diagnosis with a seizure disorder.

She has begun a campaign called "Fed is Best" to highlight cases of other babies who went so hungry thanks to breastfeeding dogma they experienced serious harm. Rather than being pro-formula, the campaign is against ideology being "pushed" at the expense of babies' health.

The idea that an over-focus on breastfeeding could be bad is darkly ironic given the history behind "Breast is best". For decades, formula makers claimed their product was better than breast milk, which proved deadly in countries where the water used to make up formula can be contaminated.

As a result, the World Health Organization recommended restrictions on how formula can be marketed. Many hospitals now strive to meet UN-approved "baby friendly" criteria that prioritise breastfeeding. Formula is discouraged, partly because giving bottles can mean babies suck less at the breast, leading to a vicious cycle of falling milk production and more formula use.

"If it takes too long to start producing milk, the baby can be in danger of dehydration and jaundice"

This is not inevitable. In fact, some research suggests that if breastfeeding isn't going well, a little formula in the first few days after birth can lead to better long-term breastfeeding rates.

Why does it need any intervention at all? One problem is that it takes women several days after birth to start producing much milk. If it takes too long, the baby can be in danger of going short of calories, getting dehydrated or jaundiced.

Breastfeeding advocates argue that insufficient milk is rare, but a study at a hospital in China found that jaundice rates had risen since the hospital started promoting breastfeeding.

The Fed is Best campaign also seeks to bust some of the more damaging breastfeeding myths. For example, women who voice concerns they might not be making enough milk are often reassured that this is unlikely as a newborn's stomach is just 5 millilitres in size. In fact, studies suggest it is about 20 millilitres.

Indeed, merchandise has been created to convey the false small size of a baby's stomach. Del Castillo-Hegyi is now trying to get one such product – a lanyard depicting a 5 millilitre stomach – taken off the market.

Breastfeeding advocates are up in arms about the Fed is Best campaign, branding it as scaremongering. Debra Bick of King's College London says a bigger priority is for health services to give more support for women who are struggling to breastfeed.

But Del Castillo-Hegyi says more focus should be on breastfed babies, including more checks in their first few days to ensure they are getting enough milk. "Promoting exclusive breastfeeding is a public health intervention that we haven't asked enough questions about," she says. ■

Desert mirage

IN THE Dubai desert, not all is as it seems. In 2008, Irenaeus Herok was driving outside the city of Dubai, when a strange set of buildings suddenly loomed up in the sand ahead.

When the photographer arrived at the shimmering towers, they turned out to be fake. A large white board, roughly 15 metres high and 80 metres wide, had been painted like a movie set. "It has this really weird effect when you drive past," Herok says. "It comes out of nowhere in the middle of the desert and looks kind of real, but you don't know what it is."

Exploring further, Herok discovered half a dozen more of these massive painted boards, each spaced about a kilometre apart. They turned out to be the vestiges of failed plans for a theme park and residential zone in the area. The developers erected these structures to give investors a lifelike vision of what the scheme would look like, says Herok.

"When you drive around Dubai, it's easy to get lost because there's just sand everywhere. You turn right, and there's sand again," he says. "So I think they put these sets here to show people how [the development] would be arranged."

But it didn't work. When Herok returned eight years later, there was no sign of a construction site nor the sets. "Like a mirage, they seem to have never existed – swallowed by the desert and the heat." Alice Klein

Photographer
Irenaeus Herok
instagram.com/iherok

Strangely attractive

Fresh suspicions have reopened the case for a radical rethink of gravity, says **Mark Anderson**

GRAVITY is supposed to be reliable. It's the familiar force that keeps our feet on the ground and Earth's atmosphere from hurtling into space. On grander scales, it has shaped the evolution of the universe. What a shame, then, that it sometimes lets you down. To square the whirligig rotations of galaxies and galaxy clusters with our picture of gravity, we have to invent a whole new form of matter that no one has ever seen: dark matter. To explain why the universe's expansion is accelerating, we have to conjure up an equally mysterious essence known as dark energy.

But what if we never really knew gravity at all? What if out there, beyond where we can easily keep our eye on it, the universal force doesn't stick to the rules?

It's a heretical idea, if not an entirely novel one. Now though, renewed scrutiny of galaxies and surprises from the realm of quantum information theory are reinvigorating the quest to rethink gravity. Radical ideas are emerging that amount to a fundamental transformation of how we understand space-time – and what gravity really is. In this picture, dark matter ceases to exist. And dark energy, rather than being something that works against gravity, might be part of what creates it.

Pretty much everything we know about gravity comes from Isaac Newton and Albert Einstein. The strength of the pull exerted by a given object declines in proportion to the square of the distance from it, Newton told us, while Einstein explained gravity as the result of massive objects curving space-time.

Newton's inverse-square law dictates that stars far from the centre of a galaxy should feel less gravitational pull, and therefore orbit more slowly, than stars closer in. But in the 1970s, astronomers including Vera Rubin noticed that farther out from a galaxy's central bulge, the velocities of stars did not continue to drop as predicted. Instead they levelled off, an observation that could only be explained if there was some invisible form of matter surrounding galaxies to provide an extra gravitational kick. We have been searching for this dark matter ever since.

Bending the rules

Well, not everyone has. In the 1980s, Mordehai Milgrom, then at Princeton University, showed that you can explain the oddball rotation speeds of outlying stars without invoking dark matter. The trick was to ditch the idea that gravity always obeys Newton and Einstein as its strength begins to wane. Milgrom's theory, known as MOND for "modified Newtonian dynamics", posited that gravity's pull tails off more gradually than Newton predicts. As soon an object's acceleration due to gravity drops below a particular value, 82 billion times weaker than what we experience on Earth, gravity ›

JULIEN PACAUD

suddenly switches to this new regime.

Milgrom had some success applying his theory to spiral galaxies, but MOND never really caught on. For starters, it failed to account for clusters of galaxies, which couldn't hold together without dark matter or modifications to gravity beyond what MOND allowed. It also seemed suspiciously ad hoc. Why would gravity's strength suddenly switch at this seemingly arbitrary point?

And yet MOND never really went away – not least because no one has actually detected dark matter. "There are two possibilities," says John Moffat at the Perimeter Institute for Theoretical Physics in Waterloo, Canada. Either we find this invisible source of additional gravity, and we reassure ourselves that Newton and Einstein were right all along. Or we don't. In which case "the alternative is to modify gravity", says Moffat.

Last year may have finally brought a tipping point. Stacy McGaugh, an astronomer at Case Western Reserve University in Cleveland, Ohio, and his colleagues took a fresh look at more than 150 spiral galaxies similar to our own Milky Way (see page 32). When they compared gravity's inferred strength for each galaxy with its disc's rotation speed, they found that the stars had anomalously high speeds farther out from the centre.

So what? That's precisely the sort of behaviour we've observed many times before, and you explain it by adding a halo of dark matter around the galaxies. But McGaugh's statistical survey included a cross-check. Taking a census of all visible matter in every galaxy, he compared its gravitational pull at every point with the rotation speed of nearby stars. The result was a surprisingly tight correlation between the galaxies' rotation speeds and the distribution of the visible matter they contain.

Darkness falls

Lee Smolin, a theorist at the Perimeter Institute in Canada, was stunned. This relationship is "tantamount to a natural law", he says – not something you expect to see if something other than visible matter dominates these galaxies.

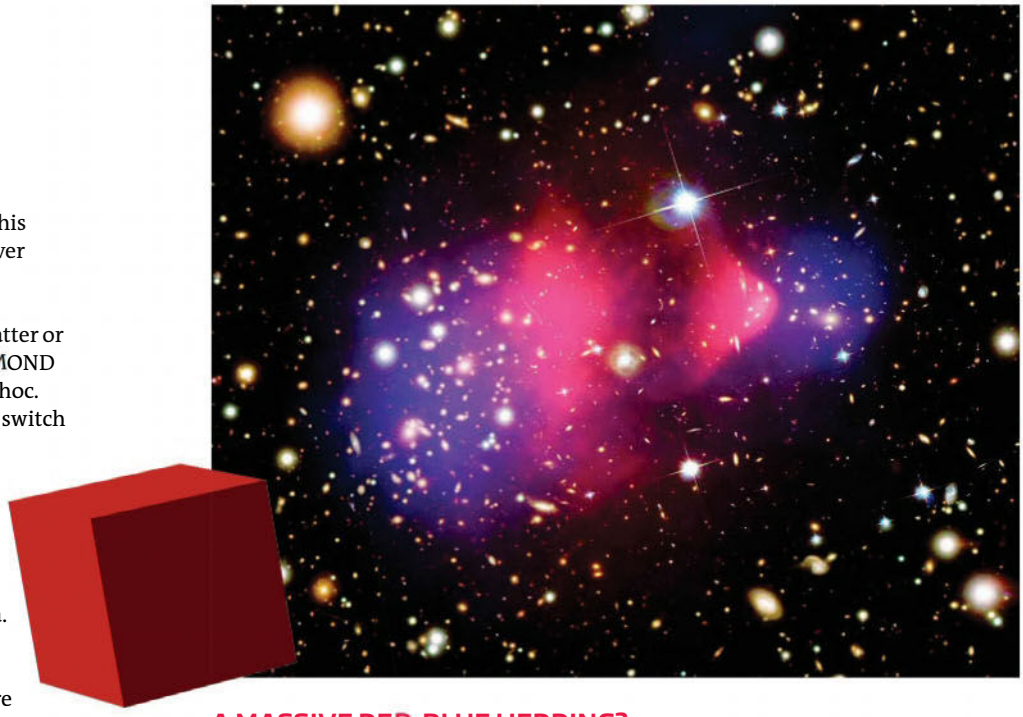
Even more eyebrow-raising is the fact that this close relationship between visible matter and the movements of stars appears to hold across such a wide range of galaxies, even though those galaxies are not thought to hold identical dark matter distributions. Dark matter is not supposed to slavishly follow the whereabouts of ordinary stuff. So either it interacts with visible matter or itself more than simple models suggest, or there is something up with gravity.

McGaugh's work is not the only thing that has reawakened this heretical notion. One of the biggest problems for MOND is the behaviour of clusters of galaxies. Like stars at the edges of galaxies, galaxies on the fringes of clusters also seems to orbit too fast – something that has been explained with dark matter. Observations of gravitational lensing, the subtle warping of light by matter, suggest that the source of the additional force catapulting the galaxies around is located somewhere other than the visible matter. You simply can't explain galaxy clusters without invisible matter, or so the story goes.

The most notorious example is the Bullet Cluster, named for its resemblance to high-speed images of projectiles shooting something to smithereens (see "A massive red-blue herring?", above). For many dark matter hunters, this is the best evidence that their quarry must exist. But Pavel Kroupa at the University of Bonn, Germany, argues exactly the opposite – only with MOND can you explain this high-speed intergalactic collision.

"It's an incredible public relations gag," he says. Kroupa argues that standard gravity is too feeble to produce galaxy collisions as hot and furious as the Bullet Cluster in a realistic time frame. Dark matter might juice an initial collision up to the high speeds we see, but it would gum up every interaction thereafter. "A dark matter halo is like a spider's web," Kroupa says. "It captures any incoming galaxy." So a pair of post-collision galaxies that are still zipping around at high speeds become really hard to explain. "This is a big, big problem for the standard model of cosmology," says Kroupa. "But with modified gravity… this problem doesn't exist."

The whole point of MOND is that over galactic and extragalactic distances, where we can never directly test its strength, gravity is stronger than we assumed. That, rather than some invisible form of matter, would be the simplest explanation as to



X-RAY: NASA/CXC/CFA/M.MARKEVITCH ET AL.; OPTICAL: NASA/STSCI/MAGELLAN/U.ARIZONA/D.CLOWE ET AL.; LENSING MAP: NASA/STSCI; ESO WFI; MAGELLAN/U.ARIZONA/D.CLOWE ET AL.

A MASSIVE RED-BLUE HERRING?

