http://bit.ly/2muxuGd

Never before seen images of early stage Alzheimer's disease

Unique images appear to contradict a previously unchallenged consensus

Researchers at Lund University in Sweden have used the MAX IV synchrotron in Lund - the strongest of its kind in the world - to produce images that predate the formation of toxic clumps of beta-amyloid, the protein believed to be at the root of Alzheimer's disease. The unique images appear to contradict a previously unchallenged consensus. Instead of attempting to eliminate beta-amyloid, or so-called plaques, the researchers now suggest stabilizing the protein.

It is a long-held belief in the scientific community that the beta-amyloid plaques appear almost instantaneously. Hence the term "popcorn plaques". The infrared spectroscopy images, however, revealed something entirely different.

The researchers could now see structural, molecular changes in the brain.

"No one has used this method to look at Alzheimer's development before. The images tell us that the progression is slower than we thought and that there are steps in the development of Alzheimer's disease that we know little about. This, of course, sparked our curiosity," says Gunnar Gouras, professor in experimental neurology at Lund University and senior author of the study.

What was happening at this previously unknown phase? Through biochemical identification the first author of the study, Oxana Klementieva, was able to look closer at these early brain changes.

The results revealed another discovery. Namely, that the beta-amyloid did not appear as a single peptide, a widely held belief in the field, but as a unit of four peptides sticking together, a tetramer.

This breakthrough offers a new hypothesis to the cause of the disease. The abnormal separation of these four peptides could be the start of the beta-amyloid aggregation that later turns into plaques.

"This is very, very exciting. In another amyloid disease, transthyretin amyloidosis, the breaking up of the tetramer has been identified as key in disease development. For this disease, there is already a drug in the clinic that stabilizes the tetramers, consequently slowing down disease progression. We hope that stabilizing beta-amyloid in a similar fashion may be the way forward in developing future therapies" says Gunnar Gouras.

The discovery could therefore alter the direction of therapy development for the disease. The aim of most clinical trials today is to eliminate plaques.

Researchers at Lund University will now try to understand the interaction patterns of beta-amyloid preceding the aggregation process. Finding the antidote to whatever breaks the beta-amyloid protein apart could open doors towards a major shift in¬ the development of therapies for Alzheimer's disease.

http://bit.ly/2npnSkN

People see black men as larger, more threatening than same-sized white men

Findings could help explain why black men more likely to be shot by police, study says

WASHINGTON -- People have a tendency to perceive black men as larger and more threatening than similarly sized white men, according to research published by the American Psychological Association.

"Unarmed black men are disproportionately more likely to be shot and killed by police, and often these killings are accompanied by explanations that cite the physical size of the person shot," said lead author John Paul Wilson, PhD, of Montclair State University. "Our research suggests that these descriptions may reflect stereotypes of black males that do not seem to comport with reality."

Wilson and his colleagues conducted a series of experiments involving more than 950 online participants (all from the United States) in which people were shown a series of color photographs of white and black male faces of individuals who were all of equal height

and weight. The participants were then asked to estimate the height, weight, strength and overall muscularity of the men pictured.

"We found that these estimates were consistently biased. Participants judged the black men to be larger, stronger and more muscular than the white men, even though they were actually the same size," said Wilson. "Participants also believed that the black men were more capable of causing harm in a hypothetical altercation and, troublingly, that police would be more justified in using force to subdue them, even if the men were unarmed."

Even black participants displayed this bias, according to Wilson, but while they judged young black men to be more muscular than the young white men, they did not judge them to be more harmful or deserving of force.

In one experiment, where participants were shown identically sized bodies labeled either black or white, they were more likely to describe the black bodies as taller and heavier. In another, the size bias was most pronounced for the men whose facial features looked the most stereotypically black.

"We found that men with darker skin and more stereotypically black facial features tended to be most likely to elicit biased size perceptions, even though they were actually no larger than men with lighter skin and less stereotypical facial features," said Wilson. "Thus, the size bias doesn't rely just on a white versus black group boundary. It also varies within black men according to their facial features."

Black men are disproportionately more likely to be killed in interactions with police, even when unarmed, according to Wilson, and this research suggests that misperceptions of black men's size might be one contributor to police decisions to shoot. But, he cautioned, the studies do not simulate real-world threat scenarios like those facing actual police officers. More research should be conducted on whether and how this bias operates in potentially lethal situations and other real-world police interactions, Wilson said.

