

<http://bit.ly/2fh9kkK>

Changes in Earth's crust caused oxygen to fill the atmosphere

"Oxygenation was waiting to happen, all it may have needed was for the continents to mature."

Scientists have long wondered how Earth's atmosphere filled with oxygen. UBC geologist Matthijs Smit and research partner Klaus Mezger may have found the answer in continental rocks that are billions of years old. "Oxygenation was waiting to happen," said Smit. "All it may have needed was for the continents to mature."

Earth's early atmosphere and oceans were devoid of free oxygen, even though tiny cyanobacteria were producing the gas as a byproduct of photosynthesis. Free oxygen is oxygen that isn't combined with other elements such as carbon or nitrogen, and aerobic organisms need it to live. A change occurred about three billion years ago, when small regions containing free oxygen began to appear in the oceans. Then, about 2.4 billion years ago, oxygen in the atmosphere suddenly increased by about 10,000 times in just 200 million years. This period, known as the Great Oxidation Event, changed chemical reactions on the surface of the Earth completely.

Smit, a professor in UBC's department of earth, ocean & atmospheric sciences, and colleague, professor Klaus Mezger of the University of Bern, were aware that the composition of continents also changed during this period. They set out to find a link, looking closely at records detailing the geochemistry of shales and igneous rock types from around the world -- more than 48,000 rocks dating back billions of years.

"It turned out that a staggering change occurred in the composition of continents at the same time free oxygen was starting to accumulate in the oceans," Smit said.

Before oxygenation, continents were composed of rocks rich in magnesium and low in silica - similar to what can be found today in places like Iceland and the Faroe Islands. But more importantly, those

rocks contained a mineral called olivine. When olivine comes into contact with water, it initiates chemical reactions that consume oxygen and lock it up. That is likely what happened to the oxygen produced by cyanobacteria early in Earth's history.

However, as the continental crust evolved to a composition more like today's, olivine virtually disappeared. Without that mineral to react with water and consume oxygen, the gas was finally allowed to accumulate. Oceans eventually became saturated, and oxygen crossed into the atmosphere.

"It really appears to have been the starting point for life diversification as we know it," Smit said. "After that change, the Earth became much more habitable and suitable for the evolution of complex life, but that needed some trigger mechanism, and that's what we may have found." As for what caused the composition of continents to change, that is the subject of ongoing study. Smit notes that modern plate tectonics began at around the same time, and many scientists theorize that there is a connection.

Smit and Mezger [published their findings today in the journal Nature Geoscience](#). The research was funded by the Natural Sciences and Engineering Research Council.

<http://bit.ly/2y9ySnO>

Sex and aggression controlled separately in female animal brains, but overlap in male brains

Brain structures that control sexual and aggressive behavior in mice are wired differently in females than in males.

This the finding of a study led by scientists at NYU School of Medicine and published online Sept. 18 in Nature Neuroscience.

Specifically, researchers found that, while control of aggressive behavior resides in same brain region in female and male mice, certain groups of brain cells in that region are organized differently.

Two separate groups of cells were found to control sex and aggression in females, whereas circuits that encourage sex and aggression in males overlapped, say the study authors. Knowing how aggressive behaviors are regulated is important because they are essential to

survival in mice, as well as in humans, which have evolved to compete for food, mates, and territory, researchers say.

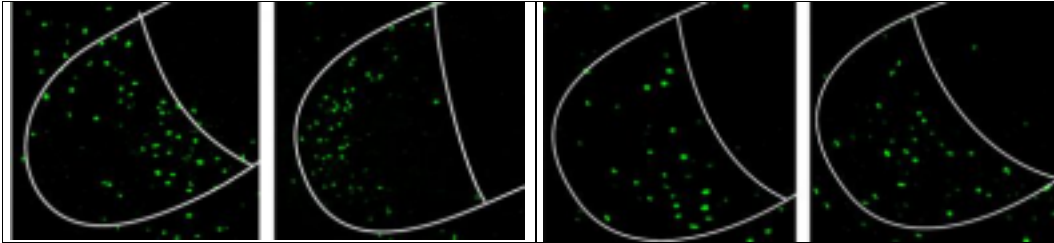


Image of female mouse brain shows different cells (in green) activated in different regions of the ventrolateral part of the ventromedial hypothalamus, or VMHvl, when the mouse is fighting (left) and mating. Courtesy of Nature Neuroscience

Image of male mouse brain shows cells (in green) activated in the same region of the of the ventrolateral part of the ventromedial hypothalamus, or VMHvl, when the mouse is fighting (left) and mating. Courtesy of Nature Neuroscience

"Our study furthers our understanding of how these behaviors are organized differently in female and male mice brains," says study senior investigator Dayu Lin, PhD, an assistant professor at the Neuroscience Institute at NYU Langone Health.

Having a detailed breakdown of brain functions by gender, Lin adds, is a "fundamental step" toward any future attempt to develop drugs that suppress extreme aggression in humans. She says research on female aggressive behavior has lagged because, in most animals, males are the more aggressive gender.

Original research by Lin and her team, published in *Nature* in 2011, was among the first to trace the origins of male aggression in mice to a distinct part of the hypothalamus, the brain region that controls body temperature, hunger, sleep, and levels of many hormones. This key part, the ventrolateral part of the ventromedial hypothalamus, or VMHvl, is located on the underside of the hypothalamus in mice and humans.

Other, recent studies that had blocked the action VMHvl cells in female mouse brains failed to trace the source of aggression control to

the VMHvl. This blockade did stop all male and female attempts to mate, but did not reduce fighting among females, says Lin.

She says these other studies -- having used a mouse type known for timidity -- did not accurately replicate natural conditions. The current study was made more realistic by including a naturally aggressive mouse strain, as well as female virgin mice eager to fight off competitors for food, and new mothers anxious to protect their pups, say the authors.

The current study monitored the brain activity of the virgin and mother mice during fights with any female or male mouse that had entered their boxed space. The brains of all study mice were tested with electrical, genetic, or chemical probes that measured which nerve cells were turned on or off, and how this affected their fighting behavior. Researchers found that, under these more realistic conditions, turning VMHvl cells on or off did control whether female mice would fight. Researchers also monitored in males and females the activity of individual cells in the VMHvl with proteins that enable them to interact with the sex hormones (e.g. estrogen receptor alpha). Such cells had previously been linked to fighting behaviors in male mice.

Experiments showed that VMHvl cells actively transmitting signals, or "firing," while females were mating were not the same cells firing when they were fighting. But in male mice, many of the same cells were firing during both activities. Further analysis showed that males had a mixed spatial distribution of the VMHvl cells involved in either behavior. In females, the cells involved in fighting were arranged along the center of the VMHvl, while those involved in mating were distributed along its borders.

Lin says her laboratory next plans to fine-tune tools for experimenting separately on the female mating and fighting VMHvl cells. She says her team also has plans to investigate the biological origins of these cells to determine how the female nerve circuitry develops in contrast to the circuitry in males.

Funding support for the studies was provided by National Institute of Mental Health (NIMH) grants R01 MH101377 and R21 MH105774. Additional funding support was provided by the Mathers Foundation, Irma T. Hirschl Trust, Ester A. and Joseph Klingenstein Fund, Whitehall Foundation, Sloan Foundation, and McKnight Foundation.

Besides Lin, other NYU Langone scientists involved in this research were co-lead investigators Koichi Hashikawa, PhD, and Yoshiko Hashikawa, PhD; Robin Tremblay, PhD; James Feng, PhD; Alexander Sobol, PhD; Walter Piper, PhD; and Bernardo Rudy, MD, PhD. Additional research support was provided by Jiaying Zhang, PhD, at Xiamen University in China; and by Hyosang Lee, PhD, at the Daegu Gyeongbuk Institute of Science and Technology in Korea.

<http://bit.ly/2jFUyfe>

Eight children born after uterus transplants

Eight children born - and the first robot-assisted operation performed. These are some of the results of 18 years of research at Sahlgrenska Academy on uterus transplants.

In Gothenburg, the elite of the research world in the field are now gathering for their first congress.

In three years, from September 2014 to today, eight children in the world have been born to mothers who had fertilized eggs returned after undergoing a uterus transplant. All of this has taken place in the scope of the research conducted at Sahlgrenska Academy since 1999.

The first birth enjoyed international attention. When children seven and eight came into the world one week apart this past summer, the framing was considerably calmer. For one of the mothers, it was her second child; she had undergone two pregnancies with the same donated uterus.

In a new project, the researchers in Gothenburg are now focusing on robot-assisted operations. The objective is to more easily handle the challenge of operating inside the woman's bowl-shaped pelvis. The basic technology is the same as in some cancer operations, such as cervical cancer operations.

"The hypothesis in our research is that we can do it significantly faster this way and with an earlier return home for the patients," says Mats Brännström, Professor of Obstetrics and Gynecology at Sahlgrenska Academy and Senior Physician at Sahlgrenska University Hospital.

One operation took place in May, four await in October and December and the remaining five will be done in coming years. Just like before, the attempts to make the women pregnant begin one year after they have received the transplanted uterus when the situation regarding medication against rejection has stabilized.

Besides the robot-assisted operations, one of which was also done in China, donations from deceased donors is also an issue of current interest. Not for the Swedish researchers, here it has always been about living and related donors, but the ideas are being considered elsewhere in the world.

Mats Brännström describes uterus transplants as an internationally growing field with a need for a common compulsory register of all procedures done in order for the researchers to get an overview. The register will be an important issue when the year-old organization, International Society for Uterus Transplantation (ISUTx) holds its first congress, until September 19.

Mats Brännström believes that uterus transplants will also be done outside the research sphere in a few years. He is well aware of the discussion of priorities in healthcare and actually does not want to have opinions on matters other than the medical.

"In the future, this method will become even more effective, and a clinical reality. We don't know if this will be in Sweden. Medically, it's fully realistic in five years, but there are many other decisions we have no control over," he says.

Facts About Uterus Transplants

Uterine infertility is the kind of female infertility that has had no treatment. More than 200,000 women in Europe are estimated to have uterine infertility. The first transplant attempt with a living donor was made in Saudi Arabia in 2000. The uterus had to be removed shortly after the procedure. In 2011, a transplant was done in Turkey with a uterus from a brain-dead patient. Several embryo return attempts were reported. Two early pregnancies ended in miscarriage bleeding. No other pregnancy has been reported

Operations 2-11 in the world with living donors were done at Sahlgrenska Academy in the scope of the world's first systematic and scientifically based study. In 2014, the first child was born.

In a new research project, the possibilities of robot-assisted operations are being studied. The project comprises ten operations of which five will be this year.

Donors, recipients, partners and children are monitored for a long time both medically, psychologically and from a quality of life perspective. Professor Mats Brännström estimates that in recent years, there have been up to 20 more operations in the world. The idea is that the ISUTx congress and an international register will contribute to a better overview and to a scientific way to drive the field of research forward with the aim of increasing safety for patients and the effectiveness of the treatment. *More information:* <http://www.isutx.org>

<http://bit.ly/2ym4sQ8>

Beta blockers not needed after heart attack if other medications taken

UNC-Chapel Hill study first to challenge current clinical guidelines

Chapel Hill, N.C. - A new study from the University of North Carolina at Chapel Hill finds beta blockers are not needed after a heart attack if heart-attack survivors are taking ACE inhibitors and statins. The study is the first to challenge the current clinical guideline that heart-attack survivors should take all three drugs - beta blockers, ACE inhibitors and statins - for the rest of their lives.

Heart-attack survivors are usually prescribed all three drugs to help prevent a second attack and death. However, the beta blockers offer no additional benefit for patients who take the other two drugs as prescribed, according to the new study, which examined the trade-offs and consequences of using some of the medicines instead of others. The findings were published today in the Journal of the American College of Cardiology.

Researchers looked at more than 90,000 Medicare patients age 65 or older who had suffered a heart attack and were prescribed a beta

blocker, ACE inhibitor or angiotensin receptor blocker and statin as preventive therapies after they were discharged from the hospital. Patients who only took the ACE inhibitor or an angiotensin receptor blocker and statin, as prescribed, were no more likely to die than those who took all three drugs.

The research team from UNC-Chapel Hill, Monash University, the University of Iowa and the University of Eastern Finland was led by Gang Fang, an assistant professor at the UNC Eshelman School of Pharmacy and senior author of the study.

Fang stressed that patients should not stop taking beta blockers or any other prescription medicine without first consulting their physician. "We are not saying that beta blockers have no value. It's just that their benefits appear to have been eclipsed by the duo of ACE inhibitors and statins, which are relatively newer drugs," Fang said.

Beta blockers were introduced more than 50 years ago and reduce blood pressure and heart rate. ACE inhibitors and angiotensin receptor blockers also reduce blood pressure and they have been around approximately 40 years. Statins reduce the amount of cholesterol and other fats in the bloodstream and have been in use for more than 30 years. For heart-attack patients, these drugs provide additional support to the heart.

For six months Fang's team followed heart-attack survivors who filled prescriptions for all three drugs to study how well they adhered to their prescription drug regimen. Being adherent was defined as taking the medicines as prescribed at least 80 percent of the time. The team then followed the patients for up to 18 months to see how many died during that time. Six months after their heart attack about half the patients in the study had stopped taking at least one of their medications as prescribed, the researchers found.

For patients who took all three drugs as prescribed, the mortality rate at one year was 9.3 percent. For patients who adhered to ACE inhibitor or ARBs and statin prescriptions but not beta blockers, the mortality rate was 9.1 percent, a statistically insignificant difference.

For patients not taking any of the medicines as prescribed, the mortality rate was 14.3 percent, a nearly 54 percent increase over adherent patients.

"The problem with this three-drug regimen is that it is difficult for people to take their medications as they are supposed to in the long term. This is especially true of older patients who are likely to already be taking many different drugs," Fang said.

Fang also noted that patients in the study who had diabetes, dementia or both were more likely to die when taking beta blockers as prescribed. Further research is warranted, he said, and physicians should exercise more caution in prescribing beta blockers for elderly heart-attack survivors with diabetes or dementia.

<http://bit.ly/2fvymJN>

This Area of the Brain May Explain a Link Between Poor Sleep and Depression

People whose brains respond strongly to rewards may be less prone to some of the negative effects of sleep deprivation, a new study finds.

By Sara G. Miller, Staff Writer | September 18, 2017 02:55pm ET

One of those negative effects is depression, according to the study. Poor sleep has been linked to depression, both as a risk factor for the mental health disorder and as a symptom of it.

But not every person with sleep troubles has symptoms of depression, according to the study, published today (Sept. 18) in the Journal of Neuroscience.

Instead, differences in how people respond to positive experiences seem to have a "small but notable protective role" in stopping symptoms of depression from happening alongside sleep problems, said senior study author Ahmad Hariri, a professor of psychology and neuroscience at Duke University.