The Bullet Cluster, actually a collision between two clusters of galaxies, is often invoked as the smoking gun for dark matter. Although the individual galaxies glided past each other, the hot gas (pink) around them collided and slowed, leaving a trail behind each cluster. Oddly, most of the mass (blue), inferred by the way it bends light, has stuck with the galaxies. Hot gas is thought to form the bulk of visible matter, so the mismatch between where the mass should be and where it is shouts dark matter.

But in recent years dissenting voices have said the ferocity of the collision is impossible in a universe dominated by dark matter. In fact, tweaking the laws of gravity might better explain this smash-up (see main story).

why things at these scales appear to move faster and collide more furiously than Newton or Einstein predict.

This is not to say MOND doesn't have some problems when it comes to gravitationally interacting galaxy clusters. In the Bullet Cluster, our telescopes point to two distinct regions of stronger gravitational lensing, and thus higher concentrations of mass, that are separate from what you would expect given the mass of the ordinary matter we observe.

Milgrom insists that his model is not nearly as imperilled by that as many claim. "You need only a little amount of unobservable matter, which could just be some normal matter like dead stars or cold gas clouds that has not been detected yet," he says.

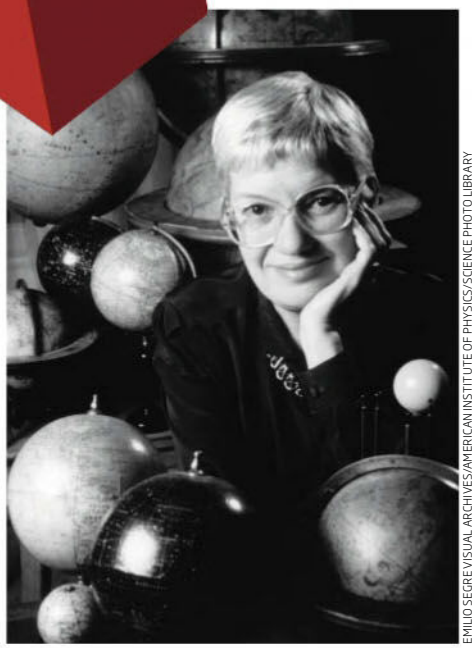
In the absence of such observations, however, others are seeking new theoretical solutions. One is a hybrid model featuring a shape-shifting version of dark matter that flows unimpeded in galaxies, creating a MOND-like extra pull, then behaves like orthodox dark matter in galaxy clusters.

The other option, suddenly in vogue again, is to modify MOND. That is precisely what Moffat has been doing. In his version, the strength of gravity varies thanks to the addition of a repulsive force that itself varies with distance, making gravity follow a Newton-like inverse square law closer in, only to peter out at distant reaches of a galaxy. In that realm gravity is stronger than Newton would allow, behaving as MOND predicts.

Moffat claims his theory can account for the rotations of galaxies and the anomalous motions of the Bullet Cluster. But what really sets it apart is that it produces stronger gravitational effects than even MOND would predict near black holes, which might give us a chance to put it to the test.

If we could see a black hole, we would see a dark disk surrounded by a shadow caused by extreme gravitational lensing. In 2015, Moffat worked out that, according to his revisions of gravity, the shadow around the supermassive black hole at the centre of the Milky Way will appear as much as 10 times bigger than general relativity predicts.

Enter the Event Horizon Telescope, a global network of radio dishes capable of capturing detailed images of black holes for the first time, set to come online this April. In principle at least, it should be able to see this bloated shadow – if it's there.



EMILIO SEGRE VISUAL ARCHIVES/AMERICAN INSTITUTE OF PHYSICS/SCIENCE PHOTO LIBRARY

Vera Rubin saw that Newton's gravity is not enough in the 1970s

Even so, whether we go for traditional MOND or Moffat's modified gravity, there is still a massive elephant in the room: the glaring absence of an underlying theory. Why would gravity suddenly deviate from what Newton and Einstein laid down, and at a seemingly arbitrary scale? The answers might lie in a radical rethink of what it actually is.

Last year, Erik Verlinde at the University of Amsterdam in the Netherlands came up with a fresh vision. Gravity, he suggests, is really an emergent phenomenon – a consequence of interactions between entangled bits of quantum information.

Entanglement is a deep but deeply counter-intuitive quantum mechanical connection between pairs or groups of particles in which actions performed on one affect the others, even if they are separated by large distances. Physicists have been able to make Einsteinian and Newtonian gravity emerge from networks of entangled quantum bits since the late 1990s. The problem is that it only works in a model universe known as anti-de-Sitter space, which doesn't behave like the one we inhabit.

The key difference is that our universe's vacuum is not conveniently quiescent. Instead, it is roiling with what we call dark energy, a mysterious substance or force thought to be responsible for the accelerating expansion of space-time.

Rather than try to work around the problem, Verlinde explored how emergent gravity might behave in a universe infused with dark energy. The result is a new picture of gravity in which this background energy imbues the entanglement of quantum bits with something akin to an extra degree of elasticity.

"It's like dark energy is acting like an elastic medium," Verlinde says. "And the matter we put in starts deforming that medium." The extra elasticity provided by dark energy, he adds, boosts gravity's strength at long range, resulting in an additional far-field effect that resembles Milgrom's MOND.

A stretch too far?

Verlinde's ideas made a big splash, but it's still not clear how coherent they are. "He starts with dark energy, and he says this leads to something that looks like dark matter," says Sabine Hossenfelder at the Frankfurt Institute for Advanced Studies in Germany. "He makes a lot of effort to embed this into the large idea of space-time emerging from entanglement, which has become very popular in recent years. But I'm not sure that is necessary."

One recent study has shown that Verlinde's recasting of gravity can explain gravitational lensing anomalies in the vicinity of some 30,000 foreground galaxies. But his theory has come under fire for predictions it makes that, in fact, diverge from MOND. A study co-authored by McGaugh, for instance, suggests that Verlinde's ideas fail to deliver on MOND's core strength – explaining the anomalous rotations of galaxies. Another found they predict planetary motions that bear no relation to what we see in our solar system.

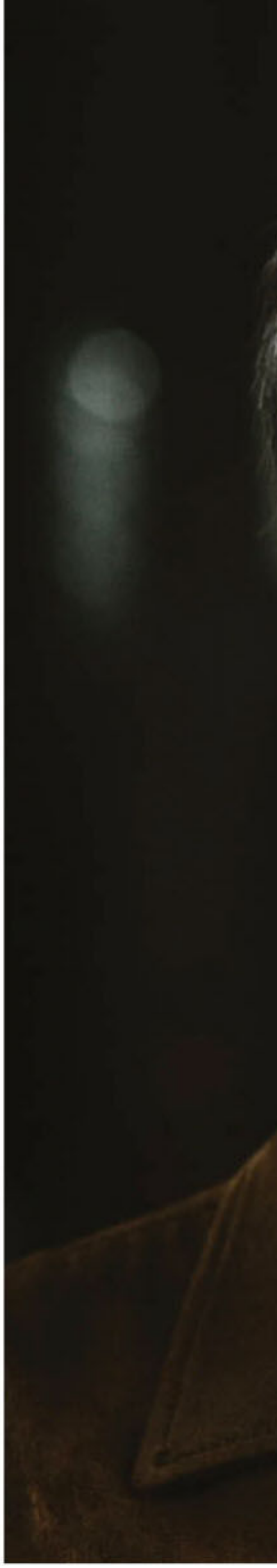
For his part, Smolin has come up with a more modest attempt to derive MOND-like physics from first principles of quantum gravity – and unlike Verlinde's theory, it doesn't produce anything that diverges from MOND. Neither of them is claiming to have a complete theory of quantum gravity. It is clear, though, that when it comes to the question of why gravity acts strangely far from home, theorists are starting to come up with answers.

"We don't know where the final theory takes us, because we don't have it yet," says McGaugh. "So there needs to be a period of uncertainty and scattershot, in order to find our way forward." ■

Mark Anderson is a science journalist based in Massachussetts

From light into dark, and back again

Once an advocate of dark matter, **Stacy McGaugh** would happily do away with it. But, he says, his friend Vera Rubin – the "queen of dark matter" – didn't hold it against him



When did you become aware of dark matter?
I took a class in the mid-1980s in which astrophysicist Scott Tremaine said it had to exist. The class was sceptical, me included.

How did he explain the need for dark matter?
Newton's law of gravity says that the stars and gas in a galaxy should orbit the galactic centre ever more slowly with increasing distance. But in the 1970s, the astronomer Vera Rubin and others had found that orbital speeds first increase as you move away from the centre, and then stay more or less constant with increasing distance, giving what's called a flat rotation curve.

Tremaine showed us a graph of such a curve. It meant that those outer stars were moving too fast, and Newtonian gravity just couldn't account for it. He showed us how both normal and dark matter were needed to explain that flat curve: the mass of dark matter provided the additional gravitational pull.

Did the idea of dark matter appeal to you?
Not at first, but I became convinced by the data that it had to be so. By the mid-1990s, however, I ran into a problem. It had to do with low-surface-brightness (LSB) galaxies, of which even the brightest examples are dimmer in the sky than the darkest night. I was a postdoc at the University of Cambridge, and I had developed my own theory of how galaxies formed using models involving dark matter. Basically, everything I had learned about these dim galaxies suggested that they were simply stretched-out versions of brighter galaxies. One of my predictions was that groups of LSB galaxies would cluster differently compared with brighter galaxies. That prediction was confirmed. I was impressed with myself.

So where was the problem with dark matter?
My theory failed to correctly predict how fast these dim galaxies were rotating. That was a big blow to my ego. The only way to make them rotate correctly in my theory was to fine-tune the dark matter: the more I stretched out the stars in a given galaxy, the more dark matter I had to throw in to balance things out. To me, this fine-tuning was a problem. You feel like the Wizard of Oz: the man behind the curtain turning knobs and dials. If you don't look behind the curtain, then yes, you can produce something that sort of looks like the data, but it's completely unnatural. I pounded my head against that problem for months.

And that struggle soured you to dark matter?
Yes, but only thanks to a chance event. Towards the end of my stay in Cambridge, physicist Mordehai Milgrom came to give a talk on his theory of gravity, called modified Newtonian dynamics (MOND). His idea was that the gravitational force is as Newton prescribed for the most part, but acts slightly differently at very low accelerations due to gravity, so there was no need for dark matter. I almost skipped the presentation because I thought it was crazy talk. Still, I went. Then at one point, in just three lines on the board, Milgrom derived equations that predicted the behaviour of LSB galaxies. And that was exactly what I had been observing and had been unable to explain satisfactorily with dark matter. I was flabbergasted.

So, did you begin working on this theory of modified gravity?
Testing MOND was not my priority, but when I got around to it, my attitude was "My data for LSB galaxies will falsify this stupid theory". The data told a different story. Then, shortly after I met Vera Rubin while we were both visiting the University of Groningen in the Netherlands, she offered me a fellowship at the Department of Terrestrial Magnetism in Washington DC, where she worked.

What was your reaction to the job offer?
Here was the queen of dark matter offering me a job, when I had suddenly become interested in this radical non-dark-matter theory! So I said, "Look, I'm interested in MOND, is that

PROFILE
Stacy McGaugh is an astronomer and professor at Case Western Reserve University in Cleveland, Ohio

Photographed for New Scientist by Andew Spear

OK? Do you still want to hire me?" She had a brief look of shock on her face, but, to her credit, she said, "Of course. It's a fellowship, you can work on whatever you want."

So that's when you started working on this radical theory in earnest?
Yes. I spent much of my time at the department going through different kinds of astronomical data to see whether MOND could explain them. People weren't giving the theory a fair shake. There was a huge dark-matter bias in the field – and it's still there. That brought out the social justice warrior in me. It turns out that MOND explains many scenarios well, though admittedly not all. It can't account for the dynamics of clusters of galaxies, for example.

Your new work is raising hackles again. What did you find?
My colleagues and I observed a large sample of different types of galaxies. We found a direct correlation between the distribution of normal matter – the stars and gas – and the rotation curves, even though the curves don't behave according to Newton's laws. It's like a there is a single, universal force law in galaxies that depends on normal matter alone. One that happens to look exactly like MOND.