The research was published in the Journal of Personality and Social Psychology. Previous research, also published in this journal, suggested that people view black boys as older and less innocent than similarly aged white boys, and that training and experience can help police overcome racial bias in shoot-don't shoot scenarios.

Article: "Racial Bias in Judgments of Physical Size and Formidability: From Size to Threat," by John Wilson, PhD, Montclair University; Kurt Hugenberg, PhD, Miami University; and Nicholas Rule, PhD, University of Toronto; Journal of Personality and Social Psychology, published online Mar. 13, 2017.

Full text of the article is available from the APA Public Affairs Office and at http://www.apa.org/pubs/journals/releases/psp-pspi0000092.pdf

http://bit.ly/2n1HquI

The science of liquorice: whether you love the dark root – or hate it

Liquorice has been used medicinally for over 4,000 years

Simon Cotton Senior Lecturer in Chemistry, University of Birmingham

There are foods that can split families, or even just couples. Love it or hate it foods. Marmite (and the Antipodean alternative, Vegemite) is one. Then there's Brussels sprouts, blue cheese, chilli peppers, coriander (cilantro), tomatoes (especially the cooked variety) … and liquorice.

***Liquorice root.* Shutterstock**

Personally, I've always liked liquorice, but there are others who feel very differently about it. There are known to be genetic reasons behind a dislike of some foods such as Brussels sprouts or coriander but no one has established this for liquorice.

Liquorice has a long history; the root of the plant *Glycyrrhiza glabral* has been used medicinally for over 4,000 years.

Liquorice has anti-inflammatory, antibacterial and antiviral properties and has been used, notably in traditional Chinese medicine, for the treatment of gastric ulcers and liver disorders, such as hepatitis B.

Among its less beneficial effects on the body is raised blood pressure, caused by the glycyrrhizic acid it contains, if you eat too much. Through its interaction with the hormone aldosterone, it causes reabsorption of sodium and excretion of potassium, resulting in an increase in sodium levels and a decrease in potassium levels – one symptom of which is muscular weakness. And pregnant women have been advised to avoid it because it pushes up levels of cortisol, the stress hormone. A study just published upon which this advice is based, drew on several hundred children born in Finland in 1998. They found that mothers who ate more liquorice (salmiak, liquorice with added ammonium chloride) gave birth to children who were more likely to have lower IQs and to suffer from ADHD.

Sweet smell of success

We more usually think of liquorice in confectionery, where an extract of raw liquorice is heated up with other ingredients such as flour, treacle, flavourings and colourings to produce a sweet, thickened product. In Britain, it is closely associated with the Yorkshire town of Pontefract, where it was first grown nearly 1,000 years ago after it was brought to Britain from the Middle East. In 1760, an apothecary named Charles Dunhill first produced "Pontefract cakes", flat, circular sweets. Dark salty liquorice is hugely popular in northern European countries such as Holland and Sweden.

Dark and salty: yum or yuck? Shutterstock

Liquorice is used as a flavouring in substances as diverse as tea and tobacco, and in drinks like the Egyptian erk sous and the French pastis. The sweetness of liquorice is principally due to the glycyrrhizin (or glycyrrhizic acid), which is around 40 times sweeter than sucrose (table sugar).

The aromas of raw liquorice and heat-processed liquorice are due to a blend of chemicals, recently identified by a research group in Munich, led by Peter Schieberle, a distinguished flavour scientist.

The team achieved this by first separating the complex mixtures of chemicals present in the liquorice, and then identifying each one by spectroscopic techniques. Aroma experts then examined each compound to find which of the molecules present actually contributed towards it (around 50). Finally, they reconstituted a mixture of these molecules, each present at its "natural" concentration, to see if the mixture had the characteristic liquorice smell (it did).

Star anise has the molecule anethole in common with liquorice, as do aniseed and fennel. **Shutterstock**

So there is no single molecule which has a liquorice smell by itself; what we smell is a blend of odours from all these molecules, which our brain "integrates". The molecules anethole (also found in aniseed, fennel and star anise) and estragole (also found in tarragon) supply an "aniseed" note, but there are many other important compounds that contribute, including 1,8-cineole eucalyptus, 2-acetyl-1-pyrroline, responsible for the "popcorn" note of many cereal products like bread and rice, and a number of aldehydes such as (E,Z)-2,6-nonadienal (found in cucumbers).

The heat-processed liquorice extract has many molecules in common with raw liquorice, but some are unique to it like maltol, which contributes a caramel note, and 3-hydroxy-4,5-dimethylfuran-2(5H)-one (sotolone), also with a caramel note at low concentrations and which is responsible for the flavour of raw cane sugar.