In other words, people in the study who didn't sleep well but whose brain activity lit up in response to a reward were less likely to also have symptoms of depression than people who didn't sleep well and whose brain activity didn't light up as much in response to a reward.

"The extent to which you have a brain that's sensitive to reward and responses to rewarding experiences buffers against ... the association between poor sleep and depression," Hariri told Live Science.

To study how the brain's response to rewards played a role in the link between poor sleep and depression, the researchers did brain scans on more than 1,100 college students. Before having their brains scanned, the students filled out questionnaires about how well they sleep at night and their mood.

During the brain scans, the students were asked to play a simple game so that the researchers could measure activity in an area of the brain called the ventral striatum, which is related to rewards. The ventral striatum acts as "the hub of reward learning," Hariri said; it's designed to learn what behaviors result in rewards and reinforce those behaviors. In the game, which took about 6 minutes to play, the researchers asked the students to guess whether the number on a playing card was higher or lower than 5, and the students were told that the better they did, the more money they'd get. For each correct answer, the student would get positive feedback - "Hey, your guess was right!" - and the researchers would see how the ventral striatum responded. (Similarly, for wrong answers, the students would get negative feedback.) Unbeknownst to the students, the game was rigged - so during six rounds of the game, they would be right 80 percent of the time or wrong 80 percent of the time.

The researchers found that the extent to which a person's brain responded to a reward (or being told they got the right answer), the less likely that person would be to show an association between poor sleep and symptoms of depression, Hariri said.

Other studies have shown that the ventral striatum plays a role in a range of symptoms of depression called "anhedonia," Hariri said. Anhedonia is basically "a fancy word for lack of pleasure," he said, and refers to symptoms such as not finding activities that were once rewarding to be pleasurable anymore, blunted emotions and a lack of motivation to participate in once-rewarding activities.

Interestingly, in the new study, the researchers found that activity in this area of the brain was linked to all symptoms of depression, not just anhedonia symptoms, Hariri said.

However, the study had some limitations, Hariri noted. For example, the researchers looked at only a snapshot in time. "What we don't have is that order of how things are happening," he said. It's unclear if a person's poor sleep came before or after symptoms of depression in the study, he said. Long-running studies are needed to confirm the findings.

Another limitation was that the research was done in relatively healthy young adults, so it's unclear if the findings would apply to older and less-healthy people.

<http://bit.ly/2xtmS3q>

Why One Med School Embraces DACA Students

The president of the Class of 2020 explains that their inclusion is part of an overarching mission to foster a community that represents all of America

I watched news coverage of the 2016 presidential election results sitting beside my roommate, an undocumented medical student attending the Loyola University Chicago Stritch School of Medicine. Today, with DACA recently being rescinded, and the future of my undocumented colleagues in jeopardy, I have a much more intimate understanding of the fear on his face that evening in November.

Like many undocumented youth, my roommate only learned of his immigration status when it came time to apply for college. He grew up in our society, was educated by our public school system where he pledged allegiance to our flag, and cherishes American values as much as anyone I know. It occurs to me now, as he faces the threat of being sent "back" to his country of birth, Thailand, that this scenario is as ridiculous as me being sent to some arbitrary country that I don't know—just as I'm getting ready to dedicate a lifelong career to the betterment of our nation's health.

Stritch was the first U.S. medical school to openly accept applications from DACA recipients, and is now home to more undocumented students than any other medical school in the country. As president of the Loyola Stritch Class of 2020, I feel compelled to speak out in support of our undocumented colleagues in light of recent attacks on their character, their right to a future as Americans and their ability to contribute substantially to the medical profession.

Our DACA classmates are among the most resilient people I have ever met. Somehow, they continue to excel in a grueling medical curriculum despite the daily demonization of their status in our national political conversation. Their perseverance is not limited to dedication in the classroom and unwavering compassion in the clinic. For example, my roommate spent this summer working at a prestigious primary care internship just to send money back to his parents in central California.

Reminding folks that "dreamers" are often capable, driven individuals should be superfluous. The only thing separating my roommate and me—two young people who have only known the U.S. as our home, who wish to contribute to this society and its ideals—is a piece of paper. We shouldn't decide whether to embrace the youth of our country, documented or undocumented, based on that piece of paper. These are human beings who stand for the same things we do regardless of vocation or the perceived "value" associated with their skill sets.

But just in case some readers are still not convinced, I will make the case for undocumented physicians. One thing most Americans can agree on, even in 2017, is that our health care system has plenty of room for improvement. The dramatic shortage of providers in underserved communities is one area of focus. American medical schools, through their role as the suppliers of physicians, are responsible for assessing this shortage and addressing it responsibly. With an aging U.S. population, the gaping hole in the primary care

workforce caused by medical students increasingly opting for higher-paying specialties and subspecialties will only get worse.

To respond to this shortage, and to simultaneously address historic inequities that have led to underrepresentation of minority groups, medical schools have shifted toward mission-based admissions initiatives that weigh more than mere GPA and standardized test scores. At Loyola, a Jesuit institution, this mission is founded on the principles of social justice and service to others. This is demonstrated in Loyola's decision to build its flagship health center (ranked number three in the state) in the underserved western suburbs of Chicago. Our "Access to Care" clinic less than a mile from our school's campus provides free health care to the local uninsured population, including individuals who are undocumented.

Health equity is further promoted by recruiting medical students from diverse backgrounds. Not only does this work to address gaps in representation for minority physicians, it also serves to improve health outcomes in underserved communities; data has shown repeatedly that underrepresented minorities in medicine go on to practice in underserved communities at higher rates. Undocumented immigrants, who live in society's shadows without access to health care, financial freedoms or the personal agency of having a driver's license, for example, are among the most disenfranchised groups in today's America.

Stritch began admitting undocumented students in 2014, in recognition of the medical profession's duty to benefit all people. Their presence will not only result in a broader reach of patients for our graduating class but is a key component of our professional development. When we confront differing identities and perspectives, we actively build empathy—perhaps the most vital quality of any good doctor.

It's important to note that inclusion of undocumented students is part of an overarching mission to foster a community of students that represents all of America—and our admissions team was remarkably

successful in this effort for the class of 2020. Being "the most diverse class in school history" doesn't just mean there are more minorities among us than ever before (which there are), it means that as a group of 160 individuals, we represent a wide range of ideas, identities and life experiences.

As a result, we were not immune to the divisive forces of 2016, when we all met. Our class's stark ideological divide has compromised personal relationships, and introduced troubling fault lines in our community—much like the recent phenomena observed in any other group of professionals across the country. It's the initial reason I ran for class president at the end of our first year—but that's another topic on which I could write volumes.

The point is: despite our striking differences, we universally recognize the importance of supporting our undocumented colleagues. Even the majority of our most conservative classmates attended our rally in solidarity with our DACA classmates, and feel strongly about their right to realize their passion for treating the sick of this nation. For us, this is not a matter of politics—it's a common sense step toward a healthier America.

<http://bit.ly/2xtMQUu>

Scientists produce best estimate of Earth's composition

Scientists at ANU have produced the best estimate of Earth's elemental composition which will help them understand how the Earth formed 4.6 billion years ago.

The Solar System began as a dense blob in a molecular cloud of hydrogen gas and dust that collapsed under its own gravity, forming the early Sun, Earth and other planets.

Co-researcher Associate Professor Charley Lineweaver said the Earth's chemical composition was set at that early stage of formation.

"The four most abundant elements - iron, oxygen, silicon and magnesium - make up more than 90 per cent of the Earth's mass, but working out exactly what the Earth is made of is tricky," said Dr

Lineweaver from the Research School of Earth Sciences and the Research School of Astronomy and Astrophysics at ANU.

"Seismological studies of earthquakes inform us about the Earth's core, mantle and crust, but it's hard to convert this information into an elemental composition. "Our deepest drilling has only scratched the surface down to 10 kilometres of our 6,400 kilometre radius planet. Rocks at the surface only come from as deep as the upper mantle."

The research is published in the international journal *Icarus* and is available [here](#).

Lead author ANU PhD scholar Haiyang Wang said the team made the most comprehensive estimates of the Earth's composition based on a meta-analysis of previous estimates of the mantle and core, and a new estimate of the core's mass.

"Our work focused on getting realistic uncertainties so that our reference model can be used in future comparisons of the Earth with the Sun, or with Mars or with any other body in the Solar System," said Mr Wang from the ANU Research School of Astronomy and Astrophysics.

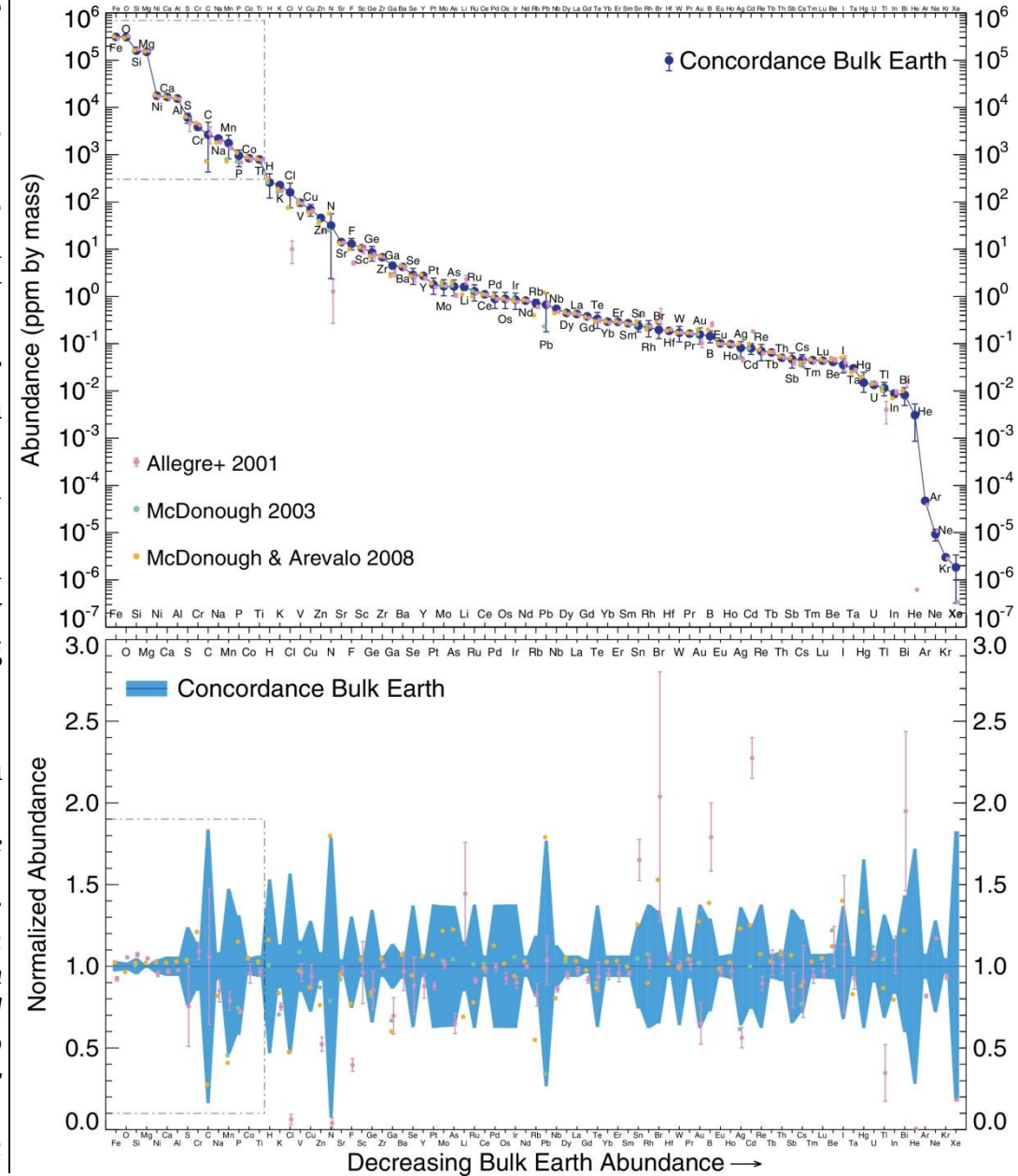
Co-researcher Professor Trevor Ireland from the ANU Research School of Earth Sciences said planetary scientists would find many uses for this new composition record. "This will have far-reaching importance, not only for planetary bodies in our Solar System but also other star systems in the universe," he said.

Haiyang Wang has received the Prime Minister's Australia Asia Award to support his PhD research at ANU.

Haiyang S. Wang et al. *The elemental abundances (with uncertainties) of the most Earth-like planet*, *Icarus* (2017). DOI: [10.1016/j.icarus.2017.08.024](https://doi.org/10.1016/j.icarus.2017.08.024)

Comparison of our concordance bulk Earth elemental abundances with the recent estimates of Allègre et al. (2001), McDonough (2003), and McDonough and Arevalo (2008). Elements for which McDonough (2003) and McDonough and Arevalo (2008) reported identical values are plotted as McDonough and Arevalo (2008) points. In the lower panel the literature values from the top panel are normalized to our concordance values. The blue band in the lower panel indicates our estimate of the uncertainties on the concordance values. 70% of the literature points fall within this band. The sum of our bulk Earth

abundances is 106 since we have rescaled the concordance PM and core abundances to 106 (see Appendix C). The literature abundances have not been rescaled to ensure their abundances sum to 106. The dashed boxes on the left in both panels contain the 15 most abundant elements and are zoomed-in on in Fig. 7.



<http://bbc.in/2xijpnJ>

'Eye worm infection could spread to UK'

An eye infection caused by a parasitic worm increasingly common in mainland Europe could spread to the UK, pet owners are being warned.

The disease, *Thelazia callipaeda* or oriental eye worm, is transmitted by a type of fruit-fly that lands on the eyes and deposits infective larvae. Cats, dogs and people catch it from the flies, which feed on eye secretions.



Fully grown worms sit on the surface of the eye and cause irritation John Graham-Brown

There have been three recent canine cases reported in the UK, but the animals had been imported from abroad. One had been brought to the UK from Romania. The other two had recently travelled to mainland Europe with their owners. All made a full recovery following drug treatment and eye washes to flush out the adult worms.

Britain has the same type of fruit-fly - *Phortica variegata* - and the concern is these could become infected and then spread the condition to people and animals in the UK, says veterinary expert John Graham-Brown, from Liverpool University, in the [BMJ publication Veterinary Record](#).

Mr Graham-Brown said there was no risk of people catching the infection directly from their pets. But dog owners should be on the lookout for signs of the infection in themselves and their pet if they had recently travelled to places where the disease was endemic.

Italy, France, Switzerland, Germany, Spain, Portugal, Bosnia-Herzegovina, Croatia, Romania, Bulgaria, Hungary, Greece, Belgium and Serbia have reported locally transmitted cases. Infected animals show a variety of symptoms, from mild conjunctivitis (pink eye) to

severe corneal ulceration, which, if untreated, can lead to blindness. Fully grown adult worms might also be visible, but the microscopic larvae cannot be seen with the naked eye.