Could dark matter explain your results?
It seems unnatural to invoke dark matter, because you have to arrange it to be just so, in just the right place, to get this one apparently universal force law to work. Also, the dark-matter theorists have to explain why it is that normal matter alone so gracefully maps on to the motions of galaxies (see page 28).

"It's like God shouting: 'There's more to gravity, not more mass in the universe!'"

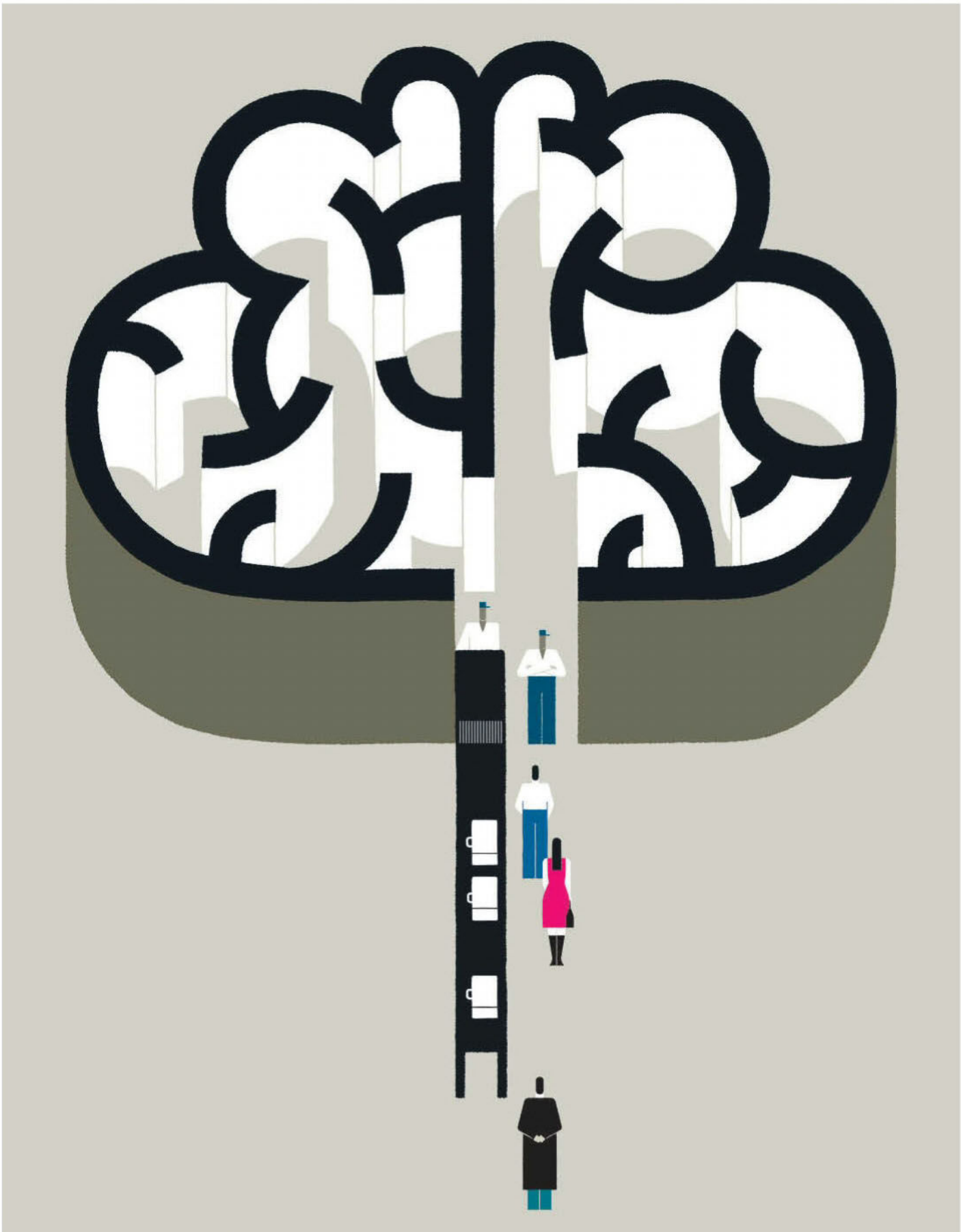
To me it's a big stop sign in the sky. If it's true that there is one force law in galaxies, it's like God shouting, "There is something more to the theory of gravity, not something more to the mass of the universe!".

Does that mean dark matter is dead?
No. While the dynamics of galaxies scream MOND, the dark-matter picture works well at larger scales in cosmology, and MOND doesn't. It doesn't seem that we can just pick one. Somehow we must reconcile the irreconcilable.

What would Rubin have said about your find?
She'd say it is early days and we still have a lot to learn. She was an open-minded scientist: always very positive about everything, incredibly curious and just wanting to know how things worked. From her I learned to appreciate the accomplishment of others, even if I didn't agree with their world view.

When Rubin died in December, some lamented that she had missed out on the Nobel prize...
Vera really didn't care about that, because she self-identified as an astronomer. She explicitly said to me, "I'm not a physicist". So, if the prize is for physics, then I think in her own mind, it didn't apply to her. I wouldn't agree. Her work had a profound influence on physics. Some say she discovered dark matter, but I don't think she'd agree with that herself. She discovered that the rotation curves of galaxies were flat. I think she found a law of nature, and that in itself is worthy of a Nobel prize. ■

Anil Ananthaswamy is a consultant for *New Scientist*



MARTIN LEON BARRETO

Border control

Covert exchanges between the brain and the immune system are transforming our understanding of neurological disorders, says **James Mitchell Crow**

YOUR brain enjoys a life of privilege. That extraordinary lump of jelly-like tissue between your ears makes up only 2 per cent of your body mass, but demands 25 per cent of your daily energy requirements. It is surrounded by the fortress of your skull to shield it from the outside world, and cushioned in a bath of nourishing fluid. It is even protected from you, insulated from the caprices of the body's immune system to guard against inflammation.

Hold fire on that last one. It turns out that the barrier between the brain and the body's defence forces isn't as impassable as we thought. Yes, it stops all sorts of unwanted interlopers, as well as frustrating attempts to get drugs into the brain via the bloodstream. But there is communication across this frontier. Over the past few years, we have begun to see that the brain is in constant dialogue with the immune system and even allows some foreign agents in – discoveries that are shedding new light on everything from epilepsy and Alzheimer's disease to autism.

The brain got its aloof reputation in the late 19th century, when German immunologist Paul Ehrlich noticed something odd. The dyes he was injecting under the skin of lab animals perfused virtually every tissue, turning organs a striking blue – except the brain. Later, people noticed that dye injected into the brain didn't stain the rest of the body.

Only in the 1960s did we figure out why. As some of the first images captured by electron microscope showed, the endothelial cells lining blood vessels in the brain are different from those elsewhere in the body. Here they form tight junctions with their neighbours, creating a seal known as the blood-brain barrier that keeps out dangerous interlopers such as bacteria. Experiments showed the seal also blocked immune cells – prompting the idea that, as far as the brain was concerned, the body's trusted guardians were dangerous too.

"Immune cells, which were the only part of the immune system we knew about, did not get into the brain, period," says William Banks at the University of Washington in Seattle. The one known exception, seen in multiple sclerosis, only seemed to prove the rule: the disease is a result of immune cells invading the central nervous system.

The idea of a border closed to the body's immune system soon established itself as dogma. And yet when researchers began to inspect the blood-brain barrier more closely, they saw that it was not entirely impenetrable.

As it became clear that various immune cells secrete a flood of signalling molecules called cytokines, researchers began to wonder if those molecules could breach the border. In 1989, Banks was among the first to show that they do. He demonstrated that a cytokine called interleukin-1 alpha, which can induce fever, is pumped across the border – a situation since proven with other immune messengers. The result was a dramatic shift in our understanding of the blood-brain barrier.

> "One component of Alzheimer's is slow damage to the blood-brain barrier"

"It is still critical, but now we look at it as an interface," says Banks. "First it creates the barrier, and then it allows exceptions." Indeed, it turns out that the cells forming the seal are in constant communication with the brain cells they protect, but also with immune cells circulating in the blood – a conversation that determines what is blocked and what is allowed to cross.

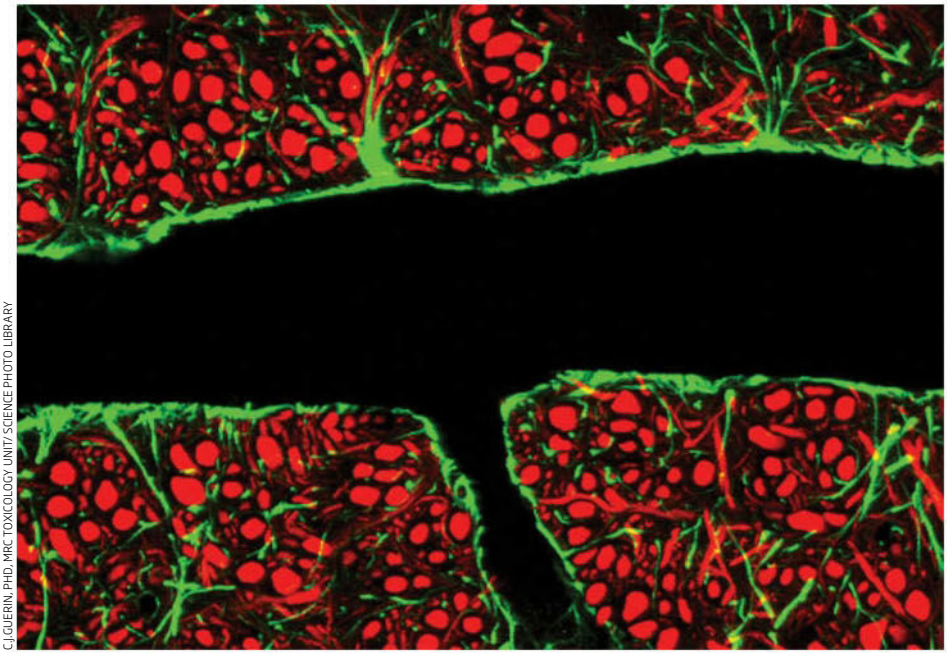
In some cases, their chatter is disrupted by acute damage: a stroke, say, or repeated concussive blows (See "When dementia strikes", page 36). But it can also shift more subtly. And by eavesdropping on the elaborate crosstalk, we have recently discovered that the blood-brain barrier is implicated in various neurological problems. "With any type of chronic infection, or with diseases like epilepsy or Alzheimer's disease, one of the components is the long, slow, irreversible damage of the blood-brain barrier," says Nicola Marchi at the Institute for Functional Genomics in Montpellier, France.

Take epilepsy. Using animal models, and by observing people whose blood-brain barrier is deliberately disrupted to allow chemotherapy drugs into the brain, Marchi has demonstrated that changes in the barrier's porousness can trigger inflammatory pathways that alter the activity of neurons, predisposing the brain to seizures. It also works the other way round. "If you have recurrent seizures, that's sufficient to promote inflammation in the brain and the opening of the blood-brain barrier," he says.

It's hard to figure out whether changes in the barrier are a cause or consequence, but there seems to be a vicious circle. Marchi hopes to develop drugs to halt this by pinning down the mechanism by which seizures lead to the leakiness that in turn allows inflammatory molecules to flood the brain. He has been focusing on pericytes, one of the most important cells forming the blood-brain barrier. In epilepsy, the connections between these and other cells in the barrier loosen, and Marchi suspects this is down to pro-inflammatory cytokines. He is plying pericytes with various cytokines to try to identify the culprit, which he would block with a drug.

Escalating inflammation in the brain seems to be a common factor in neurodegenerative diseases, Marchi says. "It's a concept that can be applied to many brain disorders."