Many of the extra molecules are generated in the heating process from reactions, such as those between sugars and amino acids present in the raw liquorice root.

So enjoy your liquorice – if you do. Just don't overdo it.

Disclosure statement *Simon Cotton does not work for, consult, own shares in or receive funding from any company or organisation that would benefit from this article, and has disclosed no relevant affiliations beyond the academic appointment above.*

Partners ***University of Birmingham*** *provides funding as a founding partner of The Conversation UK.*

http://bit.ly/2n1M9w6

Snow will melt more slowly in a warmer world – here's why

As global temperatures rise, snow will melt more slowly. Yes, you read that right – more slowly.

By Brian Owens

Warmer global temperatures will lead to less snow in many mountainous areas, says Keith Musselman, a hydrologist at the National Center for Atmospheric Research in Boulder, Colorado.

That thinner layer of snow will be less likely to last into the late spring and early summer, when melting rates are highest. Instead, it will melt slowly throughout the winter and early spring, when night-time temperatures are lower and there is less direct sunlight, releasing just a trickle of water instead of a sudden gush. In short, a warming planet will cause the snow to melt sooner but more slowly.

"The more you think about it, it becomes one of those 'aha!' stories," says Musselman, who used historical snowpack measurements and computer simulations to predict how the melting rate will change by the end of the century.

Disrupting the cycle

A slower melting rate will have serious consequences for the water cycle in areas that rely heavily on the snowpack as a water source, such as the mountains of the western US. A gentler trickle is more prone to evaporate or be sucked up by plants, making it less likely to make it into streams and groundwater reservoirs.

The slower, earlier melt could also mean fewer large spring floods – which may reassure people living along mountain rivers, but is bad news for those river systems overall. "It will have negative impacts on those ecosystems that rely on a big flush of water," says Musselman, such as many mountain river systems, and on people who live downstream.

Philip Marsh, a snow hydrologist at Wilfrid Laurier University in Ontario, Canada, says these kinds of surprising and counter-intuitive results are becoming more common as researchers learn more about the complexities of interactions between the climate and snow and refine their models to take them into account. "We've been looking at these relationships too simplistically so far," he says. "We frequently stop and say 'oh, that's more complicated than we thought'."

Journal reference: Nature Climate Change, DOI: 10.1038/NCLIMATE3225

http://bit.ly/2ncfu7W

Parenthood linked to longer life

Difference persists into old age, and not influenced by sex of child

Parenthood is associated with a longer life than childlessness, particularly in older age, when health and capacity may start to decline, finds research published online in the Journal of Epidemiology & Community Health.

By the age of 60, the difference in life expectancy, which does not seem to be influenced by the sex of the child(ren), may be as much as two years, the findings suggest.

Previous research suggests that being a parent may add years to a person's life, but it's not clear when this apparent advantage may be conferred or whether it could be influenced by the sex of the child(ren).

To find out if parenthood might help stave off death in older age, the researchers tracked the lifespan from the age of 60 onwards of all men (704,481) and women (725,290) with a birth date between 1911 and 1925 and living in Sweden, using national registry data.

The study, which ran until the end of 2014, also gathered registry data on marital status and the number and sex of any children they had.

Age specific risks of death were calculated and compared for each calendar year for people who had had at least one child and for those who were childless.

Not unexpectedly, the risk of death rose with increasing age, irrespective of whether the individuals were parents or not.

But after taking account of influential factors, such as educational attainment, the risks of death were lower among those who had had at

least one child than they were among those who were childless--and more so among men than among women.

The one year risk of death for an 80 year old man with a child was 7.4%, for example, compared with 8.3% for a childless man of the same age.

The gap in absolute death risks between the two groups rose with increasing age, and was somewhat larger for men than it was for women.

At age 60 the difference in the one year risk of death was 0.06% among men and 0.16% among women. By the age of 90 these differences had risen to 1.47% among men and to 1.10% among women.

The associations found were evident among those who were married and unmarried, but seemed to be stronger among those who weren't married--at least among the men: the difference in death risk was 1.2% among unmarried men and 0.6% among those who were married.

Unmarried men might be relying more heavily on their children in the absence of a partner, suggest the researchers, by way of a possible explanation. They are also likely to be less well educated, whereas the opposite tends to be true of women, they add.

The associations were not affected by the sex of the child(ren), as has been suggested by previous research.