Mr Graham-Brown said: "So far, there has been only one strain of the infection round in Europe. But it's been spreading quite rapidly recently. We are not sure why. "We do have this type of fly in the UK as well, so there is the potential for an infected dog to come back and give it to the fly here, and then it could spread." The flies tend to be found in areas of oak woodland during warmer months.

<http://bit.ly/2ymeBfw>

The wrong first step to revive athletes in cardiac arrest Clearing the airway to prevent 'tongue swallowing' can delay chest compressions and may contribute to unnecessary deaths, according to a new report in HeartRhythm

Philadelphia, PA - About three million people have viewed the YouTube video of the death of American collegiate basketball player Frank Gathers from cardiac arrest during a game in 1990. The sequence of the events clearly shows that for two entire minutes following his collapse, he received no form of cardiopulmonary resuscitation (CPR). New research presented in *HeartRhythm*, suggests that the main obstacle to an appropriate bystander response during athletes' cardiac arrest could be an apparently widespread myth: that "tongue swallowing" is a common complication of sudden loss of consciousness that must be avoided or relieved at all costs to prevent death from asphyxia.

"As of February 2017, the 'Hands-Only CPR Demo Video' by the American Heart Association (AHA) and the 'Learn Hands-Only CPR' from the American Red Cross had 337,104 and 227,032 views, respectively. These figures shrivel next to the staggering number of views of the videos showing Frank Gathers, who died of cardiac arrest while an entire jam-packed basketball stadium crowd watched in disbelief, without anybody providing any form of appropriate CPR," comments lead investigator Dana Viskin, MD, from the Tel Aviv

Sourasky Medical Center and Sackler School of Medicine, Tel Aviv University, Israel.

To determine whether inadequate responses by fellow team members may well be an unappreciated serious obstacle to successful resuscitation of athletes collapsing with cardiac arrest during competition, investigators reviewed 29 available videos from 1990-2017 of sudden circulatory arrest (SCA), or loss of consciousness. The rescue process of each collapsed player was analyzed with careful attention paid to the first action performed by the first to arrive on the scene. In videos in which the initial intervention was visible, 65% showed actions to prevent tongue swallowing, which included placing the player on his side or tilting his head sideways, and forcefully opening the athlete's mouth placing the rescuer's fingers in the victim's mouth, sometimes with a visible pull at the tongue. Only 38% show chest compressions. Further, a defibrillator was brought to the scene in only two cases, and in one of those, the first shock was not delivered until 10 minutes later. Of the players presenting with cardiac arrest, 36% survived.

According to Dr. Viskin, "The cardiac arrest events of athletes caught on video and available on the internet portray a very disturbing picture of fellow teammates responding to cardiac arrest incorrectly. Prevention and/or 'relief' of tongue swallowing' appears to take priority over chest compression in the majority of video-documented events."

This misplaced priority has also been encouraged by inaccurate reporting by various media, such as a BBC Sports internet article commending inappropriate resuscitation attempts by teammates and medical staff to prevent the athlete from swallowing his tongue as he lost consciousness.

In an accompanying editorial, Peter J. Kudenchuk, MD, of the Division of Cardiology/Arrhythmia Services, University of Washington, Seattle, WA, emphasizes that the initial moments following collapse are arguably the most critical, since all successive

emergency actions depend on SCA first being quickly recognized and properly treated. "It is during this time period when the battle for survival can all too easily be lost, particularly if SCA is mistaken for something less immediately life-threatening, therapies are misdirected, or not given at all."

In the past, the traditional approach to resuscitation relied on ABC, or Airway, Breathing and Chest compressions. Although this guideline was revised by the AHA in 2010 to CAB (Chest Compressions, Airway, Breathing), the old technique may still be holding sway. Dr. Kudenchuk notes that the newer guidelines assume that all collapses are due to SCA, and require only two questions to be answered: "Is the patient conscious?" and "Are they breathing normally?" Two "No" answers trigger immediate chest compressions. This "No, No, GO!" algorithm is proving to increase survival where it is being used.

This lag in understanding is of great concern. Dr. Viskin adds, "Since we began our research regarding this topic, at least three more cases had been added to our statistics, including a very recent one, not included in this present study, involving a soccer player in The Netherlands. It is interesting since the world seems to be moving forward in regard to technology, medical equipment, and research, but in a field with media exposure to millions of people worldwide, we seem to be over a decade behind."

While this study focuses on how tragic cardiac arrest might be when it strikes an athlete, Dr. Kudenchuk emphasizes that it also typifies the bystander inaction that occurs in hundreds of thousands of instances of others who fall victim to out-of-hospital cardiac arrest each year across the globe.

<http://bit.ly/2wEcyX8>

Did Ancient Greeks Deliberately Build Temples on Earthquake Faults?

Research suggests many Greek sacred sites throughout the Eastern Mediterranean were built on fissures created by earthquakes

By John Dyer, Seeker | September 19, 2017 07:00am ET

Archeologists and other scientists have long known that intoxicating gases emanating from water flowing from deep within the earth likely produced the visions of the oracle of Delphi, a seer who guided ancient Greeks with her prophecies from around 800 BC through the 4th century AD from her temple on Mount Parnassus. Now new research suggests many other Greek sacred sites were built on similar fissures created by earthquakes throughout the Eastern Mediterranean. "The ancient Greeks placed great value on hot springs unlocked by earthquakes," said Iain Stewart, professor of geoscience communication and director of the Sustainable Earth Institute at the University of Plymouth in Britain. "But perhaps the building of temples and cities close to these sites was more systematic than has previously been thought."

In a study published [recently in the Proceedings of the Geologists' Association](#), Stewart showed how temples and other structures at Mycenae, Ephesus, Cnidus, and Hierapolis were, like Delphi, built and rebuilt over earthquake faults.

In Cnidus, an ancient, ruined city in what is now southwestern coastal Turkey, for example, locals erected a temple in the same place — over a fault in hindsight — even after earthquakes wrecked it.

"You think, 'That's bad luck, isn't it?'" said Stewart, describing when he first reached his findings after reviewing his data. "Then it dawns on you. These people weren't stupid. There was this grand dawning that there was probably something deliberate here."

The pattern repeats in other cities, reflecting how Greeks viewed the underworld as the destination for the soul after death and a source of mystical power and knowledge. "We don't have a culture that looks down," said Stewart. "We have a culture that looks up to the stars."

Conversely, in his paper Stewart speculates that seismic activity could have cut off hot springs that had justified an oracle at Perachora Heraion, a sanctuary founded in honor of the goddess Hera in the 9th century BC near Corinth but then fell into disuse in 300 BC potentially after earthquakes.

Greeks also of course didn't live in cities with skyscrapers and millions of residents, either, he added. Earthquakes would have been viewed more as mystical occurrences and not natural disasters caused by the movement of tectonic plates. Historical and geological records show that earthquakes were frequent at the height of ancient Greek civilization, he added.

"It's hard [for people today] to separate the modern take that earthquakes are dangerous," said Stewart. "We kind of know too much. We know what they can do. In ancient times, they would have seen them very differently. In the course of 30 seconds, the ground would open up and then everything would go back to normal."

Stewart added that archeologists might also look at sacred sites in South America, the Middle East, and Asia to see whether earthquake fault lines played a role in their construction.

But Stewart admitted he wasn't an archeologist. More work needed to be done to fully explore his thesis, he said, adding that he published his paper as a "provocation" for other more qualified researchers to take up the topic. "I do recognize that when geologists lumber into archeological territory, they make mistakes," he said. "I'm quite happy with archeologists taking us to task."

<http://bit.ly/2fnqc05>

Comprehensive meta-analysis affirms cranberries' role in promoting a healthy urinary tract
New investigation advises doctors to recommend cranberry products as first line of defense against repeated urinary tract infections (UTIs)

CARVER, Mass. - A thorough review of dozens of studies led scientists to conclude that healthcare professionals should be telling their patients to have cranberry products as a first step in reducing recurrent UTIs. The comprehensive meta-analysis and assessment of human clinical trials, published in the official journal of the American Urological Association, The Journal of Urology®, assures practitioners and their

patients that cranberry products are a low cost, low risk and effective way to help prevent recurrent UTIs.¹

To answer the question, "Can Cranberries Contribute to Reduce the Incidence of Urinary Tract Infections?" a total of 28 studies showing results from nearly 5,000 patients were considered. Authors found a statistically significant risk reduction in repeat UTIs overall, but not significant for any particular subgroup. However, patients with recurrent UTIs who ingested cranberry products and had undergone gynecological surgery, experienced a significant reduction in UTIs.

"Our investigation supports that cranberry products can be a powerful tool to fight off frequent UTIs," explains lead author, Dr. Ângelo Luís. "While recommendations for dosage and duration of treatment require further study, the efficacy of the medicinal properties of cranberry products has been well-established."

The review explains that the medicinal properties of cranberries may be attributed to their unique polyphenol, proanthocyanidins - or PACs, for short. Their ability to keep infection-causing bacteria from sticking to the urinary tract walls may be the major reason for their effectiveness in limiting infection growth and recurrence.

According to the authors, scientists and practitioners continue to explore the use of alternative therapies in the prevention of common infections as part of the global movement to reduce antibiotic use and resistance. It is estimated that one third of women in the United States will get a UTI by the age of 24.²

"Findings like this," adds Dr. Luis, "give practitioners a viable, inexpensive, non-antibiotic option to help patients reduce the recurrence of an uncomfortable and potentially debilitating infection."

"As one of the oldest alternative therapies and U.S.-born berries, independent research such as this not only provides public health benefits, it revitalizes the enthusiasm for cranberry products year-round. The industry appreciates the efforts of these researchers and takes pride in the healthy attributes that cranberry products provide to consumers around the world," comments Terry Humfeld, executive

director of the non-profit research and education-focused organization, The Cranberry Institute.

This review was funded by Universidade da Beira Interior and bank Santander/Totta protocol post-doctoral research fellowship BIPD/ICI-FC-BST-UBI 2016 (ÂL).

About the Cranberry Institute

The Cranberry Institute is a not-for-profit organization founded in 1951 to further the success of cranberry growers and the industry in the Americas through health, agricultural and environmental stewardship research as well as cranberry promotion and education. The Cranberry Institute is funded voluntarily by Supporting Members that handle, process, and sell cranberries. Supporting Members are represented in national and international regulatory matters and research efforts are done on their behalf. For more information about the Cranberry Institute, along with the health benefits of cranberries and current scientific research, visit <http://www.CranberryInstitute.org>.

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¹ Luis A, Domingues F and Pereira, L. Can cranberries contribute to reduce the incidence of urinary tract infections? A systematic review with meta-analysis and trial sequential analysis of clinical trials. *J Urol* 2017; 614-21.

² Foxman B, Barlow R, D'Arcy H, Gillespie B and Sobel JD. Urinary tract infection: self-reported incidence and associated costs. *Ann Epidemiol* 2000; 10:509-515.

<http://bit.ly/2fnwU6>

Tool-wielding monkeys push local shellfish to edge of extinction

HUMANS aren't the only primate to have pushed their prey towards extinction. Monkeys have also over-exploited animals for food.

By Aylin Woodward

Long-tailed macaques forage for shellfish on islands off Thailand, then crack them open with stone tools. They target the largest rock oysters, bludgeoning them with stone hammers, and pry open the meatiest snail and crab shells with the flattened edges of their tools.



Don't eat all the shellfish Mark MacEwen / NaturePL

These macaques are one of three primates that use stone tools, alongside chimpanzees in Africa and bearded capuchins in South America. "Stone tools open up an opportunity for foods they otherwise wouldn't even be able to harvest," says Lydia Luncz at the University of Oxford.

Luncz wanted to investigate the impact of the monkeys' shellfish snacking on the prey themselves. Her team followed 18 macaques on their daily foraging routes along the shores of Koram and NomSao, two neighbouring islands off eastern Thailand, recording their tool selection and use. On Koram – the more densely populated island, home to 80 macaques compared with NomSao's nine – Luncz's group saw not only smaller oysters and snails, but also fewer of each species. Multiple prey species were less abundant on Koram than NomSao, with four times as many tropical periwinkles on NomSao as on Koram (eLife, doi.org/cc7d).

"It's been shown that systematic predation causes prey of smaller size," says Nathaniel Dominy at Dartmouth College in Hanover, New Hampshire. The oysters on Koram were about 70 per cent smaller than their counterparts on NomSao, and the periwinkles were less than half the size. A single tool-using monkey on Koram can eat over 40 shellfish a day, so Luncz's group thinks this predation pressure is driving these shellfish changes.

Luncz says the macaques might deplete the prey on the islands. Afterwards, they will stop using stone tools and even forget how.

"Tool use, a socially learned behaviour, has always been viewed as this positive thing that opens up resources," she says. "But by over-harvesting they're putting their technology knowledge at risk."

What's more, Dominy thinks the study might help us better understand modern humans' exit from Africa over 70,000 years ago. One idea is that our ancestors didn't travel overland, but instead followed the Asian coastline, relying on shellfish for food.

"Over time, we see a reduction in shell size in the archaeological record, which suggests a systematic use of shellfish," Dominy says. But nobody was sure whether size reduction was due to changing ocean conditions, or large-scale human predation. "This paper is the first to offer compelling evidence in support of the former," he says.

This article appeared in print under the headline "Tool-using monkeys suck shellfish dry"

<http://bit.ly/2hq3t9X>

Virus testing better than Pap test for cervical cancer screening

Five-yearly checks for human papilloma virus may replace the current biennial Pap smear regime for early detection of cervical cancer.

Andrew Masterson reports.

Human papilloma virus (HPV) tests are much more efficient at detecting potential cervical cancers than traditional Pap tests, according to a large pilot study published in the journal PLOS Medicine. The study, led by Karen Canfell of Cancer Council New South Wales, Australia, used almost 5000 cervical samples from women aged between 25 and 64 and subjected them to a randomised series of tests, ranging from standard Pap smears (a strategy known as cytology) to partial genotyping of the HPV virus.

The HPV tests detected significantly more precancerous anomalies than the Pap smears – with the Pap cohort returning a positive response in 2.7% of cases, compared to 3.8% in the HPV test group. The finding comes as Australia prepares to change its cervical cancer screening regime from recommending Pap smears every two years to HPV tests every five.

"This adds to existing evidence about how much more effective HPV screening is," says Canfell. "We now have a superior method for detecting high-grade cervical precancerous abnormalities – this will provide increased protection to women against developing invasive cervical cancer later in life."

The results are welcomed by Annika Antonsson of the Cancer Control Laboratory at the QIMR Berghofer Medical Research Institute in Queensland, Australia, who was not involved in the study. "They found that these pre-cancerous cell changes were identified at much higher rates in the groups that were tested for HPV compared to the groups that were tested with Pap smears, regardless of whether they were offered HPV vaccination or not," she says.