Perhaps the most compelling case is Alzheimer's. Neuron damage associated with this form of dementia is usually thought of ›



C.J.GUERIN, PHD, MRC TOXICOLOGY UNIT/ SCIENCE PHOTO LIBRARY

The protective wrapping around blood vessels in the brain is far from impermeable

as a result of the build-up of a protein called beta-amyloid. In recent years, however, the spotlight has also begun to fall on disruptions of the blood-brain barrier associated with the disease. We have long known about these, but generally viewed them as a consequence rather than a cause.

Now we're not so sure. In 2012, a team led by Berislav Zlokovic at the University of Southern California in Los Angeles showed that alterations in pericytes in mice bred to develop Alzheimer's-like symptoms preceded the onset of neurodegeneration. A protein made by a gene associated with Alzheimer's in humans, APOE4, appears to unleash a destructive inflammatory response in pericytes, opening up the brain to neurotoxins.

A year later, the same team showed how the failure of pericytes might relate to beta-amyloid accumulation. These specialist cells are equipped with a type of protein that acts as a pump, which is one of the major routes by which beta-amyloid is cleared from the brain. The more pericytes go awry, the less effectively the brain can rid itself of amyloid. By targeting one component in the cascade of signalling molecules that drives inflammation in pericytes, the team was able to heal the blood-brain barrier and reverse neuronal damage.

Nobody expects that patching these brain-body interfaces will be a complete solution. "I don't think that by fixing the blood-brain barrier you will cure epilepsy, just like I don't think by fixing the barrier you will cure Alzheimer's," says Marchi. "But if you fix it then maybe an antiepileptic drug will function better."

It doesn't end there, though. As our understanding of the brain's border control has evolved, we have also been forced to confront long-held assumptions that the immune system, and in particular molecules that promote inflammation, are unwelcome in the brain.

The idea has never sat well with Michal Schwartz, a neuroscientist at the Weizmann Institute for Science in Rehovot, Israel. Why would an organ as indispensable as the brain not avail itself of the sophisticated protection and repair services offered by the immune system? "It didn't make sense to me, so I decided to revisit the whole issue," Schwartz says.

Slowly but surely, she broke down the misconception. First she showed that immune cells called macrophages help restore nerve function following a spinal cord injury. Then, in 2004, Schwartz and her colleague Jonathan Kipnis showed that mice bred to lack T-cells struggled mentally and failed at the most basic cognitive tasks, such as finding a hidden platform in a pool of water.

But there were puzzles: how do macrophages enter the brain when they can't cross the blood-brain barrier? And how do T-cells, a class of white blood cells that patrol the body, influence brain function when they aren't found in brain tissue? It was in the process of answering these questions that Schwartz discovered a link between certain immune infiltrations in the brain and protection against neurodegeneration.

In 2013, she and her colleagues found that the normal blood-brain barrier rules don't apply at a structure called the choroid plexus. Here, a different kind of cellular seal separates blood from brain – one which macrophages can cross, a process controlled in part by a cytokine called interferon gamma. Schwartz found that cytokine signalling here tends to weaken as we age, reducing the number of macrophages that get into the brain. She later found that the communication across this border was shut down completely in mice bred to have a condition equivalent to Alzheimer's –

WHEN DEMENTIA STRIKES

When Tom McHale died from an accidental overdose of painkillers, aged 45, his autopsy revealed the cause of his tragic descent from respected professional American football player and successful businessman to depressed drug addict. McHale was one of the first former football players diagnosed with chronic traumatic encephalopathy (CTE) – a neurodegenerative disease that causes memory loss, impulsive behaviour, depression and dementia.

He was not the last. CTE is rare in the general population, but it seems common among American football players: of the 94 posthumously tested at Boston University since 2008, 90 were diagnosed. It is also familiar in soldiers who served in Iraq and Afghanistan, apparently due to close proximity to explosions. CTE doesn't require severe head trauma, though. Repeated mild knocks seem to be enough.

So how can a series of minor blows to the head have such catastrophic consequences? Earlier this year, Matthew Campbell at Trinity College Dublin in Ireland came up with an answer: disruptions in the blood-brain barrier, a protective cellular seal around blood vessels in the brain (see main story). Campbell's team showed that sub-concussive blows can temporarily rupture this barrier, letting in all sorts of potentially damaging cells and molecules.

CTE remains mysterious, and the blood-brain barrier probably isn't the whole story. Even so, if we could design a drug to repair the cells of the blood-brain barrier after a blow to the head, we might be able to prevent CTE and other neurological injuries.

"just when you need the macrophages most", Schwartz says.

Intrigued, Schwartz decided to see what happens to these mice when you block the signals that suppress interferon gamma production and gum up the barrier. The result was a surge of macrophages crossing into the brain and a reduction in the number of amyloid plaques. What's more, the mice showed improvements in their symptoms. Schwartz's team is now working towards human clinical trials of the technique.

Whole new gateway

The notion that inflammation in the brain isn't always bad, and that encouraging it might help us to treat intractable neurological diseases, is radical. "It's like treating frostbite with ice – totally counter-intuitive," Schwarz says. But the idea received a big shot in the arm in 2015, when Kipnis, now at the University of Virginia in Charlottesville, and his colleagues discovered yet another gateway between the body's defence system and grey matter: an entire network of vessels directly linking the brain with the immune system via the lymphatic system.

This network of waste-disposal channels is how immune cells of every stripe get to every corner of the body, and how they pass signals back and forth. It was thought to stop at the brain – that was a big part of the reason we assumed the brain was immune privileged. An accidental discovery with some lab mice revealed that we just hadn't been looking in the right place, says Kipnis.

"One of my postdocs was taking out brains, and didn't do the step every neuroscientist does and peel the covering of the brain, the meninges. And we looked for immune cells and there were tons of them!" Later they found lymphatic vessels here too. This is like the trailhead of that system – T-cell patrollers can get that far, and then they send their cytokines on into the brain proper.

The discovery adds to the case that when it comes to immune system surveillance, the brain may not be so different to the rest of the body. It's just that, "in order not to disturb the neurons, the brain pushed all other activity to the fringes", says Kipnis.

His team is looking at how this connection between the brain and immune systems alters with age, and how that changes in Alzheimer's. One of the group's most interesting recent insights, however, involves social behaviour.

Following on from Schwartz's study showing that T-cell-deprived mice struggle cognitively, Kipnis has been unpicking the mechanism at play. His team has revealed that T-cells actually release the key cytokine interferon gamma, which neurons require for normal learning behaviour.

That study revealed something even more intriguing: that the adaptive immune system's access to the brain might be involved in how we behave towards other people.

> "This new strategy is like treating frostbite with ice: totally counter-intuitive"

"We showed mice without proper immune cells will be socially abnormal," says Kipnis.

Interferon gamma was responsible for this too, leading Kipnis to muse on an explanation. "If you think that bugs and pathogens drove our evolution, then in primordial species, for two creatures to become social maybe they had to be sure they could withstand pathogens from each other," he says. So perhaps when the brain doesn't get the interferon gamma signal that all's well with the immune system, the animals become less social.

The finding could have implications for autism and schizophrenia, Kipnis says: perhaps an immune system change is an underlying cause in some cases.

Either way, Kipnis is convinced the immune system is a normal part of healthy brain function. "The initial thought was, if we see immune cells in the brain they are part of the disease," he says. "I'm saying no, they are part of the solution. Of course the immune system can get out of control, but the aim is to make things better."

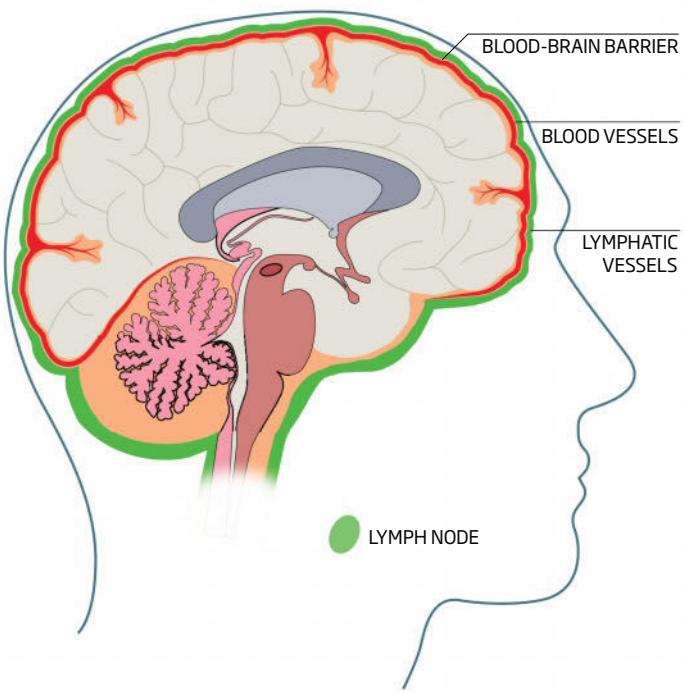
This suggests a whole new approach. "Neuroscience research today, unfortunately, is probably still 90 per cent oriented to neurons," Marchi says. "All the other cells are neglected. Considering that most brain disorders are of neurovascular origin, we need to start targeting more than just neurons."

Schwartz goes further. "You don't target treatment at the brain, you target the immune system, which does all the rest," she says. "It's a big, big turning point." ■

James Mitchell Crow is a science writer based in Melbourne, Australia

Hidden channels

The immune system is in constant contact with the brain via cells in the blood-brain barrier and a previously unknown network of lymphatic vessels



Not just a hound dog

Thousands of years of breeding make dogs perfect for delving into the genetics of personality, finds **Bob Holmes**

ELLIOTT ERWITT/MAGNUM PHOTOS

FIDO loves to cuddle on the sofa, Spot's a cheeky hound with a good eye for an unguarded slice of roast lamb, and Rover is just the best, most caring babysitter. As dog owners and lovers everywhere will tell you, pooches have personality. Some of that – quite a bit of it, in fact – is down to breeds. Terriers are notorious for their boundless energy. Border collies like to chase things. Pointers point. German shepherds and poodles learn quickly, bulldogs and beagles not so much.

All this makes dogs more than just great pets. They're also ideal subjects for geneticists eager to understand one of the great mysteries still remaining in biology: how complex behaviours are encoded in genes. If scientists can crack the code for dogs, we may be able to understand – and perhaps better manage – not only our loyal companions' behaviour, but our own as well.

Geneticists have known for decades that dogs are perfect for gene prospecting. The comparison may seem a touch unkind, but dog breeds aren't unlike strains of lab rats: each one is essentially an inbred line reared over generations to be nearly identical clones of each other. That makes both types of animal well suited to studies seeking to understand which gene does what. If you can identify subtle genetic differences between almost identical individuals, you can try to link those to variations in physical appearance, health or behaviour. It's a far cry from us humans, who tend to be very outbred and unsuited to genetic studies, which is why human genetic studies often focus on small, isolated populations such as Icelanders or the Amish.

Dog geneticists knew their day had come when new genetic tools came online in the early 2000s that made scanning entire genomes faster. This opened up the potential for revolutionary genetic searches called genome-wide association studies. The concept is simple: you compare the genomes of dogs with a particular trait – say, compulsive licking – to the genomes of dogs without it, and look for regions of DNA where the two groups differ. If a gene is involved in the trait, it will lie somewhere within these regions.

Personality is more than skin deep, in dogs as well as their masters

Back in 2008, Elaine Ostrander, a geneticist at the US National Human Genome Research Institute, and her colleagues did this with 148 dog breeds. The team searched for bits of the genome linked to breed-specific physical traits, and located several genetic variants that code for body shapes and sizes. They also included a few crude behavioural measures in their study: does the breed herd? Does it point? Is it highly trainable or resistant to teaching? Is it bold or timid?

The work kicked up some intriguing results. A number of genetic variants seemed to show up only with specific behaviours, indicating those behaviours were down to nature, not just nurture. For instance, one particular bit of DNA on chromosome 8 tended to be present only if the breed was a pointing one. This suggests that dogs that point are genetically programmed to do so.