This could be because previous research has focused on the social benefits of having a daughter, whereas some other aspect of support, such as advocacy or navigating the health system, may matter more for staving off death, and sons could just as easily provide this, speculate the researchers.

This is an observational study, so no firm conclusions about cause and effect can be drawn. Nevertheless, the researchers write: "Our finding that the association grew stronger when parents became older is further in agreement with research suggesting that childless people face support deficits only towards the end of life."

http://bit.ly/2npMc6c

Metabolism may be older than life itself and start spontaneously

A set of chemical reactions occurring spontaneously in Earth's early chemical environments could have provided the foundations upon which life evolved.

By Linda Geddes

The discovery that a version of the Krebs cycle, which occurs in most living cells, can proceed in the absence of cellular proteins called enzymes suggests that metabolism is older than life itself.

Metabolism describes the fiendishly complex network of reactions that enable organisms to generate energy and the molecules they need to survive, grow and reproduce.

The Krebs cycle – also known as the tricarboxylic acid (TCA) cycle – is at the heart of this network. It describes a circular chain of reactions that generates precursors of amino acids and lipids used to build proteins and membranes, and molecules that help the cell to produce its energy.

But how did such a complex cycle develop in the first place?

One idea is that it began only after RNA, a fundamental building block of life, came into being. Metabolic reactions are catalysed by proteins called enzymes, for which RNA provides the template – at least in modern cells.

There is, however, a problem with this "RNA world" hypothesis: if the reactions didn't already occur immediately in early life forms and provide them with a survival advantage, then there would have been no selective pressure to drive the evolution of enzymes. Furthermore, RNA itself is made from products of metabolism.

So an alternative explanation is that the Krebs cycle existed from the outset, and early life forms simply adopted it and developed enzymes to make it more efficient.

However, modern enzymes that catalyse this cycle all use very different mechanisms to do so. The idea that the type of simple,

inorganic molecule that might have existed naturally in the early oceans could catalyse such a diverse set of reactions was once dismissed by RNA-world proponents as an "appeal to magic".

No magic trick

Now Markus Ralser at the Francis Crick Institute in London and his colleagues appear to have pulled such a molecule out of the hat, and no magic wand was necessary.

Ralser previously showed that two other crucial metabolic pathways – glycolysis and the pentose phosphate pathway – could be catalysed by metal ions present on early Earth rather than the enzymes that catalyse them in modern cells.

But sceptics of the "metabolism first" idea have pointed out that these pathways only seem to run in one direction, whereas earliest life would have needed both in order to work, and the starting material for these pathways, glucose, is unlikely to have existed on early Earth.

Unable to so far provide a satisfactory answer to these problems, Ralser has shifted his focus to the Krebs cycle. Unlike with glucose, the chemicals involved at various points of the Krebs cycle have been identified on meteorites and in laboratory recreations of Earth's early oceans – so we know they were around.

"We may not be able to solve where glucose comes from so easily," says Ralser. "But if we can provide proof that the Krebs cycle could originate from a single, non-enzymatic catalyst, then we would have a very strong case that what we say about the origins of metabolism is true."

So his team took chemicals involved in the Krebs cycle and exposed them to chemicals that would have been present in early ocean sediments. Nothing happened, until they introduced a compound called peroxydisulphate, a source of highly reactive agents called sulphate radicals.

This triggered a sequence of 24 chemical reactions that were very similar – although not identical – to those seen in the Krebs cycle today.

"The most surprising thing is that again a single molecule acts as the catalyst for all of the reactions we discovered," says Ralser. "The simplicity of it is super-exciting because it gives you a plausible feeling about how it could have all started."

Sulphate radicals would had been found in abundance near hydrothermal vents, which have been suggested as possible locations at which life started, or near to sulphur-rich sediments.

Ralser believes that these hardwired chemical reactions provided a template upon which the evolutionary machinery could build once it came into being.

Unfinished cycle

However, the enzyme-free Krebs cycle that Ralser observed isn't the complete biochemical cycle as it operates in modern cells. That may have come later, after enzymes evolved.

Furthermore, the sulphate-driven cycle has so far only been shown to run in one direction (the oxidative one). In some species, the Krebs cycle can also run in reverse and help to incorporate CO2 into the building of new carbohydrates. Some think it may therefore have been involved in early carbon fixation, in which case you'd expect to see the cycle spontaneously turning in this direction too.

Until researchers can demonstrate both these things, they cannot claim that metabolism came before cells and life, some experts think.