Margaret Heffernan, behavioural researcher on HPV vaccines at RMIT University, also praises the study, but calls for more research. "It is piloting the HPV test for quality assurance and safety in a sample that includes women who have received the HPV vaccine," she says. "The study found higher detection rates that confirm the viability of introducing the test as a replacement for the current Pap test."

She urged further testing, however, to confirm the efficacy of the approach across a broader range of women, especially "women from vulnerable and therefore high-risk populations".

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Complex life evolved out of the chance coupling of small molecules

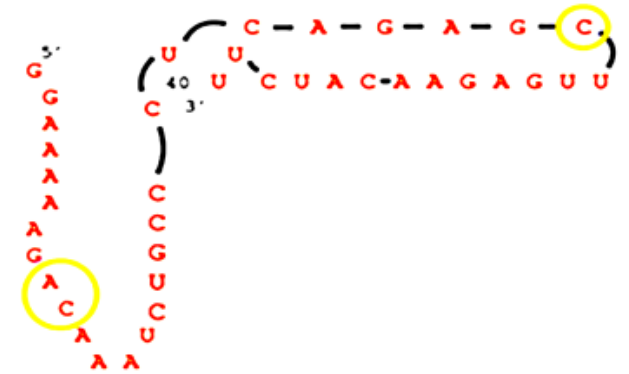
Complex life, as we know it, started completely by chance, with small strands of molecules linking up, which eventually would have given them the ability to replicate themselves.

Complex life, as we know it, started completely by chance, with small strands of molecules linking up, which eventually would have given them the ability to replicate themselves.

In this world, billions of years ago, nothing existed that we would recognise today as living. The world contained only lifeless molecules that formed spontaneously through the natural chemical and physical processes on Earth. However, the moment that small molecules connected and formed larger molecules with the ability to replicate themselves, life started to evolve.

"Life was a chance event, there is no doubt about that," says Dr Pierre Durand from the Evolution of Complexity Laboratory in the Evolutionary Studies Institute at Wits University, who led a project to find out how exactly these molecules linked up with each other. Their results are published today in the journal Royal Society Open Science, in a paper entitled "Molecular trade-offs in RNA ligases affected the modular emergence of complex ribozymes at the origin of life".

Very simple ribonucleic acid (RNA) molecules (compounds similar to Deoxyribonucleic acid (DNA)) can join other RNA molecules to themselves through a chemical reaction called ligation. The random joining together



A simple RNA molecule such as this could have been responsible for complex life. Credit: Wits University

of different pieces of RNA could give rise to a group of molecules able to produce copies of themselves and so kick start the process of life.

While the process that eventually led to the evolution of life took place over a long period of time, and involved a number of steps, Wits PhD student Nisha Dhar and Durand have uncovered how one of these crucial steps may have occurred.

They have demonstrated how small non-living molecules may have given rise to larger molecules that were capable of reproducing themselves. This path to self-replicating molecules was a key event for life to take hold.

"Something needed to happen for these small molecules to interact and form longer, more complex molecules and that happened completely by chance," says Durand.

These smaller RNA molecules possessed enzyme activity that allowed ligation, which, in turn allowed them to link up with other small molecules thereby forming larger molecules.

"The small molecules are very promiscuous and can join other pieces to themselves. What was interesting was that these smaller molecules were smaller than we had originally thought," says Durand.

The smallest molecule that exhibited self-ligation activity was a 40-nucleotide RNA. It also demonstrated the greatest functional flexibility as it was more general in the kinds of substrates it ligated to

itself although its catalytic efficiency was the lowest. "Something needed to happen for molecules to reproduce, and thereby starting life as we know it. That something turned out to be the simple ligation of a set of small molecules, billions of years ago," says Durand.

<http://bit.ly/2hquoCk>

Treating asthma or COPD with steroid inhaler raises the risk of hard-to-treat infections

People using steroid inhalers for asthma or chronic obstructive pulmonary disease more likely to suffer bacterial infections

Older people who use steroid inhalers for asthma or chronic obstructive pulmonary disease (COPD) are more likely to suffer particular bacterial infections, according to a large study published in the European Respiratory Journal ^[1].

Worldwide, asthma and COPD affect hundreds of thousands of people and they are commonly treated with steroid inhalers to reduce symptoms and improve lung function. However, the new study suggests that these inhalers also increase the risk of lung infections caused by nontuberculous mycobacteria, which are notoriously difficult to treat and resistant to a number of common antibiotics.

The research was led by Dr Sarah Brode, Assistant Professor of medicine at the University of Toronto, Canada and a staff respirologist at the University Health Network and West Park Healthcare Centre. She explained: "These infections are not particularly common but they are chronic and difficult to treat, and are associated with an increased risk of death. Treatment typically requires at least three antibiotics given for longer than a year and this can still fail to tackle the infection."

The study included 417,494 people with COPD or asthma aged 66 and older, who had all been prescribed medicine for their condition at least once. Of these patients, researchers found that 2,966 had also been diagnosed with nontuberculous mycobacteria infections and they compared this information with whether they used a steroid inhaler, the type of steroid they had used, and how much they had used it.

They found that people who were currently using steroid inhalers were around twice as likely to be diagnosed with an infection of this type, and that the longer they had been taking the steroid, the greater the risk. They also discovered that one particular type of steroid, called fluticasone, was particularly risky.

Previous research has suggested that steroid inhalers hamper the body's ability to fight infections by reducing or impairing the cells of the immune system.

Dr Brode said: "Steroid inhalers are critical treatments for managing asthma symptoms for most patients. Although they have also been shown to benefit patients with COPD, they are less important in the management of this condition, and they may only provide more benefit than harm in a sub-set of COPD patients. There is an ongoing debate on which patients with COPD should be treated with inhaled steroids.

"This research suggests that patients should discuss whether they need to use steroid inhalers with their clinicians, and whether the benefits outweigh the potential harms. If they do need to use them, they should be on the lowest effective dose.

"Clinicians should carefully consider the potential benefits and harms of steroid inhalers in patients with asthma or COPD, especially those who have already had an infection of this type in the past."

Professor Guy Brusselle, Science Council Chair of the European Respiratory Society, said: "This is a large and important observational study on the effects of steroid inhalers in older people with asthma and COPD. Although not common, infections caused by nontuberculous mycobacteria are serious and difficult to treat. We must consider the effects of steroid inhalers on the risk of these infections alongside their known benefits and side-effects. "Patients who are prescribed steroid inhalers should not stop their medicine. But, if they are concerned, they should speak to their doctor about the pros and cons of the treatment and whether it is right for them."

Dr Brode and her colleagues continue to study this group of patients and are now investigating which treatments might be most effective against nontuberculous mycobacteria infections.

<http://bit.ly/2jUDc88>

Blood Levels of Magnesium May Predict Dementia Risk

The levels of magnesium in your blood may be linked to your risk of developing dementia later in life, a new study from the Netherlands finds.

By Sara G. Miller

Compared with people in the study who had high or low levels of the mineral in their blood, those with levels in the middle range were less likely to develop dementia, according to the study, which was published online today (Sept. 20) in the journal *Neurology*.

Lead study author Dr. Brenda Kieboom, an epidemiologist at Erasmus University Medical Center in the Netherlands, said in a statement that "the results are intriguing." [6 Big Mysteries of Alzheimer's Disease]

The study did not prove that high or low levels of magnesium in the blood cause dementia, Kieboom said; rather, it showed only an association between blood magnesium levels and dementia risk. The findings "need to be confirmed with additional studies," but if they hold up, it's possible that blood tests to measure magnesium levels could one day be used to help determine who is at risk of developing dementia, Kieboom said.

Previous research suggested two possible ways that magnesium could play a role in the development of dementia, according to the study. Magnesium regulates a receptor in the brain that plays an important role in memory and learning. In addition, low levels of magnesium have been linked to inflammation, which could increase dementia risk. The study involved nearly 10,000 older adults living in the city of Rotterdam, in the Netherlands. At the beginning of the study, when the average age of the participants was 65, the people were screened for dementia, and their blood magnesium levels were measured. The participants were then followed for an average of eight years.

The researchers divided the people into five groups based on their blood levels of magnesium, and found that the people in the highest and the lowest blood magnesium groups were each about 30 percent more likely to develop dementia during the study period than those in the middle group.

Nearly all of the people in the study had magnesium levels that fell within what doctors consider a normal range, according to the study. Normal blood magnesium levels range from 0.85 millimoles per liter (mmol/L) to 1.10 mmol/L, according to the National Library of Medicine. The lowest group in the study had magnesium levels of 0.79 mmol/L or lower, and those in the highest group had levels of 0.9 mmol/L or above.

The researchers noted that the study had limitations. For example, magnesium levels were measured only once, at the beginning of the study, and could have changed over the study period. In addition, it's possible that blood magnesium levels don't fully represent the total amount of the mineral in a person's body, the researchers said.

Earlier studies have linked blood magnesium levels to other neurological conditions, including migraines and epilepsy.

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Non-avian dinosaur found to have laid blue eggs

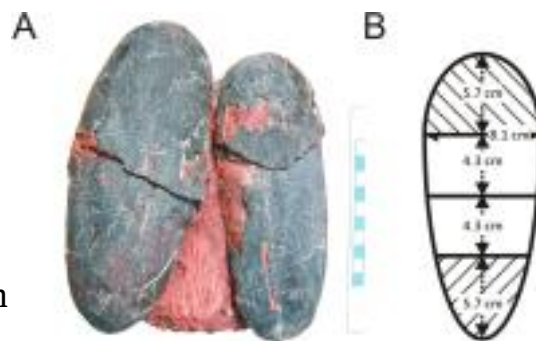
A team of researchers from Germany and the U.S. has found that a non-avian dinosaur living in what is now China laid colored eggs.

September 20, 2017 by Bob Yirka report

Phys.org - In their paper published on the peer-reviewed site PeerJ, the team describes their study of the egg fossils and what their findings suggest about the evolution of colored eggs in modern birds.

Many modern birds lay colored eggs—some are monochrome, like blue robin's eggs; others are multi-colored like those of the dove. But until now, it was believed that all dinosaur eggs were white because dinosaurs laid their eggs in protected nests. In this new effort, the researchers have found an example of a dinosaur that laid blue or green eggs.

The team reports that theirs was the first effort to seriously study color in dinosaur eggs. It came about after the team noted some Heyuannia huangi fossilized eggs that had a bluish tint—researchers had previously assumed the tint was due to mineralization, but the new team thought maybe there was more to it. Prior research had shown that Heyuannia huangi were dinosaurs with parrot-like beaks that walked on hind legs. The team used mass spectrometry and chromatographic separation to take a closer look at the eggs and detected traces of biliverdin and protoporphyrin, pigments commonly found in modern colored bird eggs. The eggs were also dated back to the Late Cretaceous period, which ran from 100 to 66 million years ago.



(A) Pair of oviraptorid Heyuannia eggs (NMNS CYN-2004-DINO-05) from the Chinese province of Jiangxi before sampling. Porosity measurements and calculations of water vapor conductance are based on these eggs. Pieces of eggshell from each of the four zones depicted in (B) were used in porosity measurements. (B) Egg model separated into four zones used for zonal porosity measurements. PeerJ (2017). DOI: 10.7717/peerj.3706

The oviraptor Heyuannia huangi were also feathered dinosaurs—many of their fossils have been found over the years, but until now, no one suspected that they laid colored eggs. The coloring, the team suggests, is a strong indication that the eggs were laid in open nests—the coloring would have served as camouflage. In modern birds, only those that lay them in open nests are colored. Their finding also shows that egg coloring began before the evolution of modern birds—it started with non-avian dinosaurs and was passed down to modern ancestors.

The researchers report that as a result of their find, they are taking a look at other fossilized dinosaur eggs to see if perhaps some of those also were colored.

Jasmina Wiemann et al. *Dinosaur origin of egg color: oviraptors laid blue-green eggs*, PeerJ (2017). DOI: 10.7717/peerj.3706

Abstract

Protoporphyrin (PP) and biliverdin (BV) give rise to the enormous diversity in avian egg coloration. Egg color serves several ecological purposes, including post-mating signaling and camouflage. Egg camouflage represents a major character of open-nesting birds which accomplish protection of their unhatched offspring against visually oriented predators by cryptic egg coloration. Cryptic coloration evolved to match the predominant shades of color found in the nesting environment. Such a selection pressure for the evolution of colored or cryptic eggs should be present in all open nesting birds and relatives. Many birds are open-nesting, but protect their eggs by continuous brooding, and thus exhibit no or minimal eggshell pigmentation. Their closest extant relatives, crocodiles, protect their eggs by burial and have unpigmented eggs. This phylogenetic pattern led to the assumption that colored eggs evolved within crown birds. The mosaic evolution of supposedly avian traits in non-avian theropod dinosaurs, however, such as the supposed evolution of partially open nesting behavior in oviraptorids, argues against this long-established theory. Using a double-checking liquid chromatography ESI-Q-TOF mass spectrometry routine, we traced the origin of colored eggs to their non-avian dinosaur ancestors by providing the first record of the avian eggshell pigments protoporphyrin and biliverdin in the eggshells of Late Cretaceous oviraptorid dinosaurs. The eggshell parataxon *Macroolithus yaotunensis* can be assigned to the oviraptor *Heyuannia huangi* based on exceptionally preserved, late developmental stage embryo remains. The analyzed eggshells are from three Late Cretaceous fluvial deposits ranging from eastern to southernmost China. Reevaluation of these taphonomic settings, and a consideration of patterns in the porosity of completely preserved eggs support an at least partially open nesting behavior for oviraptorosaurs. Such a nest arrangement corresponds with our reconstruction of blue-green eggs for oviraptors. According to the sexual signaling hypothesis, the reconstructed blue-green eggs support the origin of previously hypothesized avian paternal care in oviraptorid dinosaurs. Preserved dinosaur egg color not only pushes the current limits of the vertebrate molecular and associated soft tissue fossil record, but also provides a perspective on the potential application of this unexplored paleontological resource.

Journal reference: PeerJ

<http://bit.ly/2fJNcwD>

This Radical New Method Regenerates Failing Lungs With Blood Vessels Intact

“Replace and refresh approach” flushes sick cells from a diseased lung and reseeds the empty matrix with healthy ones

By [Shelly Fan](#) - Sep 20, 2017

Save for the occasional burning pain that accompanies a run, most people don't pay much attention to the two-leafed organ puffing away in our chests.

But lungs are feats of engineering wonder: with over 40 types of cells embedded in a delicate but supple matrix, they continuously pump oxygen into the bloodstream over an area the size of a tennis field. Their exquisite tree-like structure optimizes gas exchange efficiency; unfortunately, it also makes engineering healthy replacement lungs a near-impossible task.