Pointing wasn't the only behaviour for which the group found genetic signals. There were also markers associated with breeds that are skilled herders, ones that are easy to train and others that are particularly bold. The study showed that some behaviours could be traced back to genetics, but it was limited by its use of genetic markers known as SNPs. These are scattered more or less evenly across the genome, so finding one that is linked to a specific behaviour only tells you that a gene involved in the behaviour must lie nearby. Without being able to single the genes out, that was as far as the early studies could go.

That barrier has now fallen with the advent of faster, cheaper genome sequencing. Last year, a team led by Carlos Alvarez at Nationwide Children's Hospital in Columbus, Ohio, took pooch genetics one step further. The team used nearly 7000 questionnaires completed by dog owners about their pets' behaviour, especially fear and aggression. They selected the chilled-out breeds and ›

ETIENNE ANSOTTE/REX/SHUTTERSTOCK

TONY MENDOZA/GALLERYSTOCK. BOTTOM RIGHT: TESSA BUNNEY/MILLENNIUM IMAGES, UK

It's all in the genes: begging beagles and the classic yappy lapdog

compared their genomes to those of more aggressive breeds. Sure enough, they found several gene variants that increase the risk of fearful or aggressive behaviours. Two of those are active in the amygdala, a key brain region for processing fear.

Alvarez also found an explanation for the old cliché that small dogs are yappy and have more bite. Breeds like Dachshunds and Yorkshire terriers carry a version of at least one gene, *IGF1*, that makes them more aggressive towards their owners and other familiar dogs, and contributes to their diminutive stature. The questionnaire showed that small dogs carrying this variant of *IGF1* weren't more aggressive towards strangers, which suggests that it isn't just a case of them feeling more vulnerable. *IGF1* performs a range of physiological functions throughout the body, and Alvarez speculates that it may affect both size and behaviour, perhaps through different pathways.

The findings may have implications that go far beyond the vague annoyance of snappish lapdogs. "The genetics and physiology of fear and aggression are very ancient," says Alvarez, meaning they are shared by many different animals – including humans. If the same genetic pathways can be muted with drugs in humans, then snappy Spot's purebred lineage could end up shedding light on his owner's unfortunate tendency to fly off the handle.

Those conclusions are a bit of a stretch for now. Genes could have a big or a small role in human aggression – we don't yet know. Other "nurture" factors like upbringing and nutrition no doubt also play a part. But comparisons are already being drawn between some dog behaviours and human obsessive compulsive disorder.

"If you saw a dog with a compulsive disorder for 5 minutes, you wouldn't know there was anything wrong," says Elinor Karlsson, a geneticist at the University of Massachusetts Medical School. "But if you lived with it, you'd see that it did the same behaviour over and over for hours." Think hour upon hour of tail-chasing – bull terriers and German shepherds are particularly prone to this. Dobermans, meanwhile, compulsively suck their flanks and lick their paws. It can be quite distressing. A quick search online will kick up countless pictures of Dobermans that have licked themselves to the point of bleeding.

Dogs and their masters

Karlsson and her colleagues have identified four gene variants that occur far more often in dogs with compulsive behaviour than in their unaffected peers. All four play a part in communication between nerve cells, which makes sense for a trait involving behaviour. Three of the four also interact with genes implicated in human OCD. And at least one drug that treats the human condition, an antidepressant called clomipramine, also works in dogs – evidence that the shared genetic underpinnings can be clinically important.

That said, Karlsson warns that genes don't tell the whole story. "It's not like coat colour, where you get the gene and you get the colour," she says. Even dogs with the OCD-prone gene variants are only slightly more likely to develop compulsive disorders, so other factors are important as well.

Some of the unexplained variation in compulsive behaviour is probably the result of other genes whose importance isn't yet recognised. In 2016, a team led by Nick Dodman, a veterinary behaviourist at Tufts University in North Grafton, Massachusetts, dug a little deeper by comparing Dobermans with severe compulsive behaviour to others with milder forms. They found several other genes – some related to the neurotransmitter serotonin and others involved in stress pathways – that appear to modify the intensity of the behaviour.

The genetic approach is beginning to pay off even in more complex social behaviours. For example, Per Jensen and his colleagues at Linköping University in Sweden studied the willingness of dogs to solicit help from people to solve difficult problems. The team gave a group of beagles an impossible problem: a box containing a food treat, but with a lid that the dog couldn't open. Faced with this, most dogs make eye contact with a nearby human. Their closest wild relatives, wolves, show no such tendency, which suggests that this willingness to interact with humans may lie at the heart of domestication. Jensen's team measured how quickly each dog turned to a human, how close it approached and how often it solicited help, and then scanned their DNA for associated genes.

The experiment pinpointed variants of five genes that were linked to how vigorously the dogs sought help from humans. Changes in three of the five genes are linked to autism in

humans. This suggests that the propensity of Jensen's beagles to interact with humans could involve the same genes and pathways that can incline people with autism to avoid human interactions. "If that were true – and obviously, we have to look at this in other breeds and other contexts – then I think dogs may prove to be interesting models for autism disorders in humans," says Jensen.

Behaviours such as these are just the beginning. Dogs have another enormous advantage for behavioural biologists. "There are millions of pet dogs in the world, and they live with humans who spend a lot of time watching their behaviours," says Karlsson. So the pets don't need to undergo hours, weeks or months of tedious and costly observations.

Sequence your pet

At least two major research efforts are now beginning to tap into all that knowledge. Karlsson is spearheading one, known as Darwin's Dogs. Dog owners – more than 11,000 to date – volunteer to fill out detailed behavioural questionnaires on their pets, and some also provide genetic samples. A second programme, headed by Adam Boyko at Cornell University in Ithaca, New York, offers what is essentially a canine version of the personal-genomics service 23andMe. Dog owners pay Boyko's company, Embark, to analyse their pet's genome. They get a glimpse of its ancestry and genetic risk factors; Boyko gets the dog's genetic profile. In late 2016, Embark teamed up with Dognition, a company that uses games to measure dogs' cognitive skills. By combining cognitive and genetic profiles, Boyko and Dognition's Brian Hare plan to work out the genes behind intelligence, trainability and sociability.

Both Darwin's Dogs and the Embark/Dognition collaboration are in their infancy, but the researchers behind them expect rapid progress. "A year from now, or two years from now, we're going to have answers to some really big questions," says Hare.

The hope is that these projects can shine a light on how genes control traits we associate with personality – an idea science-fiction films have toyed with in the past. "If you were to ask me 10 years ago if it was doable, I would have said absolutely, positively no. No way," says Ostrander. "If you ask me now, I say absolutely, positively yes."

If Ostrander is right, what might the future hold? At the simplest level, knowing the genes that influence behaviour in dogs might help breeders, trainers and owners understand the best way to handle a particular dog. An unusually nervous one might need a gentler hand in training, or be a poor choice as a service dog.

Of course, you could argue that experienced trainers don't need fancy genetics to know which dog has a gentle or jumpy temperament. Could there be a more drastic use for all this information? Instead of making allowances for our pets' adorable but occasionally infuriating personalities, could we simply correct genetic "flaws"? A kennel owner in the US has already proposed doing this, albeit not for a behavioural trait. David Ishee suggests using CRISPR gene-editing technology to swap out a defective gene that causes severe urinary stones in Dalmatians. "If it could be done safely, why wouldn't you do it? It'll make the dogs healthier," says Karlsson. However, she notes, most behaviours are likely to be harder to modify in this way. For one thing, they often depend on several different genes, rather than just one. And each of those may have several unrelated functions – *IGF1* is a good example of this – so the likelihood of unwanted side effects is high. Finally, nurture is clearly important as well as nature: even a dog that was genetically modified to be less aggressive is likely to bite if mistreated. All this means that behavioural genetic modification may not yield enough benefit to be worth confronting the sticky ethical issues.

"DACHSHUNDS AND YORKSHIRE TERRIERS HAVE A GENE THAT MAKES THEM BOTH AGGRESSIVE AND SMALL"

In the long run, the biggest questions – and also the biggest ethical minefield – have to do not with our best friends but with ourselves. Do some humans share the same genetic predispositions to aggressive behaviour that Alvarez finds in dogs? If so, should society treat such people differently from their more placid kin, or even edit aggression out of future generations? The ethical issues this raises make dealing with dogs seem easy by comparison.

Sooner or later, we will need to grapple with these questions and our growing ability to meddle in genetic destiny. It may seem cruel, but if we can tackle them in dogs first, that might help. "We can't stop it," says Karlsson, "so I think the more we talk about it, the better off we are in the long run." ■

Go fetch was genetically selected over thousands of years

Bob Holmes is a consultant for *New Scientist* based in Edmonton, Canada

A great stripy puzzle

Matthew Cobb finds a true story more inspiring than any fiction

Zebra Stripes by Tim Caro, University of Chicago Press

ACCORDING to Rudyard Kipling's *Just So Stories*, the zebra got its stripes by standing half in the shade and half out, "with the slippery-slidy shadows of the trees" falling on its body. In *Zebra Stripes*, Tim Caro, a professor of wildlife biology at the University of California, Davis, sets out to test all the hypotheses explaining this most mysterious yet obvious phenomenon.

A bit of thought reveals the scale of the challenge: if having stripes is such a good thing, why do no other animals living on the African savannah, or non-zebra members of the horse family, *Equidae,* have stripes? Something, or some things, very specific to zebra ecology must have driven the evolution of this apparent adaptation, and will explain its absence in closely related species, and in species that share its niche.

The challenge isn't simply to come up with an explanation that "makes sense" (science would remain at *Just So* level were that the case) but to create predictions that flow from that explanation, and then to test them experimentally.

Caro lists dozens of theories, most of which boil down to five common factors: camouflage (protection from predators); warning coloration (zebras can bite); communication (social behaviours); temperature regulation (stripes may help resist the heat); and ectoparasites (biting flies might not like stripes).

Each of these and their many variants is explored in turn, as Caro describes how he tested them, and how virtually all – including the generally favoured camouflage theory – fail. In the end, the data converge on a single explanation: the role of biting flies (this is not a spoiler – the media pounced on it in 2014 after Caro's paper was published in *Nature Communications*).

FRANS LANTING/NATIONAL GEOGRAPHIC CREATIVE

As it turns out, the presence of stripes in zebras entirely overlaps with the distribution of certain species of biting fly. For reasons that are still unclear, flies dislike landing on striped surfaces. It is possible that the wiring of the fly eye means they cannot see stripes of some widths. Zebras may need to deter flies because their hair is shorter and thinner than that of other savannah animals so they are more susceptible to bites.

Something very specific to zebra ecology accounts for those stripes

Zebra Stripes isn't a piece of popular science writing, but an entrancing monograph that revels even in the minor details – from descriptions of how Caro used Photoshop to measure the stripes, through the number of paces he took in the bush wearing a zebra pelt to record how many insects settled on him, right down to the nitty-gritty of the statistical tests he used to make sense of his data.

This isn't in the slightest bit boring or hard to understand, as Caro's logical approach guides the reader. At every step, he pulls aside the curtain, revealing to the public exactly how science is done.

Caro's dogged exploration of every possible explanation, his determination to extract testable predictions and then compare those with reality, are all reminiscent of Charles Darwin in *On the Origin of Species*. Like *Origin,* Caro's book is also one long argument – not with the reader, but with the data, as he tussles with the annoying facts. In the end, the reader is compelled by the weight of evidence, which converges strongly on the flies.

"At every step, the book pulls aside the curtain, revealing to the public how science is done"

In the right hands, this book could change lives. Had I read something like this at 18, I would have tried to join Caro's research group, or at least have a career emulating his work. It's too late for me, but I predict that this marvellous book will encourage a new generation to get into the field and tackle evolutionary biology's remaining enigmas, with or without the help of Kipling. ■

Matthew Cobb is a zoologist at the University of Manchester in the UK

The power of useless

We desperately need knowledge for its own sake, says **Simon Ings**

The Usefulness of Useless Knowledge by Abraham Flexner, companion essay by Robbert Dijkgraaf, Princeton University Press

Knowledge for Sale: The neoliberal takeover of higher education by Lawrence Busch, MIT Press

SHELBY WHITE & LEON LEVY ARCHIVES CENTER, INSTITUTE FOR ADVANCED STUDY, PRINCETON

Luminaries at Princeton's Institute for Advanced Study in the 1930s

IN 1930, the US educator Abraham Flexner set up the Institute for Advanced Study, a research centre at Princeton University where leading lights as diverse as Albert Einstein and T. S. Eliot could pursue their studies, free from everyday pressures.