"This is a neat paper and the findings are striking and careful," says Nick Lane, an evolutionary biochemist at University College London. "But this is strictly the oxidative Krebs cycle, which is certainly not ancient. It probably became oxidative after the rise of molecular oxygen in the atmosphere.

"Before that, there was a reductive Krebs cycle, which fixed CO2 using H2, and which is still found in some ancient bacteria," says Lane. "They are not simulating the reductive Krebs cycle at all."

What's more, even if all three fundamental metabolic pathways – the Krebs cycle, pentose phosphate pathway and glycolysis – can proceed

in the absence of enzymes, there's still the question of how life's other components came into being.

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

"How do you bring these elements together in one environment and in non-extreme conditions, and make them work?" he asks. "This is still a big challenge."

Journal reference: Nature Ecology & Evolution: DOI: 10.1038/s41559-017-0083

http://bit.ly/2muJZkM

Light scattering spectroscopy helps doctors identify early pancreatic cancer

New optical tool predicts malignant potential of cysts with 95 percent accuracy, compared to 58 percent accuracy with current test

BOSTON - Pancreatic cancer has the lowest survival rate among all major cancers, largely because physicians lack diagnostic tools to detect the disease in its early, treatable stages. Now, a team of investigators led by Lev T. Perelman, PhD, Director of the Center for Advanced Biomedical Imaging and Photonics at Beth Israel Deaconess Medical Center (BIDMC), has developed a promising new tool capable of distinguishing between harmless pancreatic cysts and those with malignant potential with an overall accuracy of 95 percent. The team's preliminary data was published online today in the journal *Nature Biomedical Engineering*.

The new device uses light scattering spectroscopy (LSS) to detect the structural changes that occur in cancerous or pre-cancerous cells by bouncing light off tissues and analyzing the reflected spectrum. The results could help guide physicians' decision making when considering whether the presence of pancreatic cysts requires surgery, a high-risk procedure. Today, because of the lack of less-invasive diagnostic methods, more than half of these procedures turn out to have been unnecessary.

"About one-fifth of pancreatic cancers develop from cysts, but not all lesions are cancerous," said Perelman, who is also Professor of Medicine and Professor of Obstetrics, Gynecology and Reproductive Biology at Harvard Medical School. "Considering the high risk of pancreatic surgeries and the even higher mortality from untreated pancreatic cancers, there's an obvious need for new diagnostic methods to accurately identify the pancreatic cysts that need surgical intervention and those that do not."

In Perelman and colleagues' series of experiments, the LSS technique achieved 95 percent accuracy for identifying malignancy. Cytology - the only pre-operative test currently availably - is accurate only 58 percent of the time. While the new technique requires further testing, LSS could represent a major advance against pancreatic cancer.

"This tool is a technology that is transformative in the evaluation of pancreatic cysts," said co-lead author Douglas K. Pleskow, MD, Clinical Chief of the Division of Gastroenterology and Director of the Colon and Rectal Cancer Program at the Cancer Center at BIDMC. "It provides a high level of precision in the detection of potential malignant transformation of these cysts."

Pancreatic cysts are common, and today's high-definition scanning technologies like MRI and CT imaging are detecting them with increasing frequency. Despite their high resolution, these scanners provide doctors with limited information about cysts' malignant potential.

Currently, physicians rely on minimally-invasive fine needle aspiration (FNA) biopsies to test pancreatic cysts for malignancy. The biopsy removes fluid from the cysts, which is then analyzed for cancer cells and other telltale signs of the disease, a process called cytology. However, the test fails to detect cancer about half the time, leaving high-risk surgery as the current gold-standard means of diagnosing pancreatic cysts.

To test the accuracy of the LSS system, Perelman and colleagues collected and analyzed the reflected light from 13 cysts taken from

recent surgeries. Next, they compared their findings with the results from pre-operative imaging, FNA biopsies and post-operative tissues analysis. In all cases, the LSS diagnosis agreed with the post-operative analysis.

In a second experiment, the LSS tool was tested in 14 patients with pancreatic cysts who were undergoing the standard FNA biopsy. Measuring less than half a millimeter in diameter, the miniature experimental LSS fiber-optic probe was inserted in the FNA needle. Physicians spent two minutes or less measuring optical spectra from the internal cyst surface before collecting fluid from the cysts as part of the traditional biopsy. Out of nine patients whose cysts had been definitely diagnosed as either cancerous or benign, all were correctly identified by LSS.

Next, the researchers will assess the LSS system's accuracy by continuing to analyze post-operative tissues as they become available.