Rather than building lungs from scratch, scientists take a “replace and refresh approach”: they take a diseased lung, flush out its sickly, inflamed cells and reseed the empty matrix with healthy ones.

It's an intricate procedure—nevertheless, the delicate branches of blood vessels are often completely destroyed during the process. Without blood to deliver nutrients and molecules to the newly seeded cells, the graft fails.

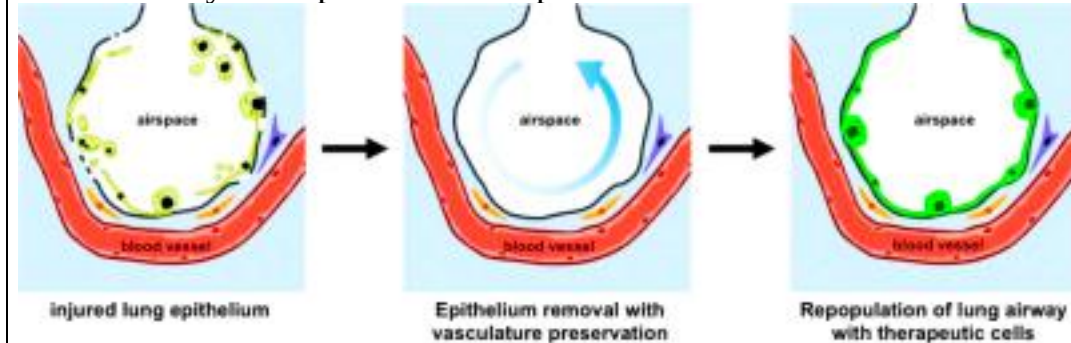
What if, thought Dr. Gordana Vunjak-Novakovic at Columbia University, rather than removing all cells from a donor lung, we gently clean out only the diseased cells in the airway without touching blood circulation?

This week, Vunjak-Novakovic's team published a “[radically new approach](#)” to bioengineering lungs: making scaffolds with blood vessels intact.

When researchers added back therapeutic human cells that line the lung's airways to a rat lung scaffold, the foreign cells—in this case, epithelium cells—homed to the correct location, attached, and thrived.

Because lung failure often stems from diseased epithelium cells, [says](#) study author Dr. N. Valerio Dorrello, this new method allows us to regenerate lungs by treating just the injured cells.

Dr. Matthew Bacchetta, who also worked on the project, sees the method as a “[transformative](#)” way to obtain lungs ready for transplant. Because lungs are notoriously bad at repairing themselves, in severe cases the only real option is a transplant.



N. Valerio Dorrello and Gordana Vunjak-Novakovic, Columbia University

It's a hard sell—only up to [20 percent](#) of patients are still alive ten years later, the procedure is expensive, and the demand for donor lungs far exceeds the supply.

These new “vascularized” lungs bring us one step closer to the penultimate goal: transplanting lungs made from a patient's own cells, seeded onto a donor scaffold from a cadaver or even primate or pig.

The patients' cells give the scaffold a complete immune makeover, lowering the risk of immune rejection—a main reason why transplants fail. “As a lung transplant surgeon, I am very excited about the great potential of our technique,” he [says](#).

First Breath

Engineering functional lungs is nothing short of a moonshot, even in the ambitious field of regenerative medicine. The lung is a real jungle: at the microscopic level, the tree-like airways contain alveoli, tiny bubble-like structures where the lungs exchange gas with our blood. Both arteries and veins enwrap the alveoli like two sets of mesh pockets.

At least a half dozen cellular denizens work in tandem to keep the alveoli spheres inflated, to guard the organ against infections, and to enforce the structure of its many branches. This three-dimensional complexity is why we ruled out the possibility of growing lungs from scratch, [explains](#) Dr. Laura Niklason, a biomedical engineer at Yale University who was not involved in the new study.

Back in 2010, Niklason had a [brilliant idea](#): rather than relying on synthetic templates that mimic the organ's intricate structure—a “very tall order,” she [says](#)—scientists could use nature's own template, the lung's matrix, as a jumping off point. Niklason's approach is similar to stripping down a house to its bare bones—weight-bearing beams, struts and bolts—and reworking the rest to its new owner's tastes.

As a proof-of-concept, Niklason's team used a detergent that washed away the cells and blood vessels from a rat lung. They then soaked the lung matrix scaffold inside a “bioreactor” that mimics the conditions of a growing fetus. When the team reseeded the scaffold with a cocktail of cells, the lung regrew its blood vessels, alveoli and tiny airways with the right types of cells—[all within four days](#).

In the ultimate test of functionality, Niklason's team transplanted the regrown lungs back into living rats. A few seconds later, the lung inflated, turning bright red as it took in oxygen and blood supply.

It's just an initial step, the team [wrote](#) at the time. The lungs only survived up to two hours in the donor's body, and subsequent analysis revealed bleeding and blood clots within the airway and regrown capillaries. One potential reason is this: the blood vessels may not have formed proper junctions with the alveoli. While still allowing gas exchange, this eventually causes blood leaks into the lungs.

Breath of Fresh Air

If newly-grown blood vessels form malfunctioned junctions, why not preserve the originals instead?

That's exactly what Vunjak-Novakovic's team tackled in the new [study](#) published in Science Advances. Adapting Niklason's technique, the team inserted a tube into the airway of a newly harvested rat lung

and pumped through a gentle detergent that only removed the lung's epithelial cells—the inner lining. Blood vessels, in contrast, were washed with an electrolyte solution similar to Gatorade.

With this small change, we removed over 70 percent of epithelial cells—which are often the root of lung diseases—but maintained the vasculature, the authors [say](#).

Like cartographers mapping a new land, the team next probed the integrity of the vessels. Injecting tiny beads that glow under UV light into the lung's main artery, they watched as the beads flooded the twisting capillaries, glowing bright within the larger vessels.

In contrast, there were no obvious signs of glowing beads within the airway or alveoli, suggesting that the blood vessels were intact—no leakage!

With scaffold in hand, the team next marinated the structure with human lung epithelium cells. As a bonus, they also used lung cells derived from induced pluripotent stem cells (iPSCs). iPSCs are made from a patient's own cells—often skin cells—and can be coaxed to become nearly any other cell type with the right cocktail of signals.

Because iPSCs retain the person's immune profile, scaffolds seeded with these cells have a much lower chance of being rejected.

Within a mere 24 hours, the team detected signs of the newly seeded cells within the lung scaffolds. Under the microscope, the newcomers attached to the right spot, stabilized and begun rapidly dividing to repopulate the missing cells. The lung grafts also had a boost in breathing power—they could expand more fully—gaining back roughly 50 percent of what was lost during the detergent treatment.

A Breath Away?

The study stops short at the final test: transplanting the engineered lung back into a recipient. As with older generation scaffolds, the newly minted lungs could also develop deadly blood clots or bleeding once reintroduced into a living, breathing animal.

What's more, the team only used a mild detergent in their preparation to preserve the lung's integrity. The result was a partial cleanout with

some of the rats' own epithelial cells still intact. These injured stragglers may provide important information to the new, healthy cells, so this could be an unexpected bonus, the authors [explain](#). Whether they are friend or foe will have to be tested in a future study.

The technology needs a lot more work before it could be used in humans, but Vunjak-Novakovic and colleagues are already excited about potential new treatment options. This study provides proof-of-concept evidence that our approach works, the authors [write](#). We show, for the first time, that it's possible to wash out diseased lung epithelial cells without touching blood vessels.

What really gets the team excited is this: although freshly harvested rat lungs were used in this study, in theory the method could be used without removing the lung.

This is "[transformative](#):" patients with injured lung epithelial cells could be irrigated with the detergent to remove the sickly cells. Doctors can then harvest their skin cells and transform them into healthy lung cells to reseed the lung.

"Every day, I see children in intensive care with severe lung disease who depend on mechanical ventilation support," [says](#) Dorrello. We may be on our way to an entirely new treatment solution for these patients and regenerate their broken lungs, he [says](#).

<http://bit.ly/2ylztmv>

Work on China's mission to Mars 'well underway'

China's programme to launch a mission to Mars in 2020 is "well underway", its top planner said Wednesday as the country moves forward with its ambitious space programme.

The probe will carry 13 types of payload including six rovers, the official Xinhua news agency said. "The Mars exploration programme is well underway," it cited the mission's chief architect Zhang Rongqiao as saying. "The payloads will be used to collect data on the environment, morphology, surface structure and atmosphere of Mars." Zhang was speaking at the Beijing International Forum on Lunar and Deep-space Exploration, which began Wednesday.

The Long March-5 carrier rocket will blast off from the Wenchang Space Launch Centre in the tropical island province of Hainan, Xinhua said. Once the probe is in orbit around Mars after a seven-month journey, a lander will separate from it and touch down in the red planet's northern hemisphere. The lander will then deploy rovers to explore the surface.

Beijing sees its multi-billion-dollar space programme as a symbol of its rise and of the Communist Party's success in turning around the fortunes of the once poverty-stricken nation. In July it successfully launched the Long March-4B, its first X-ray space telescope to study black holes, pulsars and gamma-ray bursts.

And in April the country's first cargo spacecraft completed its docking with an orbiting space lab—a key development in China's goal of having its own crewed space station by 2022.

<http://bit.ly/2wKwJ5x>

Mysterious flesh-eating bacteria is raging in Australia ***Cases doubled, severity is increasing, but authorities don't even know how it spreads.***

[Beth Mole](#) - 9/21/2017, 2:05 AM

In the last year, cases of a ghastly but mysterious [flesh-eating bacterial infection](#) have more than doubled in Victoria, Australia, raising alarm among health experts.

There were 239 cases of the flesh-eating infections in the past 12 months, according to [figures](#) (PDF) released this week by health authorities. In 2016, there were only 102 reported cases, while 2015 and 2014 tallied just 58 and 47. And the rate of new infections is currently skyrocketing: in the past few months, case counts hit nine per week, according to [Australia's Nine News](#). The number of severe cases has also doubled.

While the rises alone are enough to worry health experts, the fact that virtually nothing is known about the cause of the infection has some dismayed. "I'm at the forefront as a clinician trying to treat patients and getting more and more overwhelmed but also distressed at the fact

that we are doing nothing to try and prevent people getting it in the first place,” Dr. Daniel O’Brien, of Royal Melbourne and Geelong hospitals, told Nine News.

Baffling bacteria

The infections are caused by *Mycobacterium ulcerans*, a slow-growing bacterium that causes gaping, palm-sized ulcers. Sometimes called [Buruli ulcers](#), the lesions seem to dissolve skin and gnaw away at tissue. The bacteria are known to lurk around Victoria, but experts don’t know where it lives or how it spreads.

“There are theories about transmission via mosquitos, theories about it being in the soil and getting through wounds, theories about whether some animals are involved, in that we know that some possums can be affected by it,” O’Brien said. “But we don’t actually know where it lives, why it’s there, and how it gets spread to humans. How can we possibility halt an epidemic when we don’t have that basic information?”

The bacterium was first identified in Australia in 1948, but the ulcers get their name from Buruli county (now called Nakasongola) in Uganda, where researchers reported large numbers of ulcers in the 1960s. It’s now known to skulk in at least 33 countries, and the World Health Organization considers it “largely a problem of the [poor in remote rural areas](#) (PDF).” It causes a few thousand cases worldwide each year, most of which are in children under the age of 15.

Antibiotics are usually effective at treating the lesions. But, early treatment is critical to reducing skin loss and tissue damage. Victims often need surgery to clean out dead flesh and repair wounds.

Zombie limbs

There are currently no prevention strategies or vaccines. Despite decades of knowing about the bacteria, researchers are still stumped by them. A study published just this April added weight to the idea that the bacteria are somehow linked to [disturbed water sources](#), such as those with flooding or deforestation.

A [study out last month](#) looked at where the ulcers show up on people. The study of 579 patients found that they tend to cluster on arms and legs. The researchers, co-led by O’Brien, concluded: “We propose that targeting behavior by biting insects rather than direct contact with a contaminated environment best explains the lesion distribution we observed.”

One likely source of confusion for researchers is the slow-growing nature of *M. ulcerans*. It’s unclear how quickly symptoms show up after a person is infected, but authorities estimate that it’s somewhere in the range of four weeks to nine months. Such a large window can make it difficult to pinpoint when or where an exposure occurred, let alone how. “For me this is an urgent health problem that our community needs addressed,” O’Brien said. “I think our government should be putting significant amounts of money into trying to stop it and that means research.”

This week, a 13-year-old girl from Tyabb, Victoria, started [an online petition](#) to urge Health Minister

Greg Hunt to provide more research funding for the disease.

The girl was stricken with a Buruli ulcer on her knee in April and is still recovering. She needed three surgeries to clear out rotting flesh.



([Here's a picture of her ulcer](#), but warning, it's gruesome.)

“It’s still not looking that great,” she said. “We call it the zombie leg.”

<http://bit.ly/2xrnmlVL>

Extinct Big-Mouthed Frogs May Have Dined on Dinos Giant, armored amphibian may have preyed on juvenile dinosaurs

By Mindy Weisberger, Senior Writer

A group of modern frogs whose comically rotund bodies and giant mouths earned them the nickname "Pac-Man" frogs is attracting attention - not for the size of their maws, but for the power of their bite. And their extinct relative, known as a "devil frog," may have

packed even more of a mouthy wallop, researchers reported in a new study.

Recently, researchers conducted the first measurements of bite strength in frogs. Initially, the scientists calculated the bite force in small "Pac-Man" frogs, also known as South American horned frogs. Then, the researchers scaled up their findings to determine bite force in an extinct relative, a giant, armored amphibian known as *Beelzebufo ampinga*, or "devil frog," that lived about 65 million to 70 million years ago.



Open wide! South American horned frogs have big mouths that deliver surprisingly powerful bites. Kristopher Lappin

The scientists' findings showed that the extinct devil frog would have had a vise-like mouth grip even more powerful than that found in living horned frogs. Combined with its enormous size, *Beelzebufo*'s bite strength could have allowed the animal to prey even on juvenile dinosaurs, the scientists reported in a new study.

Most frogs have relatively weak jaws and feed on small prey that they subdue primarily with their sticky tongues, the study authors wrote. But roly-poly South American horned frogs in the genus *Ceratophrys* have an exceptionally powerful bite that allows them to snatch and hold prey almost as big as the frogs themselves. The researchers questioned how strong that bite would have been in similarly big-headed frogs that lived millions of years ago.

The scientists began by looking at Cranwell's horned frogs, testing the bite force of eight frogs measuring between 1.6 and 3.8 inches (4.0 and 9.6 centimeters) in length, with heads that were 0.6 to 1.3 inches (1.5 to 3.2 cm) long and 0.9 to 1.8 inches (2.2 to 4.6 cm) wide. Researchers had the frogs clamp their mouths on a force transducer — a device for measuring bite force — made of two metal plates padded with leather strips to protect the frogs' jaws.