For Flexner, the world was richer than the imagination could conceive and wider than ambition could encompass. The universe was full of gifts and this was why pure, "blue sky" research could not help but turn up practical results now and again, of a sort quite impossible to plan for.

So, in his 1939 essay "The usefulness of useless knowledge", Flexner listed a few of the practical gains that have sprung from what we might, with care, term scholastic noodling. Electromagnetism was his favourite. We might add quantum physics.

Even as his institute opened its doors, the world's biggest planned economy, the Soviet Union, was conducting a grand and opposite experiment, harnessing all the sciences for their immediate utility and problem-solving ability.

During the cold war, the vast majority of Soviet scientists were reduced to mediocrity, given only sharply defined engineering problems to solve. Flexner's better-known affiliates, meanwhile, garnered reputations akin to those enjoyed by other mascots of Western intellectual liberty: abstract-expressionist artists and jazz musicians.

At a time when academia is once again under pressure to account for itself, Princeton's reprint of Flexner's essay is timely. Its preface, however, is another matter. Written by current institute director Robbert Dijkgraaf, it exposes our utterly instrumental times. For example, he employs junk metrics such as "more than half of all economic growth comes from innovation". What for Flexner was a rather sardonic nod to the bottom line, has become for Dijkgraaf the entire argument – as though "pure research" simply meant "long-term investment", and civic support came not from existential confidence and intellectual curiosity, but from scientists "sharing the latest discoveries and personal stories". So much for escaping quotidian demands.

"The structures throttling today's scholars come not from Soviet-style planning, but market principles"

We do not know what the tightening of funding for scientific research that has taken place over the past 40 years would have done for Flexner's own sense of noblesse oblige. But this we can be sure of: utilitarian approaches to higher education are dominant now, to the point of monopoly. The administrative burdens and stultifying oversight structures throttling today's scholars come not from Soviet-style central planning, but from the application of market principles – an irony that the sociologist Lawrence Busch explores exhaustively in his monograph *Knowledge for Sale*.

Busch explains how the first neo-liberal thinkers sought to prevent the rise of totalitarian regimes by replacing governance with markets. Those thinkers believed that markets were safer than governments because they were cybernetic and so corrected themselves. Right?

Wrong: Busch provides ghastly disproofs of this neo-liberal vision from within the hall of academe, from bad habits such as a focus on counting citations and publication output, through fraud, to existential crises such as the shift in the ideal of education from a public to a private good. But if our ingenious, post-war market solution to the totalitarian nightmare of the 1940s has itself turned out to be a great vampire squid wrapped around the face of humanity (as journalist Matt Taibbi once described investment bank Goldman Sachs), where have we left to go?

Flexner's solution requires from us a confidence that is hard to muster right now. We have to remember that the point of study is not to power, enable, de-glitch or otherwise save civilisation. The point of study is to create a civilisation worth saving. ■

Thar she blows

Meet the blue whale in all its glory, says **Sandrine Ceurstemont**

Out of the Depths: The blue whale story, Royal Ontario Museum, Toronto, Canada, to 4 September

WHEN Mark Engstrom arrived in Newfoundland to salvage a blue whale carcass, he was bracing himself for the epic stink characteristic of a decomposing whale. In fact, since this beast is the most massive animal that ever lived, "the smell was twice as big as usual", he says.

Coming across a blue whale carcass is rare because the animals usually sink when they die. But in April 2014, two washed ashore on the east coast of Canada. This allowed Engstrom and his team from the Royal Ontario Museum in Toronto to dismember one animal and study it. They carefully removed the bones to reconstruct the skeleton, and kept various parts like fat, baleen and an earwax plug – whose tree-like rings can be counted to determine age. "It took six days," says Engstrom.

Now a multisensory exhibition at the museum brings the animal back to life. It contains the world's most complete blue whale skeleton, as well as some of its supersized body parts, and a recreation of some of its unique traits. Visitors can even walk into a cast of the whale's jaws.

The show also delves into the mysteries the team is trying to solve. "We're interested in finding out how it has been changed by evolution," says team member Oliver Haddrath. Examining the whale's genes should provide clues. Haddrath has been working on sequencing its genome from DNA extracted from a fin, the best-preserved body part since it remained immersed in cold seawater.

The team hopes to answer questions, such as how the blue whale lost its teeth and colour vision. Whales see in black and white and are sensitive to blue light, which gives them better vision in the dim depths where they hunt the krill that make up almost all their diet. Visitors can experience what a krill swarm looks like to a whale via a new simulation, says Haddrath.

The genome should also help estimate the size of past populations. Blue whale numbers fell dramatically after commercial whaling took off in the early 1900s – producing oil and other products from blubber – but there are no solid figures. The genetic diversity of just a few individuals could shed light on those numbers, which in turn would help to reveal the fate of current populations. For example, "People are now saying [blue whales off the California coast] are back to pre-hunting numbers but if we don't know what those were, we can't make that claim," says Haddrath.

The blue whale's heart, which is the largest of any animal, is also enigmatic because few have been recovered before. Visitors can see the heart, immortalised using plastination techniques. After the show, its anatomy and function will be studied.

"Coming across a blue whale carcass is rare as the massive animals usually sink when they die"

The team was surprised to find that the heart didn't live up to rumours of being as big as a car. "It might fit inside a small car, but it's not the massive structure that we thought it was," says Jacqueline Miller, who worked to preserve it.

The hearts of most land mammals account for 1.5 per cent of their body mass. But from the team's initial calculations, the heart from their whale makes up about 0.25 per cent of the creature's body mass. If it is indeed the case, they are curious to find out why, as well as how it manages to function.

The team has also recreated the blue whale's sound, the loudest of any animal. In one room, a projection gives visitors the impression that a blue whale is swimming next to them while they feel vibrations from its calls, which are partly outside the range of human hearing.

And the experience wouldn't be complete without a whiff of dead whale. Engstrom thinks he has cracked it, but he's keeping the process secret. ■

Sandrine Ceurstemont is a writer based in Morocco

BRIAN BOYLE

Immortalised: the most complete blue whale ever goes on display

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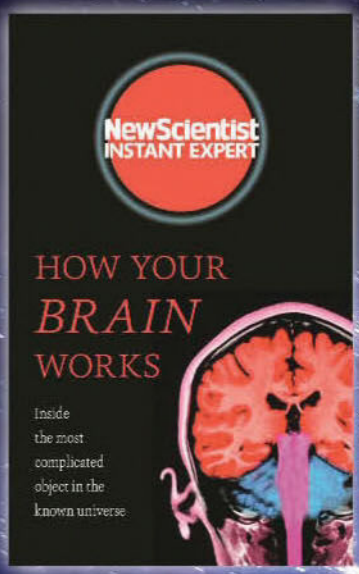

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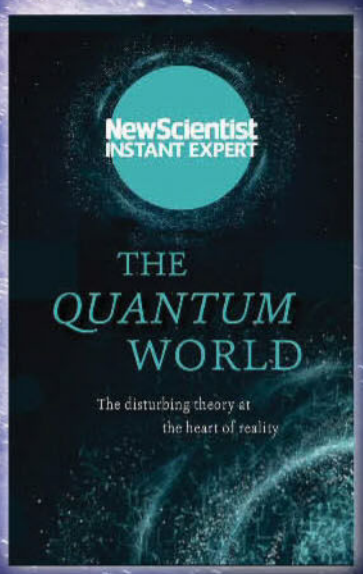

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Postdoctoral Position

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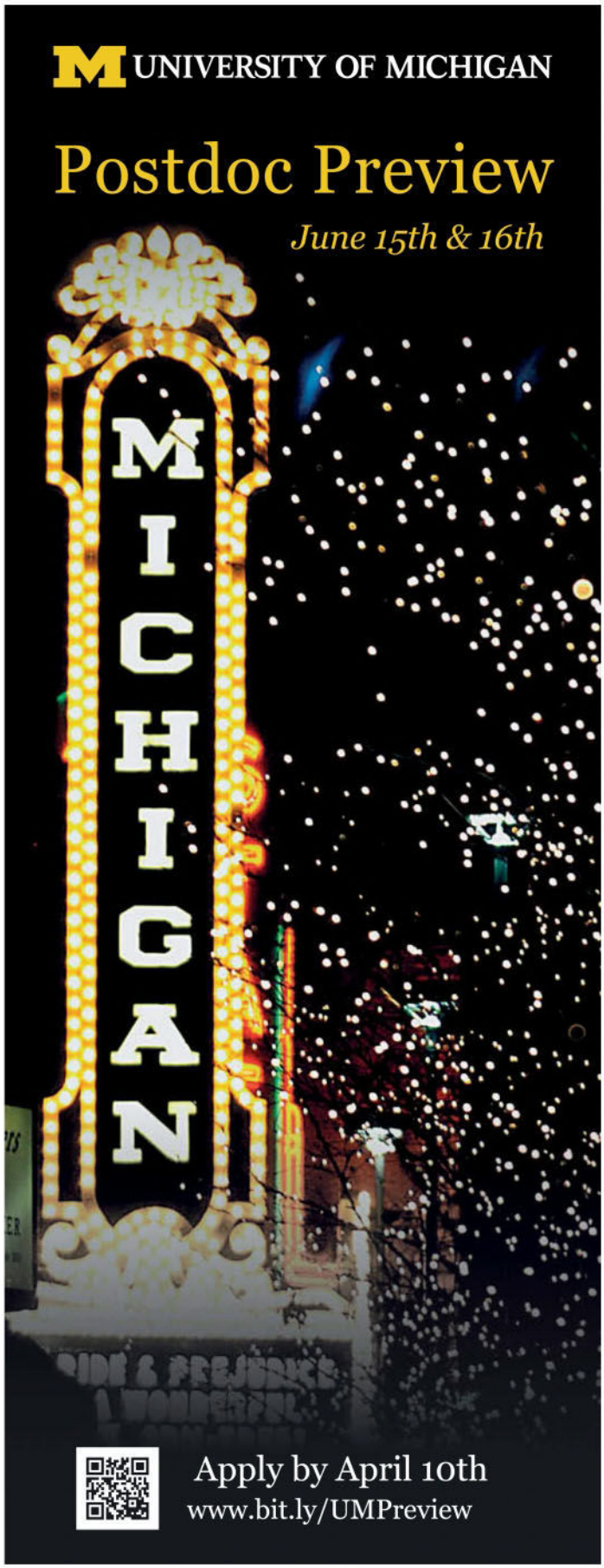

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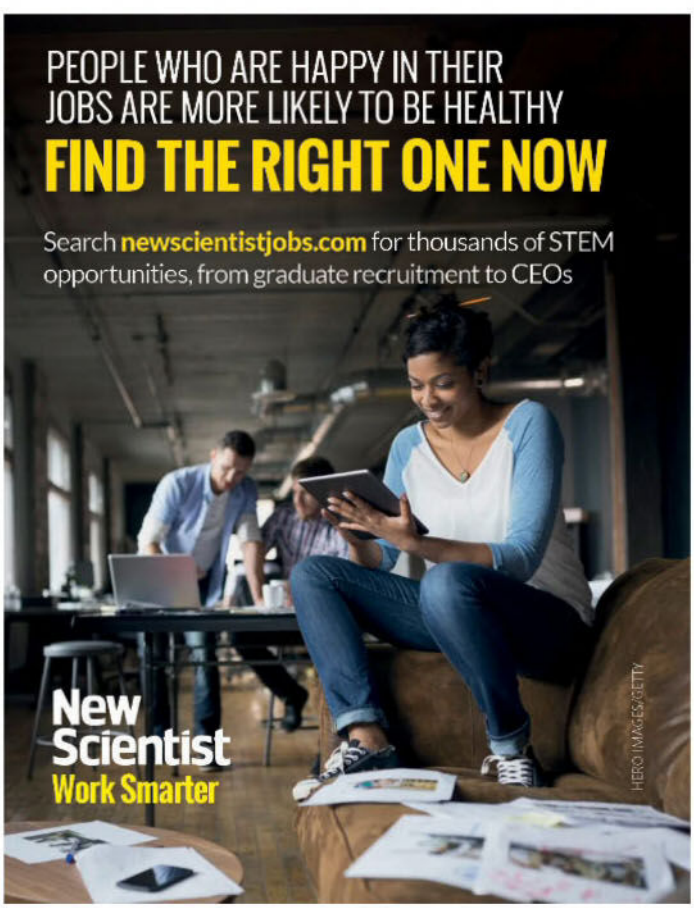