In addition to Perelman and Pleskow, the study authors include BIDMC investigators: co-corresponding author Le Qiu, PhD; lead author Lei Zhang, PhD; Vladimir Turzhitsky, PhD; Eric U. Yee, MD; Tyler M. Berzin, MD; Mandeep Sawhney, MD; Shweta Shinagare, MD; Edward Vitkin, PhD; Yuri Zakharov, PhD; Umar Khan; Fen Wang, MD; Jeffrey D. Goldsmith, MD; Ram Chuttani, MD; and Irving Itzkan, PhD; and Saveli Goldberg, PhD, of Massachusetts General Hospital.

This work was supported by the National Institutes of Health grants R01 EB003473 and R01 CA205431 and the National Science foundation grants CBET-1402926 and CBET-1605116.

About Beth Israel Deaconess Medical Center

http://bit.ly/2mLeRzb

Early Earth had a hazy, methane-filled atmosphere

Thick clouds of methane forced hydrogen to leave the atmosphere, enabling today's oxygen-rich air to develop

More than 2.4 billion years ago, Earth's atmosphere was inhospitable, filled with toxic gases that drove wildly fluctuating surface temperatures. Understanding how today's world of mild climates and breathable air took shape is a fundamental question in Earth science.

New research from the University of Maryland, the University of St. Andrews, NASA's Jet Propulsion Laboratory, the University of Leeds and the Blue Marble Space Institute of Science suggests that long ago, Earth's atmosphere spent about a million years filled with a methane-rich haze. This haze drove a large amount of hydrogen out of the atmosphere, clearing the way for massive amounts of oxygen to fill the air. This transformation resulted in an atmosphere much like the one that sustains life on Earth today.

The group's results, published March 13, 2017 in the early online edition of the Proceedings of the National Academy of Sciences, propose a new contributing cause for the Great Oxidation Event, which occurred 2.4 billion years ago, when oxygen concentrations in the Earth's atmosphere increased more than 10,000 times.

"The transformation of Earth's air from a toxic mix to a more welcoming, oxygen-rich atmosphere happened in a geological instant," said James Farquhar, a professor of geology at UMD and a co-author of the study. Farquhar also has an appointment at UMD's Earth System Science Interdisciplinary Center. "With this study, we finally have the first complete picture of how methane haze made this happen."

The researchers used detailed chemical records and sophisticated atmospheric models to reconstruct atmospheric chemistry during the time period immediately before the Great Oxidation Event. Their results suggest that ancient bacteria--the only life on Earth at the time--produced massive amounts of methane that reacted to fill the air with a thick haze, resembling the modern-day atmosphere of Saturn's moon Titan.

Previous studies by many of the same researchers had identified several such haze events early in Earth's history. But the current study is the first to show how rapidly these events began and how long they lasted.

"High methane levels meant that more hydrogen, the main gas preventing the build up of oxygen, could escape into outer space, paving the way for global oxygenation," said Aubrey Zerkle, a biogeochemist at the University of St. Andrews and a co-author of the study. "Our new dataset constitutes the highest resolution record of

Archean atmospheric chemistry ever produced, and paints a dramatic picture of Earth surface conditions before the oxygenation of our planet."

The methane haze persisted for about a million years. After enough hydrogen left the atmosphere, the right chemical conditions took over and the oxygen boom got underway, enabling the evolution of all multicellular life.

The key to the researchers' analysis was the discovery of anomalous patterns of sulfur isotopes in the geochemical records from this time. Sulfur isotopes are often used as a proxy to reconstruct ancient atmospheric conditions, but previous investigations into the time period in question had not revealed anything too unusual.

"Reconstructing the evolution of atmospheric chemistry has long been the focus of geochemical research," said Gareth Izon, lead author of the study, who contributed to the research while a postdoctoral researcher at St. Andrews and is now a postdoctoral researcher at the Massachusetts Institute of Technology. "Our new data show that the chemical composition of the atmosphere was dynamic and, at least in the prelude to the Great Oxidation Event, hypersensitive to biological regulation."

This release is based on text provided by the University of St. Andrews.

The research paper, "Biological regulation of atmospheric chemistry en route to planetary oxygenation," Gareth Izon, Aubrey Zerkle, Kenneth Williford, James Farquar, Simon Poulton, and Mark Claire, was published March 13, 2017 in the Proceedings of the National Academy of Sciences.