Once the researchers knew the frogs' bite force, they could scale that measurement up by adjusting parameters such as the frog's head and body size and estimating the accompanying changes in muscle size, the study said. Next to the small "Pac-Man" frogs, the extinct devil frog was gargantuan, with a body measuring about 16 inches (41 cm) long and a head reaching about 6 inches (15 cm) in width.

The study's calculations predicted that at that size, the devil frog's bite would have been as powerful as that of a wolf or of an adult female lion or tiger. That certainly would make *Beelzebufo* capable of taking down small crocodiles or dinosaurs that shared its habitat — especially if its hunting habits were similar to the aggressive and tenacious chomp of the "Pac-Man" frogs, the researchers explained.

"Horned frogs have quite an impressive bite, and they tend not to let go," the study's lead author, A. Kristopher Lappin, a professor of biological sciences at California State Polytechnic University in Pomona, said in a statement.

Lappin noted that he spoke "from experience," though he did not provide details as to what exactly that experience was.

By comparison, the bite of the much larger — and possibly dinosaur-consuming — devil frog would have been "remarkable," Lappin said in the statement. "Definitely not something I would want to experience firsthand."

The findings were published online yesterday (Sept. 20) in the [journal Nature: Scientific Reports](#).

<http://bit.ly/2xxvFRe>

Study links brain inflammation to suicidal thinking in depression

Patients with major depressive disorder have increased brain levels of a marker of microglial activation, a sign of inflammation

Philadelphia, PA - Patients with major depressive disorder (MDD) have increased brain levels of a marker of microglial activation, a sign of inflammation, according to a new study in *Biological Psychiatry* by researchers at the University of Manchester, United Kingdom. In the

study, Dr. Peter Talbot and colleagues found that the increase in the inflammatory marker was present specifically in patients with MDD who were experiencing suicidal thoughts, pinning the role of inflammation to suicidality rather than a diagnosis of MDD itself.

"Our findings are the first results in living depressed patients to suggest that this microglial activation is most prominent in those with suicidal thinking," said Dr. Talbot. Previous studies suggesting this link have relied on brain tissue collected from patients after death.

"This paper is an important addition to the view that inflammation is a feature of the neurobiology of a subgroup of depressed patients, in this case the group with suicidal ideation," said Dr. John Krystal, Editor of Biological Psychiatry. "This observation is particularly important in light of recent evidence supporting a personalized medicine approach to depression, i.e., that anti-inflammatory drugs may have antidepressant effects that are limited to patients with demonstrable inflammation."

In the study, first author Dr. Sophie Holmes and colleagues assessed inflammation in 14 patients with moderate-to-severe depression who were not currently taking any antidepressant medications. Immune cells called microglia activate as part of the body's inflammatory response, so the researchers used a brain imaging technique to measure a substance that increases in activated microglia.

The evidence for immune activation was most prominent in the anterior cingulate cortex, a brain region involved in mood regulation and implicated in the biological origin of depression, confirming the results of a previous study that first identified altered microglial activation in medication-free MDD patients. Smaller increases were also found in the insula and prefrontal cortex.

"The field now has two independent reports -- our study and a 2015 report by Setiawan and colleagues in Toronto -- showing essentially the same thing: that there is evidence for inflammation, more specifically microglial activation, in the brains of living patients during a major depressive episode," said Dr. Talbot.

This link suggests that among depressed patients, neuroinflammation may be a factor contributing to the risk for suicidal thoughts or behavior. According to Dr. Talbot, the findings "emphasise the importance of further research into the question of whether novel treatments that reduce microglial activation may be effective in major depression and suicidality."

The article is "[Elevated translocator protein in anterior cingulate in major depression and a role for inflammation in suicidal thinking: a PET study](#)," by Sophie E. Holmes, Rainer Hinze, Silke Conen, Catherine J. Gregory, Julian C. Matthews, Jose M. Anton-Rodriguez, Alexander Gerhard, and Peter S. Talbot. It appears in Biological Psychiatry, published by Elsevier.

The authors' affiliations and disclosures of financial and conflicts of interests are available in the article.

John H. Krystal, M.D., is Chairman of the Department of Psychiatry at the Yale University School of Medicine, Chief of Psychiatry at Yale-New Haven Hospital, and a research psychiatrist at the VA Connecticut Healthcare System. His disclosures of financial and conflicts of interests are available [here](#).

<http://bit.ly/2yLJQqa>

The surprising, ancient behavior of jellyfish

Jellyfish and humans may seem wildly different, but both still need to sleep

At first glance, humans seem to have very little in common with Cassiopea, a primitive jellyfish. Cassiopea is brainless, spineless, and spends essentially its entire life sitting upside down on the ocean floor, pulsating every few seconds.

However, Caltech scientists have now discovered that, as different as our daily schedules may seem, humans and jellyfish actually start and end their days with the same behavior: sleep. This finding that jellyfish sleep implies that sleep is an ancient behavior, largely untouched by millennia of evolution.

The work was a collaboration between three Caltech laboratories led by: Paul Sternberg, Thomas Hunt Morgan Professor of Biology and Howard Hughes Medical Institute Investigator; Viviana Gradinaru (BS '05), assistant professor of biology and biological engineering, Heritage Medical Research Institute Investigator, and Director of the Center for Molecular and Cellular Neuroscience of the Tianqiao and

Chrissy Chen Institute for Neuroscience at Caltech; and Lea Goentoro, assistant professor of biology.

The work appears online in the September 21 issue of *Current Biology*. "It may not seem surprising that jellyfish sleep--after all, mammals sleep, and other invertebrates such as worms and fruit flies sleep," says Ravi Nath, the paper's co-first author and a graduate student in the Sternberg laboratory.

"But jellyfish are the most evolutionarily ancient animals known to sleep. This finding opens up many more questions: Is sleep the property of neurons? And perhaps a more far-fetched question: Do plants sleep?"

In order to be considered "sleeping," an organism must meet three critical criteria. First, it must demonstrate a period of reduced activity, or quiescence. Second, the organism must exhibit a decreased response to otherwise-arousing stimuli while in the quiescent state. Finally, the organism must show an increased sleep drive when it is deprived of sleep.

"When humans sleep, we are inactive, we often can sleep through noises or other disturbances which we might otherwise react to if we were awake, and we're likely to fall asleep during the day if we don't get enough sleep," says Claire Bedbrook, co-first author and a graduate student in the Gradinaru laboratory. "We might seem extremely different from jellyfish, but we both exhibit a similar sleep state."

So, how do you prove that a jellyfish is asleep?

First, to demonstrate quiescence, the team set up a system of cameras to monitor the jellyfish around the clock. They discovered that the jellyfish go through periods of inactivity at night, only pulsing about 39 times per minute, compared to about 58 times per minute during the day.

Next, the team set out to prove that the animals had an increased arousal threshold during this period of decreased activity. The team set a jellyfish on a platform higher up in the tank and pulled the

platform out from underneath the animal once the jellyfish showed signs of quiescence.

Normally, an alert jellyfish would immediately swim to the bottom of the tank. But the jellyfish in a sleep state floated in the water for up to five seconds before "waking up" and reorienting itself.

Finally, the researchers needed to show that, when deprived of sleep, the jellies would exhibit an increased sleep drive--just as humans do after a sleepless night.

To do this, the researchers pulsed water at the animals every 10 seconds for 20 minutes, effectively "poking" them to keep them awake. They then observed that the jellyfish were more likely to fall into the quiescent state during the day, when they would normally be active.

Though this work demonstrates that jellyfish exhibit sleep behavior, the genetic mechanisms that underlie sleep remain unknown.

"Many animals have the same genes that govern sleep," says Michael Abrams, co-first author and a graduate student in the Goentoro laboratory.

"Though it was beyond the scope of our project to measure gene expression in jellyfish, we tested the effects of compounds that in other animals are known to promote sleep, such as melatonin. We found that these compounds did affect jellyfish sleep in the predicted ways, suggesting that their underlying sleep mechanism is similar to those of other organisms -- including humans."

The project was a collaborative effort across several laboratories with different areas of expertise. In addition to the three first authors and their advisers, the team had help from Ty Basinger, a lab technician in the Goentoro lab, in working with the jellyfish; from Justin Bois, lecturer in biology and biological engineering, with computational analysis; and from Professor of Biology David Prober, an expert on how genes and neurons regulate sleep.

*The paper is titled "The Jellyfish *Cassiopea* Exhibits a Sleep-like State." Funding was provided by the National Institutes of Health, the James S. McDonnell Foundation for Complex Systems Science, the National Institute of Mental Health, the National Institute of Neurological Disorders and Stroke, the National Science Foundation, the Heritage Medical Research Institute, and the Howard Hughes Medical Institute.*

<http://bit.ly/2fIoz3c>

Personality changes don't precede clinical onset of Alzheimer's, FSU study shows

No evidence to support the idea that personality changes begin before the clinical onset of mild cognitive impairment or dementia

TALLAHASSEE, Fla. -- For years, scientists and physicians have been debating whether personality and behavior changes might appear prior to the onset of Alzheimer's disease and related dementias.

Now, the findings of a new and comprehensive study from FSU College of Medicine Associate Professor Antonio Terracciano and colleagues, published today in the journal JAMA Psychiatry, has found no evidence to support the idea that personality changes begin before the clinical onset of mild cognitive impairment (MCI) or dementia. "We further found that personality remained stable even within the last few years before the onset of mild cognitive impairment," Terracciano said.

Terracciano, College of Medicine Associate Professor Angelina Sutin and co-authors from the National Institute on Aging examined data from the Baltimore Longitudinal Study of Aging. The study looked at personality and clinical assessments obtained between 1980 and July 2016 from more than 2,000 individuals who initially showed no cognitive impairment. About 18 percent of study participants later developed MCI or dementia.

"We compared whether personality change in people who later developed dementia differed from those who remained cognitively normal," Terracciano said. "Unlike previous research, this study examined multiple waves of self-rated personality data collected up to 36 years before participants developed any sign of dementia."

What the researchers found is that the trajectory of personality traits did not differ between those who would later develop dementia and those who did not.

While personality change was not an early sign of dementia, Terracciano's study provides further support that personality traits

(including high levels of neuroticism and low levels of conscientiousness) are risk factors for dementia.

For physicians and loved ones, personality changes remain an important consideration in the care of those who have already experienced the clinical onset of MCI or dementia. Increasing apathy, irritability, mood changes and other behavioral symptoms impact quality of life for both patients and their caregivers.

<http://bit.ly/2fpADJV>

BU: Resurgence of whooping cough may owe to vaccine's inability to prevent infections

Global resurgence of pertussis, or whooping cough, in recent years can largely be attributed to the immunological failures of acellular vaccines

The startling global resurgence of pertussis, or whooping cough, in recent years can largely be attributed to the immunological failures of acellular vaccines, Boston University School of Public Health (BUSPH) researchers argue in a new journal article.

The article, published in F1000 Research, points to the differences in mucosal immunity between whole-cell pertussis (wP) vaccines and the newer acellular pertussis (aP) vaccines, first introduced in the 1990s, as playing a pivotal role in the resurgence of the disease.

"This disease is back because we didn't really understand how our immune defenses against whooping cough worked, and did not understand how the vaccines needed to work to prevent it," said Christopher J. Gill, associate professor of global health at BUSPH and lead author of the article. "Instead we layered assumptions upon assumptions, and are now find ourselves in the uncomfortable position of admitting that we may have made some crucial errors. This is definitely not where we thought we'd be in 2017."

Up until the 1950s, there were millions of cases of whooping cough around the globe each year, with numerous fatal cases in infants. The introduction of whole-cell pertussis (wP) vaccines led to a 99 percent reduction in cases. Later, as wP vaccines raised concerns of possible

rare neurologic adverse events, aP vaccines were licensed and used in a number of countries starting in the early 1990s. Since then, cases of whooping cough have risen sharply. In 2014, there were more than 32,000 cases reported in the US. "The resurgence of pertussis in the US to its highest levels since the 1940s emphasizes the need for answers to these questions," the authors wrote.

The researchers examined mathematical models of pertussis transmission, data derived from the aP and wP vaccines responses in animals, and recent insight into the immunology of pertussis and pertussis vaccines. They found that, contrary to existing assumptions, although both vaccines blocked symptomatic disease, wP vaccines blocked also infections in animals while aP vaccines did not. Other differences included wP vaccines' ability to induce a stronger herd immunity and robust TH17 responses, which confer mucosal immunity, while aP vaccines only induced TH2 responses.

Experimental and immunologic data has shown that aP vaccines do not provide herd immunity, while mathematical models imply otherwise. The researchers proposed a hypothesis to reconcile the contradictory findings: Herd effects from aP vaccines may be the result of modifications in disease presentation that lead to reduced possibility of transmission rather than induced resistance to infection.

The researchers also considered the role of several known factors in the rise of whooping cough cases, including detection bias, waning of immunity, and evolutionary shifts in the bacteria's genome. They found that, while contributing to the increase in incidence, these factors alone do not fully explain existing epidemiologic data.

Citing the urgency of the growing health crisis, the authors emphasized the need to go beyond the limitations of animal models and provide human data to further examine the arguments put forth in their article. "The resurgence of pertussis in the aP vaccine era is evolving into a slow-moving global public health crisis," the researchers wrote. "But, with the public's trust in vaccines waning, this has also become a public relations crisis."

Don Thea, professor of global health at BUSPH, was a co-author on the article.

<http://bit.ly/2hrZVnq>

Excess dietary manganese promotes staph heart infection ***Too much dietary manganese -- an essential trace mineral found in leafy green vegetables, fruits and nuts -- promotes infection of the heart by the bacterium Staphylococcus aureus ("staph").***

The findings, reported this week in the journal Cell Host & Microbe, add to the evidence that diet modifies risk for infection. The discovery also suggests that people who have excess levels of tissue manganese, including those who consume dietary supplements with high concentrations of the metal, may be at increased risk for staph infection of the heart.

"The human body does a wonderful job of regulating nutrient levels, and a traditional Western diet has plenty of minerals in it. The idea of super-dosing nutrients needs to be given careful consideration," said Eric Skaar, Ph.D., MPH, Ernest W. Goodpasture Professor of Pathology and senior author of the current study.

Skaar and his colleagues studied the impact of dietary manganese on staph infection in a mouse model. Most of the mice that consumed a high manganese diet -- about three times more manganese than normal -- died after infection with staph. The investigators discovered that the animals on the high manganese diet were particularly susceptible to staph infection of the heart, which was a surprise, said Skaar, who is also professor of Pathology, Microbiology and Immunology and director of the Vanderbilt Institute for Infection, Immunology and Inflammation.

"We know very little about how manganese is moved around and regulated. It's a mystery why high manganese affects staph infection of a single organ," he said.

The researchers found that excess manganese inactivates a key line of defense against pathogens: the innate immune system's reactive oxygen burst. Normally, in response to staph, "neutrophils pour into

the site of infection and blast the bacteria with reactive oxygen species," Skaar explained. The excess manganese counters this blast.