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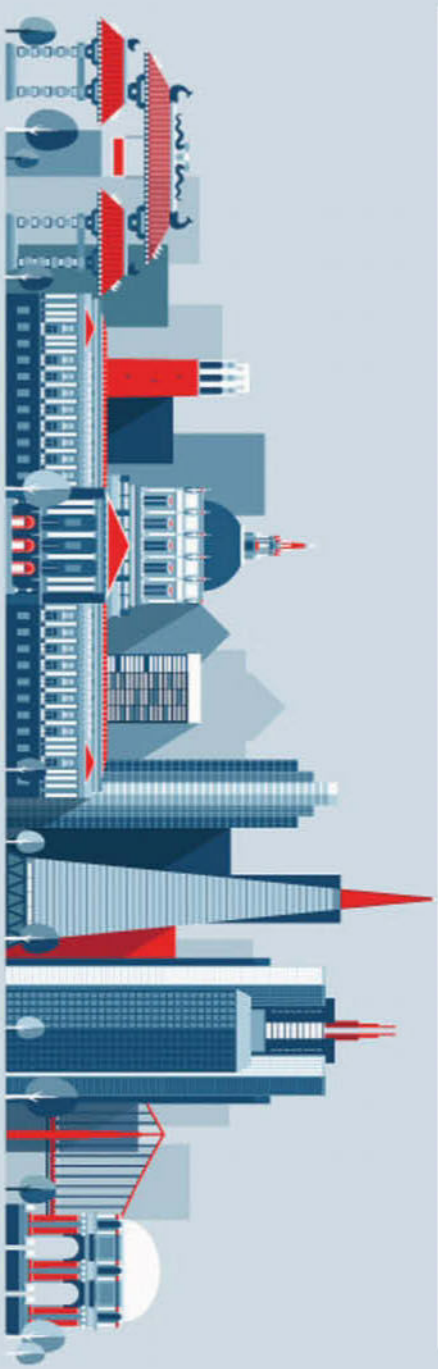

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EDITOR'S PICK

The perils of rescuing whales

From Geoffrey Patton, Silver Spring, Maryland, US
You report mass whale strandings (18 February, p 7). In the late 1980s, I headed the response to 20 to 30 pilot whales beaching in Fort Myers, Florida. We had an experienced team and a veterinary student trying to assess and evaluate animals in the shallows.

We decided to try to turn them all back to sea, got them moving and went for celebratory respite at a local eatery. There, we got another call that they had beached again not too far south.

We rushed there and, although the animals were obviously not in better shape, headed them back to sea. They restranded in the Florida Keys a day later and another team was called out to deal with them.

The repeated efforts were seen as wasteful and useless, although all we intended was to learn, collect data and improve such efforts in future. At the time, I lacked the public relations savvy to navigate the acrimony I received from managers and others in the field.

I still believe we need improved data collection on the animals' health and need to develop better hypotheses about why they strand.

When cuts come back to bite the cutter

From Michael Zehse, London, UK
You report encouraging news about the treatment of drug-resistant tuberculosis (18 February, p 6). Here in England, fewer new TB cases have been reported over the past five years.

But has our government thought through its policy of severely restricting access to the National Health Service by "foreigners"? This could lead to TB and other infectious diseases being propagated more widely.

Rescuing Pluto as a full-blown planet

From Laurel Kornfeld, Highland Park, New Jersey, US
Michael Brown attempts to impose one view of the status of Pluto as if it were fact (4 March, p 24). The planetary scientists who support Pluto's planet status are not, as he describes, "a small but vocal group". Neither did the 2006 decision he mentions to reject Pluto retaining its planet status involve a majority of astronomers. I believe only 4 per cent of the International Astronomical Union voted, and most weren't planetary scientists, but other types of astronomer.

Brown's claim that nothing about Pluto has changed since 2006 ignores the extensive findings of the New Horizons mission, which show Pluto to be a geologically living object with windblown dunes like those on Earth and Europa; flowing glaciers not seen anywhere else in the solar system besides Earth and Mars; tectonic forces; an internal heat source; cryovolcanoes; and even a possible subsurface ocean.

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"This is definitely not proof of intelligence!"

Dan Einon questions whether a bee that plays "golf" is something to be welcomed (4 March, p 20)

We can have a scientifically consistent definition of "planet" that includes numerous subcategories to account for both varying orbital dynamics and intrinsic properties.

Was Mars elsewhere when it had water?

From Shane Jones, Ipswich, Queensland, Australia

Chelsea Whyte discusses why water on Mars still doesn't make sense (18 February, p 25). Is it not possible that 3.5 to 4 billion years ago, when Mars was both warmer and wetter, the planet was orbiting closer to the sun?

The editor writes:

■ Several readers have asked this. Mars could indeed have been closer to the sun early in its history. But it must have been in its present orbit by about 4 billion years ago, when the water-carved features formed. After that time, there aren't any plausible mechanisms to move its orbit: the solar nebula would have long cleared out and really giant impacts would have ceased.

Humility in naming the Anthropocene period

From F. White, Nottingham, UK

Chelsea Whyte reports evidence of a new geological period – the Anthropocene – in minerals (4 March, p 11). But the comedic writer Douglas Adams in 1980 proposed that "advanced civilisations" would be marked in the geological record by a thin dark line he called the Shoe Horizon, composed of highly compressed trainers.

I fear we aren't living in a new period, but witnessing the climax of a 10,000-year event that will end the Holocene. We could justifiably name this event the Anthropic Horizon. Call the coming age, epoch or period the Anthropocene if you must, but we aren't quite there yet and shouldn't suppose that it is likely to greatly feature our species. A scintilla of humility is called for, and naming of the next age is best left to whatever conscious entity may inhabit it.

Cholesterol, statins, diet and heart health

From David Spence, London, Ontario, Canada

Michael Brooks quotes me saying that muscle problems from statins are more common in real-world practice than in clinical trials (11 February, p 28). Statins do cause muscle problems and in high doses slightly increase the risk of diabetes, but do not cause liver damage, nor the many other problems attributed to them.

While aching and cramps are fairly common (perhaps found in 25 per cent of patients), weakness and wasting of muscles is not. Often these can be avoided by reducing the dose of statin and adding ezetimibe, a drug that blocks absorption of cholesterol.

It is indeed complete nonsense to say it isn't proven that lowering cholesterol with statin drugs reduces the risk of heart attacks. It is also nonsense to say that lifestyle doesn't matter. Diet can reduce fasting cholesterol levels somewhat and can reduce the risk of heart attacks. However, it is true that cholesterol-lowering drugs are required to achieve the really low levels that markedly reduce coronary risk.

But the fasting level of blood cholesterol is not what diet is ›

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really about. The fasting cholesterol level is mainly produced by the liver overnight and is like a baseline – it tells us what arteries were exposed to for the last few hours of the night.

For about 4 hours after a high-fat or high-cholesterol meal, there is a marked rise in oxidative stress and the artery lining becomes sticky, twitchy and inflamed.

Statins lower the baseline level of cholesterol, but diet determines what happens on top of that for the 16 or so non-fasting hours. Both are important. One problem with studying the link between cholesterol, heart disease and statins in the US is that people's diets are so bad it is hard to show that anything is harmful. In 2015, the American Heart Association said that 0.1 per cent of Americans eat a healthy diet, and only 8 per cent a moderately healthy one.

Fighting infection and avoiding harm

From Jan Meulendijk, Walwyn's Castle, Pembrokeshire, UK

I sympathise with Jane Plumb and other parents who are confronted with an infection with Group B Strep in their newborn (Signal Boost, 18 February). I understand why they would favour the introduction of a test for the presence of Strep B in pregnant women.

But according to the UK Office for National Statistics, there were approximately 700,000 live births in the UK in 2014. Plumb says some 100 of those end fatally or in life-changing disability due to Group B Strep, or one in 7000. If a quarter of pregnant women carry the bacterium, and all those testing positive were offered treatment, almost 175,000 women and fetuses would be exposed to antibiotics they would not need, to prevent those 100 cases.

In the same issue, you report on a study hinting at long-lasting effects of antibiotics in newborn mice, including lung disease and a weakened immune system (p 20). The author of that study went on to question the blanket use of antibiotics in caesarean sections, where only about 200 must be treated to prevent one bad outcome. Considerations such as these make me oppose blanket antibiotic treatment where statistics indicate little benefit to women or babies in general.

Altruism is for life, not just that heroic dive

From Guy Cox, St Albans, New South Wales, Australia

Your leader article on "effective altruism" quotes philosopher Peter Singer as asking, since you would naturally save a drowning child even if you were wearing expensive clothes, why you would not donate the cost of the clothes to charity (25 February, p 5). This formulation omits an all-important factor: time.

In my 71 years, I have once saved a drowning child. I was clothed at the time. My son has saved one drowning child in his 33 years and was in the water already, so there was no sartorial cost.

Saving a child when clothed is likely to be, at most, a once-in a-lifetime happening. I would suggest that someone whose total lifetime donations to charity amounted only to the cost of a set of expensive clothes would be regarded as a veritable Scrooge.

My annual donations to charity well exceed my clothes budget, but then I am not a dressy person.

Is Trump playing clever after all?

From Bob Cory, Altrincham, Cheshire, UK

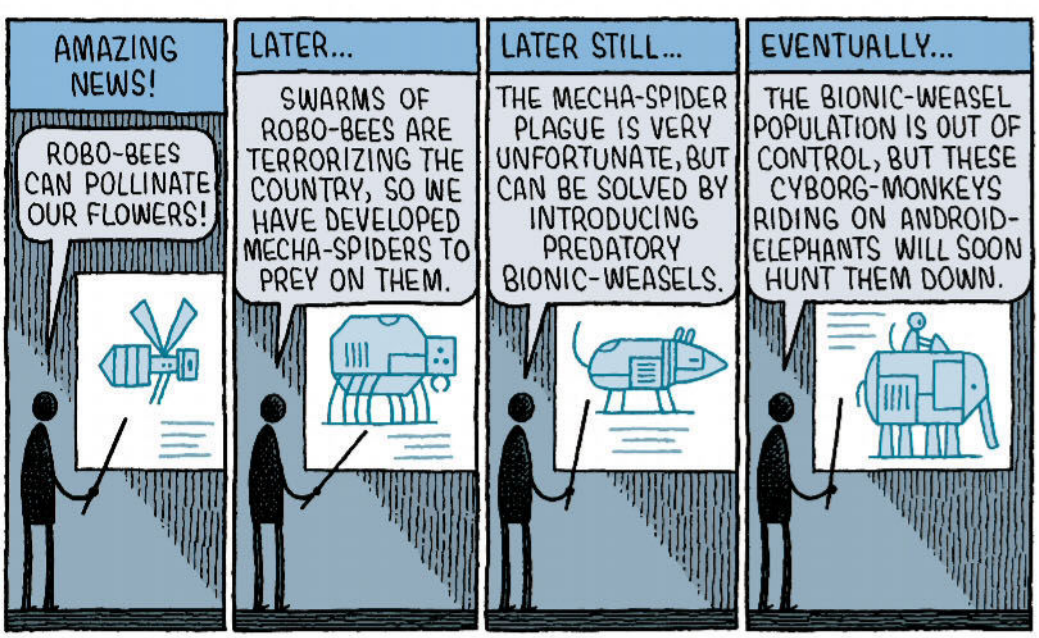
So Allen Frances thinks Donald Trump is stupid (25 February, p 24). If you read some of Trump's books you will see that his outrageous and unpredictable behaviour is a deliberate and calculated ploy to promote brand Trump and become US president.