This work was supported by the Natural Environment Research Council (Award Nos. NE/H016805 and NE/J023485), the Scottish Alliance for Geoscience, Environment and Society, The Geological Society of London's Alan and Charlotte Welch Fund, NASA (Award No. NNX12AD91G), The Royal Society, and the European Research Council (Award No. 678812). The content of this article does not necessarily reflect the views of these organizations.

http://bit.ly/2mv7N9i

For hospitalized patients, spending more on care doesn't buy better health

Spending more doesn't always mean you get better health

Hospitalized patients treated by physicians who order more or more expensive tests and procedures are just as likely to be readmitted or to die as patients treated by doctors who order fewer or less expensive tests, according to research led by Harvard Medical School and the Harvard T.H. Chan School of Public Health.

The study, published in JAMA Internal Medicine on March 13, is believed to be the first to examine the impact of individual physicians' spending patterns on patient outcomes.

"If you spend more money on a car or a TV, you tend to get a nicer car or a better TV," said study senior author Anupam B. Jena, the Ruth L. Newhouse Associate Professor of Health Care Policy at Harvard Medical School. "Our findings show that's not the case when it comes to medical care. Spending more doesn't always mean you get better health."

Research on variation in spending and outcomes between geographic regions and between hospitals has produced mixed results, but most evidence suggests that greater spending does not reliably translate into better outcomes.

What has been missing from the picture, the authors said, is how individual physician spending within the same hospital translates into patient health. That insight, the researchers added, is a key piece of the puzzle because individual doctors make most of the clinical decisions that drive spending and affect outcomes.

"Before now, most of the research and efforts aimed at cutting spending and improving the value of care have been aimed at hospitals, health systems and groups of doctors," said the lead author Yusuke Tsugawa, a research associate at the Harvard T.H. Chan School of Public Health. "The differences between hospitals and regions are important, but they're only part of the puzzle. Our findings show how important it is to consider the differences between individual doctors in any effort to improve health care."

The researchers analyzed outcomes among Medicare fee-for-service patients aged 65 years and older who were hospitalized for a

nonelective medical condition and treated by an internist between 2011 and 2014. Health care spending varied more across individual physicians within a single hospital than across hospitals, even after accounting for differences between hospitals and patient populations, the data showed.

Overall, 8.4 percent of the total variation in health care spending could be explained by differences between individual physicians, compared to 7 percent explained by differences between hospitals.

Next, researchers examined the link between physician spending and patient outcomes. When they compared lower- and higher-spending physicians, the researchers found no difference in 30-day patient mortality, nor did they see a difference in readmissions, two factors regarded as key measures of quality of care.

Jena, who is also a physician at Massachusetts General Hospital, cautioned that it's too soon to say whether the results mean that higher-spending physicians could simply spend less with no ill effects for patients.

"Say you have two painters. One usually takes two hours to paint a room, and one takes six hours. You can ask the slow painter to hurry up, but you might end up with a room that's sloppily painted, or with one of the walls the wrong color," Jena said. "That's obviously a situation we want to avoid in health care."

It could be that some doctors don't fully consider the costs associated with the tests and procedures they order, Jena said, and so policymakers or insurers could create incentives to curb some of the more wasteful spending. On the other hand, Jena said, some doctors might just be less efficient than others and may need additional resources to arrive at a proper diagnosis or an effective treatment.

Whatever the causes of the variation, Jena added, these findings underscore the impact of decisions made by individual doctors on health care spending.

This research was supported with funding from the Office of the Director, National Institutes of Health (NIH Early Independence Award, Grant 1DP5OD017897-01) and by the Social Science Research Council and St. Luke's International University (Tokyo, Japan).

http://bbc.in/2mRX2R2

New drug for one in five breast cancers

Many more women could be helped by a new type of breast cancer drug, say experts

Biological therapies are currently only available as part of clinical trials, but hold great promise

Experts estimate as many as one in five patients might benefit

By Michelle Roberts Health editor, BBC News online

Around 10,000 women a year in the UK might benefit from a new type of breast cancer treatment, say scientists. Biological therapies can help fight breast cancers caused by rare, inherited genetic errors like the BRCA one actress Angelina Jolie carries.

Now a new study by experts at the Wellcome Trust Sanger Institute suggests these targeted drugs may also work in many other women who do not have these risky genes. The drugs could be effective in one in five breast cancers, say the researchers. That's 20% of patients - far more than the 1 to 5% who develop the cancer alongside having faulty BRCA genes.

One biological therapy or PARP inhibitor, called olaparib, is already used on the NHS to treat advanced ovarian cancer. It is not yet approved as a breast cancer drug, although some UK women are taking it in clinical trials.