"It's striking that a single dietary change can inactivate one of the most powerful branches of innate immune defense and lead to fatal infection," Skaar said.

The protein calprotectin -- another line of defense -- usually acts as a "sponge" to mop up manganese and other metals. Keeping nutrients away from pathogens is known as "nutritional immunity." For reasons that are not clear, however, calprotectin is completely ineffective in the high manganese hearts, Skaar said.

Staph is the leading cause of bacterial endocarditis (infection of the inner lining of the heart chamber and heart valves) and the second most frequent cause of bloodstream infections.

Interestingly, some populations of people have both increased risk for staph infections, particularly endocarditis, and higher than normal levels of tissue manganese, Skaar noted. These populations include intravenous drug users, patients with chronic liver disease and patients on long-term intravenous diets.

In ongoing studies, Skaar and his colleagues are working to understand how manganese is transported and regulated in vertebrates and why the heart is particularly susceptible to fatal staph infections when manganese levels are high. They are also exploring the impact of other nutrient minerals and vitamins on infection.

Lillian Juttukonda, an M.D./Ph.D. student, is the first author of the Cell Host & Microbe paper. The research was supported by grants from the National Institutes of Health (AI101171, AI107233, AI069233, AI073843), the Department of Veterans Affairs, the Vanderbilt Digestive Disease Research Center and the Defense Advanced Research Projects Agency.

<http://bit.ly/2xsbHG1>

Higher manganese levels in children correlate with lower IQ scores, UC study finds

Study finds children with higher levels of Manganese had lower IQ scores

CINCINNATI--A study led by environmental health researchers at the University of Cincinnati (UC) College of Medicine finds that children in East Liverpool, Ohio with higher levels of Manganese (Mn) had lower IQ scores. The research appears online in the journal *NeuroToxicology*, available in advance of publication.

The study analyzed blood and hair samples of 106 children 7 to 9 years of age from East Liverpool and surrounding communities, who enrolled in the study from March 2013 to June 2014. Working with a trained registered nurse from East Liverpool, participants and their caregivers were also given cognitive assessments and questionnaires at the time the samples were taken. The study found that increased Mn in hair samples was significantly associated with declines in full-scale IQ, processing speed and working memory.

Manganese is an element generally found in combination with iron and many minerals. It plays a vital role in brain growth and development, but excessive exposure can result in neurotoxicity. Manganese is used widely in the production of steel, alloys, batteries and fertilizers and is added to unleaded gasoline.

Erin Haynes, DrPH, associate professor in the Department of Environmental Health and lead author of the study, was approached by East Liverpool school district officials in 2013, prompted by concerns of students' academic performance, paired with the knowledge that Mn concentrations in the area have exceeded U.S. Environmental Protection Agency (EPA) reference levels for more than a decade.

"There are socioeconomic issues at play, however, they are also compounded by potentially significant environmental exposures," says Haynes, who collaborated with the Kent State East Liverpool Campus and the community group Save our County Inc., formed in 1982 by East Liverpool residents in response to the proposed construction of a hazardous waste incinerator in their community. "Children may be particularly susceptible to the neurotoxic effects of ambient Mn

exposure, as their brains are undergoing a dynamic process of growth and development."

After concerns of elevated airborne levels of Mn, the school district superintendent in East Liverpool requested testing students for manganese along with neuropsychological tests. A pilot study overseen by Haynes found levels of Mn at double the level in children from the other CARES study cohort, and further investigation was pursued to examine the association between Mn exposure and child cognition.

Located in northeast Ohio along the Ohio River, East Liverpool has a demonstrated history of environmental exposures, with EPA records showing elevated levels of manganese concentrations since 2000. In 2005, East Liverpool was deemed by the EPA to be a potential environmental justice area, afflicted with major environmental exposures, and a 2010 EPA report noted manganese concentrations detected by all monitors in East Liverpool had "consistently exceeded" health-based guidelines set by the agency.

With a declining population of just 11,000, just 7.3 percent of East Liverpool residents have a college degree. The East Liverpool school district reports a higher than average percentage of students in special education (19 percent) versus the Ohio state average of 13 percent.

The school board learned of Haynes' research studying manganese in Marietta, Ohio. Marietta was the original location of the Communities Actively Researching Exposure Study (CARES) which was initiated in 2008 based on community concern about exposure to manganese from a nearby metallurgical manufacturing company. Cambridge, Ohio serves as the comparison community, and the CARES research has since expanded into East Liverpool. In previous studies, the CARES scientific team has found that both too low and too high levels of manganese can be associated with lower neurodevelopment.

Marietta and East Liverpool have some of the highest levels of ambient manganese in the country, Haynes says, and notes that their studies continue in these areas and include neuroimaging, "as we

continue to advance our understanding of the impact of manganese on neurodevelopment, and help to define the lines between essential benefit and toxicological harm."

The study also included researchers from Cincinnati Children's Hospital Medical Center, Icahn School of Medicine at Mount Sinai, University of Albany, New York State Department of Health and the late Roxanne Burns, PhD, chair of the biology department at Kent State University East Liverpool Campus. "Dr. Roxanne Burns was a strong advocate for the study and for the community, and we are deeply indebted to her contributions," says Haynes.

This research was supported by the National Institute of Environmental Health Sciences (R01ES016531, R21ES021106, and P30-ES06096) and NIH/NCRR8UL1TR000077. The researchers cite no conflicts of interest.

<http://bit.ly/2weFoZF>

700-year-old saint myth has been proven (almost) true Scientists confirm that the age and content of an old sack is in accordance with a medieval myth about Saint Francis of Assisi.

For more than 700 years the Friary of Folloni near Montella in Italy has protected and guarded some small fragments of textile.

According to the legend the textile fragments originate from a sack that appeared on the doorstep of the friary in the winter of 1224 containing bread sent from Saint Francis of Assisi, who at that time was in France. The bread was allegedly brought to the friary by an angel. Ever since that cold winter's night the sack has been guarded by the friary, and today the last few remaining fragments are kept as a relic in a well protected shrine.

In line with the legend

A Danish/Italian/Dutch team of researchers led by Associate Professor Kaare Lund Rasmussen from University of Southern Denmark has had the opportunity to conduct scientific studies of the alleged bread sack fragments. Their study is published in the journal *Radiocarbon*.

C-14 analysis revealed that the textile can be dated to 1220-1295.

The age is in line with the legend, says Kaare Lund Rasmussen, a chemist, and specialized in archaeo-chemical analyses.

There was probably bread in the sack

The researchers also looked for traces of bread in the textile. They did this by looking for ergosterol, a sterol for the fungal kingdom and encountered in several types of mould. Ergosterol can be a potential biomarker for brewing, baking or agriculture.

Our studies show that there was probably bread in the sack. We don't know when, but it seems unlikely that it was after 1732, where the sack fragments were inmured in order to protect them. It is more likely that bread was in contact with the textile in the 300 years before 1732; a period, where the textile was used as altar cloth -- or maybe it was indeed on the cold winter's night in 1224 -- it is possible, says Rasmussen.

Scientific measurements cannot prove a legend or belief. What they can do, is either to de-authenticate the object or show accordance between the physical/chemical evidence and the legend, say the researchers in their paper, published in the journal Radiocarbon.

Belief versus science

The researchers have not addressed the issue of how the bread sack ended up on the doorstep of the friary.

This is maybe more a question of belief than science, says Rasmussen. The bread sack: According to legend the bread sack miraculously appeared on the doorstep of the friary in 1224. For 300 years it was used as an altar cloth. During this time pieces were cut off and given to other religious institutions in Italy. After an earthquake in 1732 a new friary was built and the remaining sack fragments were inmured. I 1807 the fragments were moved to the main church, Santa Maria del piano. In 1817 half of the textile was returned to the friary. In 1999 the remaining half returned. Today the fragments of the textile are kept in a reliquary.

Kaare Lund Rasmussen is a chemist. He often uses his expertise for solving archaeological mysteries. He has been involved in investigating the dead sea scrolls, the death of Renaissance astronomer and alchemist Tycho Brahe and skeletons from cemeteries in Denmark, Germany and Italy.

<http://bbc.in/2fK12yV>

New antibody attacks 99% of HIV strains

Scientists have engineered an antibody that attacks 99% of HIV strains and can prevent infection in primates.

By James Gallagher Health and science reporter, BBC News website

It is built to attack three critical parts of the virus - making it harder for HIV to resist its effects. The work is a collaboration between the US National Institutes of Health and the pharmaceutical company Sanofi. The International Aids Society said it was an "exciting breakthrough". Human trials will start in 2018 to see if it can prevent or treat infection.

Our bodies struggle to fight HIV because of the virus' incredible ability to mutate and change its appearance. These varieties of HIV - or strains - in a single patient are comparable to those of influenza [during a worldwide flu season](#). So the immune system finds itself in a fight against an insurmountable number of strains of HIV.

Super-antibodies

But after years of infection, a small number of patients develop powerful weapons called "broadly neutralising antibodies" that attack something fundamental to HIV and can kill large swathes of HIV strains. Researchers have been trying to use broadly neutralising antibodies as a way to treat HIV, or prevent infection in the first place. The study, [published in the journal Science](#), combines three such antibodies into an even more powerful "tri-specific antibody".

Dr Gary Nabel, the chief scientific officer at Sanofi and one of the report authors, told the BBC News website: "They are more potent and have greater breadth than any single naturally occurring antibody that's been discovered." The best naturally occurring antibodies will target 90% of HIV strains. "We're getting 99% coverage, and getting coverage at very low concentrations of the antibody," said Dr Nabel.

Experiments on 24 monkeys showed none of those given the tri-specific antibody developed an infection when they were later injected with the virus.

Dr Nabel said: "It was quite an impressive degree of protection." The work included scientists at Harvard Medical School, The Scripps Research Institute, and the Massachusetts Institute of Technology.

'Exciting'

Clinical trials to test the antibody in people will start next year.

Prof Linda-Gail Bekker, the president of the International Aids Society, told the BBC: "This paper reports an exciting breakthrough.

"These super-engineered antibodies seem to go beyond the natural and could have more applications than we have imagined to date. "It's early days yet, and as a scientist I look forward to seeing the first trials get off the ground in 2018. "As a doctor in Africa, I feel the urgency to confirm these findings in humans as soon as possible."

Dr Anthony Fauci, the director of the US National Institute of Allergy and Infectious Diseases, said it was an intriguing approach. He added: "Combinations of antibodies that each bind to a distinct site on HIV may best overcome the defences of the virus in the effort to achieve effective antibody-based treatment and prevention."

<http://bit.ly/2wPf9bT>

Multi-gene test predicts Alzheimer's better than APOE E4 alone

Test acts as a genetic 'risk factor' to help identify preclinical Alzheimer's dementia

A new test that combines the effects of more than two dozen genetic variants, most associated by themselves with only a small risk of Alzheimer's disease, does a better job of predicting which cognitively normal older adults will go on to develop Alzheimer's dementia than testing only for the well-known genetic variant APOE E4, a scientific team led by researchers at UC San Francisco and UC San Diego has found.

APOE E4 has long been considered the strongest genetic predictor of whether someone is likely to develop Alzheimer's, although it is only carried by 10 to 15 percent of the population and recent research suggests its effects have been overstated. The polygenic hazard score

(PHS), a test developed by the research team that carried out the new study, provides risk estimates for the remaining 85 to 90 percent of people who do not carry at least one copy of APOE E4 but still have some combination of other genetic variants that put them at risk of Alzheimer's.

"Beyond APOE E4 by itself, our polygenic hazard score can identify cognitively normal and mildly impaired older folks who are at greatest risk for developing Alzheimer's-associated clinical decline over time," said Chin Hong Tan, PhD, a postdoctoral scholar at UCSF and first author of the paper, published Sept. 22, 2017, in *Annals of Neurology*. The researchers looked at five years of data on 1,081 subjects from the National Alzheimer's Coordinating Center (NACC) who did not have dementia, and found the PHS test could predict how long it would take for them to progress to Alzheimer's dementia, as well as how steep their cognitive decline would be, even after taking into account whether they were carriers of APOE E4.

Autopsies of those who did develop Alzheimer's showed that, even among those who did not carry a copy of the APOE E4 variant, a higher PHS was associated with a higher level of amyloid plaque - a protein aggregate that is a hallmark of Alzheimer's -- in the brain. These patients also showed steeper declines on cognitive tests during their lifetimes. Older individuals in the highest PHS percentiles also showed the highest incidence of Alzheimer's, which is diagnosed with cognitive tests and brain pathology, regardless of their APOE E4 status.

Many scientists now believe that rather than being a disease of aging, Alzheimer's may be the result of a disease process that begins years, perhaps decades, before symptoms of dementia appear. This is thought by many to be one reason why so many Alzheimer's drugs tested on older people with dementia have failed in clinical trials. The new PHS could help in the search for ways to identify those at risk of Alzheimer's long before they show symptoms of dementia, so they can

be treated before the disease begins ravaging their brains, the researchers said.

"Our findings have strong implications for disease stratification and secondary prevention trials in Alzheimer's, as well as direct-to-consumer genetic tests, some of which have recently received FDA clearance," said Anders Dale, PhD, Professor of Neurosciences and Radiology at UC San Diego and co-author of the new study.

The PHS test enables researchers to calculate an age-specific risk of developing Alzheimer's, based upon each person's share of 31 genetic variants, plus APOE E4. The test makes its predictions by using genetic data from more than 70,000 people in the NACC database, the International Genomics of Alzheimer's Disease Project and the Alzheimer's Disease Genetics Consortium.

"Unlike other polygenic risk scores, the continuous PHS measure is based on a survival framework and incorporates US-based Alzheimer's incidence rates," said Rahul Desikan, MD, PhD, an assistant professor in the Department of Radiology and Biomedical Imaging at UCSF, and co-senior author of the paper. "Rather than a diagnostic test, PHS may serve as a genetic 'risk factor' for preclinical Alzheimer's disease."

Other authors include Jacinth Tan, PhD, Christopher Hess, MD, PhD, and William Dillon, MD, of UCSF; Bradley Hyman, MD, PhD, of Massachusetts General Hospital; Gerard Schellenberg, PhD, of the University of Pennsylvania Perelman School of Medicine; Lilah Besser, PhD, and Walter Kukull, PhD, of the University of Washington; Karolina Kauppi, PhD, and Linda McEvoy, PhD, of UC San Diego; Ole Andreassen, MD, PhD, of the University of Oslo; and Chun Fan, MD, PhD, of UC San Diego.

<http://bbc.in/2xxRIYo>

Alarm as 'super malaria' spreads in South East Asia **The rapid spread of "super malaria" in South East Asia is an alarming global threat, scientists are warning.**

By James Gallagher Health and science reporter, BBC News website

This dangerous form of the malaria parasite cannot be killed with the main anti-malaria drugs. It emerged in Cambodia but has since spread through parts of Thailand, Laos and has arrived in southern Vietnam.