Too little money to be worth spending on space

From Sam Edge, Ringwood, Hampshire, UK

The UK government is offering £10 million to promote UK-based space launches (25 February, p 7)? This is a waste of money because it is ridiculously little. Compare it with the expenses bill for the House of Commons, which runs to around £100 million a year.

TOM GAULD



For the record

- **Arsenic levels in water in the Quebrada Camarones region of Chile's Atacama desert exceed 1 milligram per litre (25 February, p 10).**
- **The ways in which the masked birch caterpillar signals include drumming with its mandibles and dragging its anal "oars" (4 March, p 19).**
- **Nerve agent VX would better be described as the most toxic synthetic chemical known (4 March, p 6).**
- **Larry Weiss is chief medical officer of US start-up AOBiome (4 March, p 22).**

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SIGNAL BOOST

Offering your projects a helping hand

An icy trek to beat bullying

EACH year, in England alone, bullying forces 16,000 children out of school. The charity Red Balloon works to combat this problem, to defeat bullying and help rebuild the self-esteem and confidence of as many children as possible who have become caught in its grip.

Six years ago, I was one of those children and I found myself unable to leave my house. A year later, I was in one of Red Balloon's Learner Centres. It changed my life.

In December 2015, I became the third youngest person to walk to the South Pole, which I did to raise money for Red Balloon. Although the expedition was a tremendous physiological challenge, with altitude, frostbite and weight loss to deal with, I can honestly say that the Antarctic was the most beautiful place I have ever seen.

Next month, I will be taking part in a substantially harder expedition. This time, I will be walking to the North Pole, again to raise money and awareness of Red Balloon and the problems it counters. The Arctic has all the dangers of the Antarctic along with some others of its own, such as polar bears and the risk of falling through cracks in the shifting sea ice. But if I complete this challenge, I will become, aged 18, the youngest person ever to walk to both poles.

I am doing this expedition partly to demonstrate that I am fully recovered from the compromised immune system I suffered as a result of being bullied. But mostly I am doing it to give something back to the charity that gave me back my life.

Last time, I raised £15,000, which is the bursary money for three children to attend a Red Balloon Learner Centre. This time around, I would like to raise the same again – or more if it can be done. **Bea Edwards, Red Balloon fundraiser**

To read more about the expeditions or if you are interested in helping a child get back their life and giving them the confidence to reach their full potential, visit **beansonline.blog**, or check out **www.redballoonlearner.org**

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PAUL MCDEVITT

ARE you sitting comfortably? If not, Feedback suggests splashing out on what could be your final resting place – if only because you'll have no money left for other furniture.

"I saw this beautiful urn and naturally I thought of you," writes Crispin Agar. "Apparently it contains the ashes of scientific reason." What is it, exactly? Feedback couldn't fit the item's full name as quoted on Amazon, an indigestible word salad of 25 fringe therapy buzzwords, but let's call it the Healthy Urn for short.

The blue and white lotus-flower-decorated "air bath" is a sort of one-person sauna you clamber inside to bathe in clouds of "nano anions" released by the gemstone-studded interior. Predictably, cosplaying as a giant sweaty vase delivers manifold benefits so far overlooked by medical science, including a strengthening of "biosome natural curability", aiding of the "seven human body systems", killing of "bad cells", prevention of ageing, and that perennial classic, improved circulation. Presumably, it will also uncrease your clothes.

While Feedback is tempted by the idea of a cosy capsule in which to flee the cares of the world, we think the £18,300 price tag might leave furrows in our brow that even a steam bath couldn't smooth out.

We note, finally, that the makers of the Healthy Urn mention its use for senility. We agree it could be, though as a diagnostic rather than a cure.

PREVIOUSLY Ed Prior worried about the diminishing superlatives available for ever-bigger radio telescopes, with the Very Large Telescope (VLT) in Chile soon to be overshadowed by the Extremely Large Telescope (ELT) (22 February).

Margaret Pitcher writes to point out that "there's always 'cyclopean', already used to suggest great size, and Cyclops did have one big eye."

Alan Carter, meanwhile, thinks that "perhaps in this era of Trump the answer should be the Bigly Large Telescope". As an added bonus, "it would share an abbreviation with a popular sandwich, which could help with sponsorship opportunities too."

Lastly, Paul King suggests that "the successor to the Extremely Large Telescope would obviously be the FET".

READERS will recall Feedback pondering a device offered by Echo Elemonics, which promised to add fizz to your life by infusing drinking water with hydrogen (18 February), and a London restaurant that was in the odd habit of adding imported seawater to their pizza dough (11 March).

Sifting through the flotsam of his own inbox, David Cox discovers something that combines both notions in *Internal Medicine Review* (not to be confused with the *Internal Medicine Journal*).

David forwards an abstract plucked from its pages, expounding the benefits of "functional water", that is to say water imbued with micronutrients and airy promises of well-being. Feedback was under the impression that water was rather functional already, but what do we know?

The abstract notes that functional water has already seized attention for its many health benefits, noting the liquid's capacity to "maintain or restore physiological self-regulation", which Feedback thinks could be another way of saying a couple of litres per day will keep you alive.

It also notes rather grandly that "formation of a super-coherent biological plasma-like physiological water is considered one of the most important characteristics of a healthy physiological state", which we think means that it's a good idea to be filled with blood if you want to stay healthy.

The abstract closes with an endorsement of drinking "deep ocean mineral water" for its health benefits, a recommendation that Feedback will politely decline – unless it comes wrapped in dough with melted cheese on top.

STAYING on the topic of fizzy water, David Wendt (and many others among you) writes to chide us for inadvertently coupling the hydrogen ions added by Echo Elemonics, describing them as hydrogen molecules. We promise less delinquent chemistry in future issues.

SOARING over the maple trees and red-blazered mounted police, John Carpenter discovers that the mineral water served to him on a recent Air Canada flight quotes a nitrate content of "<0". "It looks as though they have found a way to make matter disappear," says John.

Perhaps this is a dual use technology? Feedback notes that nitrates are an important component of explosives – just the sort of thing you might need to neutralise in a hurry on a plane. In which case, this Canadian mineral water sounds like the one liquid you should be allowed in your carry-on luggage.

YES to paddock survey, announces the *Newark Advertiser*, as amateur archaeologists are given the go-ahead to study Warner's Paddock, believed to be in "the oldest part

of the town" of Bingham, UK. The newspaper reports that the survey will be performed by "carrying an imagining tool across every part of the field".

Waiting with bated breath for the results is Peter Duffell, who thinks "The sky is the limit. Perhaps they can imagine that they will find Lord Lucan, Atlantis or any manner of things."

"Nikole Lewis… announces the discovery of seven Earth-like planets orbiting a nearby star at NASA Headquarters in Washington, DC," writes the UK's *Daily Telegraph*. "They didn't have to look far," muses Crispin Piney

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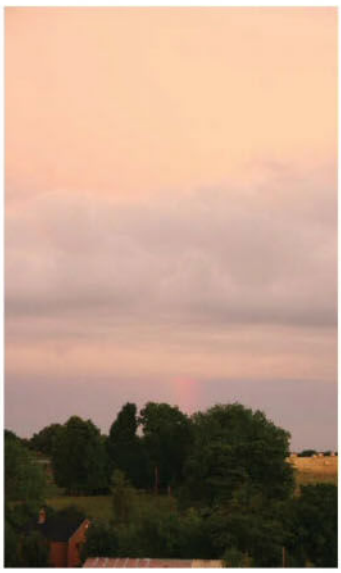
Sunset rainbow

I took the first of these two photos not long after the local sunset time of 9.02 pm on Stanley Common in Derbyshire, UK, on 17 July 2014. The second was taken at 9.22 pm, a few minutes before the last trace of the rainbow evaporated. In the first photo, there is a cloud in front of the rainbow. In the second, the rainbow has persisted after the sun had gone down. How did this occur and is it possible to calculate the height of the rain clouds causing the rainbow from the time difference I've given?



■ For this place and date, a sunset calculator actually gives a sunset time of 9.22 pm, not 9.02 pm. Even if local topography affected the exact timing, it is not surprising that the rainbow was still visible at 9.22 pm, but disappeared a few minutes later.

A rainbow is produced by refraction and reflections inside

"Rain clouds are too thick to let sunlight through easily, so do not contribute to a rainbow"

droplets of water, such as raindrops or mist droplets left in the air after rain. The rainbow appears in the sky directly opposite the sun, and its light at each point is an accumulation of all contributing droplets in that line of sight. A rainbow therefore has no specific position in terms of distance from the observer: any droplet in the air anywhere between the observer and the far distance (in the opposite direction to the sun) can add to the effect if it happens to be receiving sunlight.

A limiting factor is that rain clouds, although consisting of condensed water drops, are too thick to let sunlight through easily. As a result, they don't contribute to a rainbow and also block any rainbow light originating behind them.

Regarding the first photo, part of the rainbow does indeed seem to be missing where a particular cloud is located. If there is no trace of rainbow in front of this cloud, it suggests that all the water droplets producing the rainbow are located further away than this cloud.

But none of this has any bearing on the actual distance to this cloud or to the heights of any of the rain clouds, so these cannot be calculated.

Richard Swifte
Darmstadt-Eberstadt, Germany

■ If you were looking towards the sun, it is very possible that it wasn't a rainbow you were seeing. There are a number of atmospheric effects that look a little like rainbows. Here, you may instead be seeing iridescence, which is the diffraction of

"Some atmospheric effects look a little like rainbows. It is possible you are seeing iridescence or a halo"

sunlight by altocumulus clouds high in the atmosphere.

Alternatively, you may have been seeing a halo, which is a similar diffraction effect caused by ice crystals in cirrus and cirrostratus clouds.

Lewis O'Shaughnessy
Oxford, UK

This week's questions

OUT OF THE BLUE

The sky is blue because the atmosphere scatters the shorter wavelength (blue) end of the visible light spectrum more than the red end. As a result, more of this reaches the ground when the sun is high in the sky. Why then aren't we aware of being bathed in a bluish light? Or do we in fact see a bluer world, but, because we always see it this way, our senses accept it as neutral? And is that why a tinge of blue is often added, for instance to washing detergent, to make things look whiter?

Philip Cunliffe
Bristol, UK

FOUL-MOUTHED RESPONSE

Given the importance of the microbiota in our gut for well-being, is antibacterial mouthwash a good idea?

Grainne Collins
Dublin, Ireland

FLAT ABOUT FIZZ

Why does my wife like wine with bubbles more than flat wine? Do the bubbles add to the taste or does her preference come from marketing and cultural context? And why do I not like fizzy wine?

Nick Beale
Newcastle upon Tyne, UK

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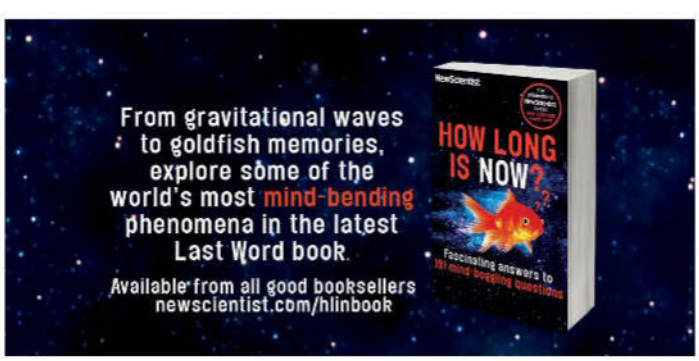


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