The team at the Oxford Tropical Medicine Research Unit in Bangkok said there was a real danger of malaria becoming untreatable.

Prof Arjen Dondorp, the head of the unit, told the BBC News website: "We think it is a serious threat. "It is alarming that this strain is spreading so quickly through the whole region and we fear it can spread further [and eventually] jump to Africa."

Failing treatments

In a letter, [published in The Lancet Infectious Diseases](#), the researchers detail the "recent sinister development" that has seen resistance to the drug artemisinin emerge. About 212 million people are infected with malaria each year. It is caused by a parasite that is spread by blood-sucking mosquitoes and is a major killer of children. The first choice treatment for malaria is artemisinin in combination with piperazine. But as artemisinin has become less effective, the parasite has now evolved to resist piperazine too.

There have now been "alarming rates of failure", the letter says.

Prof Dondorp said the treatment was failing around a third of the time in Vietnam while in some regions of Cambodia the failure rate was closer to 60%. Resistance to the drugs would be catastrophic in Africa, where 92% of all malaria cases happen.

'Against the clock'

There is a push to eliminate malaria in the Greater Mekong sub-region before it is too late. Prof Dondorp added: "It's a race against the clock - we have to eliminate it before malaria becomes untreatable again and we see a lot of deaths. "If I'm honest, I'm quite worried."

Michael Chew, from the Wellcome Trust medical research charity, said: "The spread of this malaria 'superbug' strain, resistant to the most effective drug we have, is alarming and has major implications for public health globally. "Around 700,000 people a year die from drug-resistant infections, including malaria.

"If nothing is done, this could increase to millions of people every year by 2050."

<http://bit.ly/2yB66h0>

New Zealand's iconic kiwi birds may be losing their sight *Not all birds need to see. Blind but perfectly healthy kiwis have been found living in New Zealand.*

By **Andy Coghlan**

The flightless nocturnal birds may be evolving to lose their eyesight altogether, suggest the researchers. The blind kiwis seem able to survive just as well using other senses such as touch, smell and hearing, so maintaining good eyesight might be a waste of energy.



Flightless... and sometimes sightless Joel Sartore, National Geographic Photo Ark

The blind birds were discovered during a study of 160 [Okarito brown kiwis](#) (*Apteryx rowi*) found in the Okarito forest on New Zealand's South Island. "We found a very high prevalence of birds with eye lesions," says [Alan Tennyson](#) at the [Museum of New Zealand Te Papa Tongarewa](#) in Wellington. "A third of them had eye problems."

But the biggest surprise was chancing upon three sightless birds. "The finding of completely blind birds in good physical condition was absolutely stunning," says team member [Christopher Murphy](#) at the University of California, Davis.

"No other birds are known to have a free-living population of blind [individuals]," says Tennyson. But plenty of other animals, such as [moles](#) and cave-dwelling fish, have evolved blindness. "Vision is not essential for survival in all animals." The discovery could help explain how species lose their sense of sight, a process called regressive evolution.

Genes for blindness

The most likely explanation is that kiwis do not need vision because of where and how they live: they are active at night, and their habitat offers plenty of food and no predators, apart from introduced animals

such as stoats. "Kiwis are flightless and generally nocturnally active, and have very good senses of smell, hearing and touch, so it seems that vision is not essential for their survival, at least for some individuals," says Tennyson.

Other researchers speculate that a gene called *Sonic hedgehog* might be responsible for the dumping of vision. This gene is important in development and has been implicated in other animals losing their sight, such as the [Mexican blind cavefish](#).

Sonic hedgehog could potentially enhance the function of touch and smell sensors in the kiwis' long beaks, at the expense of visual function. "Eye degeneration can be seen as 'collateral damage'," as the birds adapt to their "nocturnal, lightless niche in which normal, functioning eyes are not necessary", says [Stanley Sessions](#) at Hartwick College in Oneonta, New York. "That's our best guess as to what's going on in these birds."

The blind Okarito brown kiwis are a great opportunity to study how visual systems evolve and change, says Tennyson. But only if they survive: they are [endangered](#), with about 400 left in the wild.

Journal reference: BMC Biology, DOI: [10.1186/s12915-017-0424-0](https://doi.org/10.1186/s12915-017-0424-0)

<http://bit.ly/2weowT4>

Moldy Rock Pulled from 2,500 Feet Underground *Swedes searching for nuclear waste storage site stumble on a pocket of fungi that may reveal a huge hidden reservoir of life*

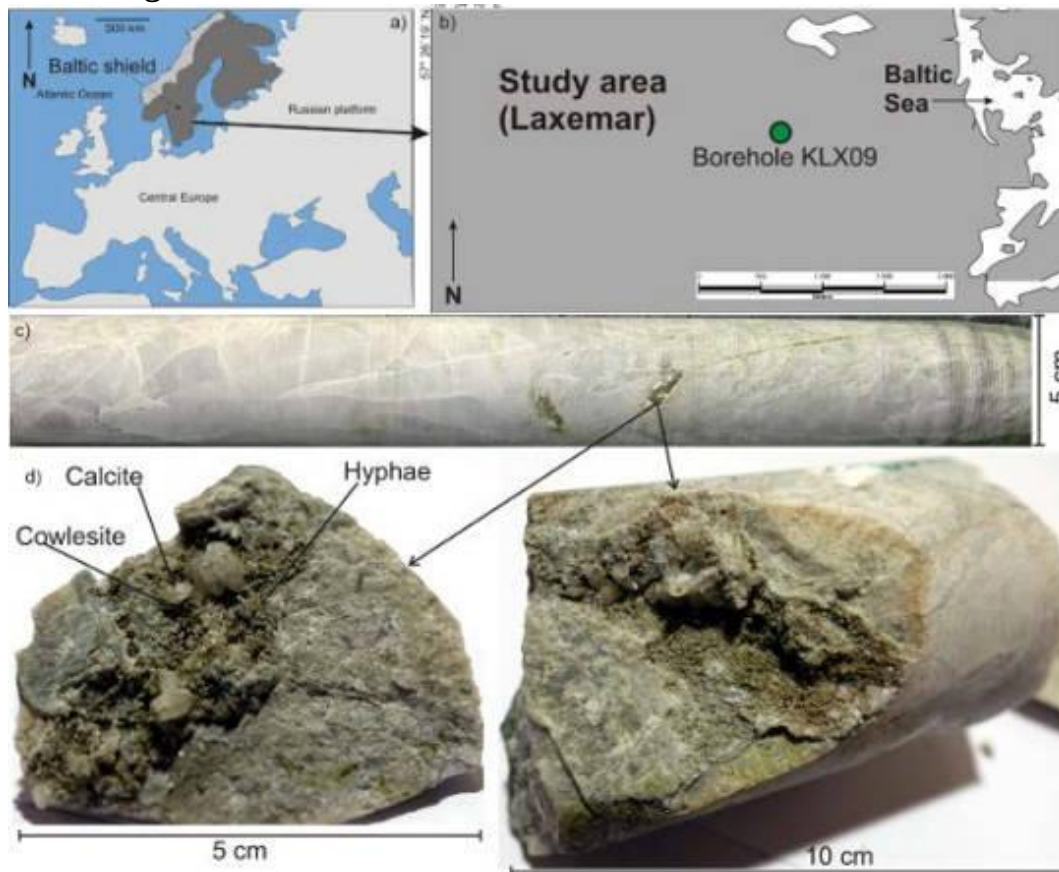
By [Jennifer Frazer](#) on September 22, 2017

Nuclear waste repositories are not known for curb appeal. Yet they are unpleasant necessities for enlightened nations seeking to stow the waste of one of the only relatively carbon-neutral fuels on Earth.

As a result, engineers have often sought to bury the stuff. In the United States, some of our radioactive waste has found its (hopefully) eternal resting place [in a salt bed over 2,000 feet deep near Carlsbad, New Mexico](#), where it will eventually be swallowed by flowing salt.

Sweden has also begun exploring the geological solution to nuclear pollution by probing its own bedrock. At nearly 2,500 feet below

ground at Test Borehole KLX09, about a mile from the Baltic Sea in southeastern Sweden, engineers and geologists recently encountered something that must have shocked them.



Credit: [Draket et al. 2017](#).

In a core sample – a slender cylinder of stone extracted by drill – they found a small cavity inside a vein of quartz. Inside was a brownish powder mingled with crystals of [calcite](#) and cowlesite. When they examined the powder under the microscope, they came to a startling conclusion: it was the half-fossilized remains of fungi. They had found a moldy pocket of rock. Generally speaking, rock isn't supposed to mold ([lichen](#) fiestas notwithstanding).

When I was in college, my microbiology professors were just beginning to report that there might be life in the deep crust. Scattered

reports from gold mines were hinting that bacteria might not just survive but thrive in hot, wet fissures deep underground.

Now we know that bacteria permeate cracks in Earth's crust, feeding off hydrocarbons and other foods trapped within. Even relatively large animals (for the subsurface) [called nematodes have been discovered in mines at depths of over two miles](#).

That got microbiologists thinking: if bacteria are abundant down there, why not fungi? Fungi have been found growing pretty much everywhere we have looked. [They have also shown themselves to be capable miners](#), tunneling through rock by secreting acid from their growing tips.

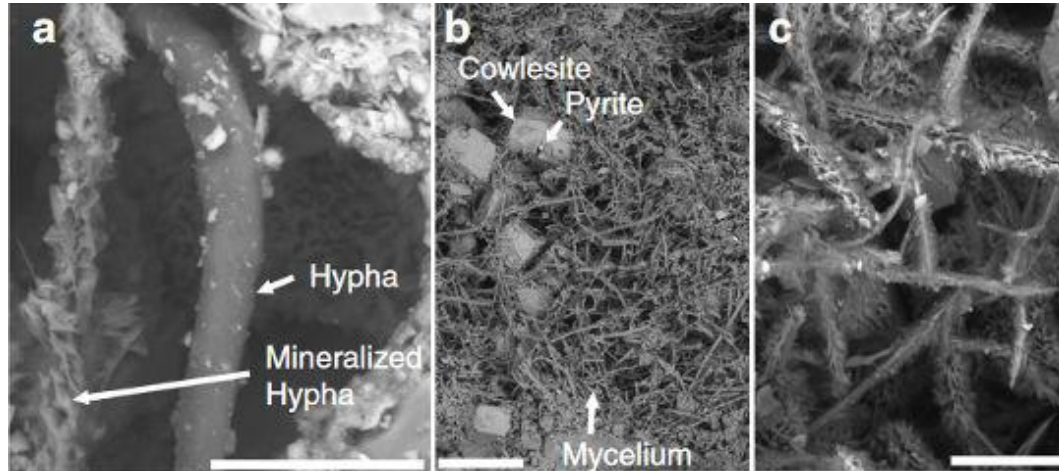
Searching for truly subterranean fungi (and not just drifters from above) is difficult and expensive, though, generally involving engineers with drill rigs rather than a eager biologists with a net or box. As a result, the world that remains unseen and unsampled is profound: scientists estimate that up to 19% of Earth's biomass is contained in the deep continental subsurface. That's a fifth of all life on Earth!

A handful of studies have turned up underground fungi or suggested they live there indirectly, but such studies are "surprisingly rare", write the Swedish and German authors of the paper describing the fungal pocket in [Nature Communications](#) in July.

The fungi hypothesized to live in deep fractures are presumed to survive without oxygen (there being no ready source nearby), but we know little about anaerobic fungi as they are scarce in habitats above ground. There is *one* place that we have found and studied anaerobic fungi in detail, however: the [rumen](#) of cattle. Inside this hot, dark digestive bag where [cud](#) is produced, fungi generate hydrogen, a fuel that tiny microbes living there can use to make organic chemicals that benefit the entire community.

The pocket from the Swedish borehole is, according to the authors, the first discovery of fungi grown in place in deep bedrock. They were partly fossilized and partly organically preserved, itself a rare finding.

Under the microscope, the scientists could see a network of entangled branching and fusing filaments, a pattern characteristic of fungi. Fungus-like microbes called [actinobacteria](#) never fuse, and fungal mimics called [oomycetes](#) (the most famous of which caused the Irish potato famine) fuse only when reproducing.



a) Organically preserved fungal filament 10 micrometers in diameter next to a mineralized hypha of the same diameter. b) Mycelium-like structures partly overgrowing cowlesite. c) Branching and anastomosing mineralized hyphae.

Credit: [Drake et al. 2017](#).

The filaments in the pocket also contained a mineralized central strand -- a common feature of fossilized fungi -- grew in a mat resembling a biofilm, and appear to have carved channels into rock which they contacted [just as fungi near the surface sometimes do](#). Yet fungi, unlike bacteria and plants, cannot make their own food. They must feed on the waste products or remains of other organisms. So what were they eating in this tiny crack a half a mile from the surface?

The scientists could not see any direct evidence of bacteria or other life in the fissure. However, pyrite crystals found among the fungal filaments were enriched in a form of sulfur preferred by microbes called sulfate-reducing bacteria -- one of the very same groups that grow with anaerobic fungi inside cattle.

If this was indeed the case, both organisms would have benefited each other: bacteria could have fed on the hydrogen and organic waste

products made by the anaerobic fungi, while the fungi fed on sugars and other organic chemicals produced by the bacteria.

Together, the combination of fossilized filaments and isotope-skewed pyrite suggests, they claim, that the deep biosphere in continental rocks may be “a neglected vast fungal habitat.”

If true, this surfeit of subsurface fungal life could pose a long-term threat to our plans for deep disposal of toxic and radioactive waste, the very thing the drillers were pursuing when they encountered the moldy rock. Both anaerobic fungi and their bacterial cohorts could dissolve or corrode barriers between waste and rock if it served their purposes. That includes leading contenders like [zeolite](#) barriers for high-level radioactive waste and copper canisters for spent nuclear fuel. We might want to take that into consideration in future designs.

It is melancholy for me, however, to think of life under these conditions, radioactive waste or no. These fungi will never know the kiss of rain, the sparkle of sunshine, or the freshness of a gentle breeze. All these things would assuredly kill them. They life they know – the only life they will ever know – might as well be on another planet, eternally dark, eternally hot, and eternally lonely, trapped under pressure in a tiny fissure half a mile from Earth’s surface.

They, on the other hand, are probably as happy as pigs in a waller.

Henrik Drake, Magnus Ivarsson, Stefan Bengtson, Christine Heim, Sandra Siljeström, Martin J. Whitehouse, Curt Broman, Veneta Belivanova, Mats E. Åström. *Anaerobic consortia of fungi and sulfate reducing bacteria in deep granite fractures*. *Nature Communications*, 2017; 8 (1) DOI: [10.1038/s41467-017-00094-6](https://doi.org/10.1038/s41467-017-00094-6)