For the latest work, published in the journal Nature Medicine, the researchers looked at the genetic make-up of breast cancer in 560 different patients. They found a significant proportion of them had genetic errors or "mutational signatures" that were very similar to faulty BRCA. Given the close similarity, these cancers might be treatable with biological therapies too, they reasoned.

They recommended clinical trials to confirm this.

Baroness Delyth Morgan, from Breast Cancer Now, called the early results "a revelation". "We hope it could now lead to a watershed moment for the use of mutational signatures in treating the disease," she said.

One of the researchers, Dr Helen Davies, said there was also the potential to treat other types of cancers with these drugs.

Biological therapies have already had some promising results for treating prostate cancer. They change the way cells work and help the body control the growth of cancer.

High-risk genes

Carrying certain gene mutations, like faulty BRCA, increases a woman's risk of developing breast cancer, although it does not mean she will definitely go on to get cancer.

Some women - like Angelina Jolie - opt to have surgery to have their breasts removed to lower their lifetime risk.

Vicki Gilbert, 54 and from Swindon, found out she was carrying high-risk genes - but only after she developed breast cancer.

"I had been thinking about getting tested anyway because there was quite a lot of cancer on one side of my family. But then I was diagnosed with breast cancer out of the blue, before I even had a chance to go for the genetic screening."

She says finding out, even after the event, was helpful. "When you get cancer you do think 'Why me?' I don't know for sure if it was because of the genes that I inherited. That would be impossible to say. But, for practical reasons, it is useful to know that I carry these genes."

Vicki has been free of cancer for around seven years, but still has regular checks because of her increased genetic risk.

Women can lower their lifetime risk of breast cancer by exercising regularly, eating a good diet, maintaining a healthy weight, avoiding cigarettes and limiting how much alcohol they drink.

http://bit.ly/2mvwtNS

Men face greater risk of death following osteoporosis-related fractures

Men face a greater risk of mortality following a fracture related to osteoporosis

Men face a greater risk of mortality following a fracture related to osteoporosis, a common disease where the bones become weak and brittle, according to new research presented today at the 2017 Annual Meeting of the American Academy of Orthopaedic Surgeons (AAOS).

"Although women are more likely to sustain an initial, osteoporosis-related 'fragility fracture,' men have similar rates of incurring a subsequent fracture and are at greater risk for mortality after these injuries," said lead study author Alan Zhang, MD, an orthopaedic surgeon and assistant professor at the University of California, San Francisco.

Osteoporosis is a major health problem affecting more than 44 million Americans and contributing to an estimated 2 million bone fractures per year. Because diminished estrogen can contribute to bone loss, menopause-age women have traditionally been the focus of osteoporosis prevention efforts.

In this study, researchers reviewed the Medicare Standard Analytic Files (SAF) database to identify patients, age 65 and older, who had a diagnosis of osteoporosis and sustained a fragility fracture between 2005 and 2009. Patient records were stratified by sex and the location of a first fracture. In addition, the incidence of second fragility fractures was compared between men and women during a three-year follow-up period, as well one-year mortality rates.

Of the more than 1 million patients identified with a diagnosis of osteoporosis in the analysis, 87 percent were female and 13 percent male. Among these patients:

Women had a five-fold higher risk for an initial fragility fracture compared to men, and yet the relative risk for a subsequent fragility fracture within three years of the first fracture was slightly lower for women compared to men.

Men who required surgical treatment for an initial fragility fracture were more likely to suffer a subsequent fragility fracture within three years. The exception was in men who suffered a vertebral (spinal) compression fracture (in these instances the risk was comparable).

Men had higher one-year mortality rates for almost all fracture types studied (18.7 percent in men versus 13.9 percent in women). The only exception was ankle fractures where one-year mortality rates between

men and women were comparable (8.1 percent for men and 8.4 percent for women).

"The key findings from this study show that patient sex can affect the risk for sustaining a fragility fracture related to osteoporosis," said Dr. Zhang. "These findings may be used to better counsel patients after an initial fragility fracture and to improve predictive tools for monitoring subsequent injuries."

Last year, AAOS joined the American Orthopaedic Association, the Orthopaedic Trauma Association (OTA) and the International Geriatric Fracture Society (IGFS) in drafting and approving new patient guidelines for preventing fragility fractures. "Orthopaedic Care of Patients with Fragility Fractures" recommends that physicians proactively screen, monitor, and if necessary, assist in getting treatment for all elderly and other at-risk patients for osteoporosis following an initial bone fracture to prevent subsequent fractures.

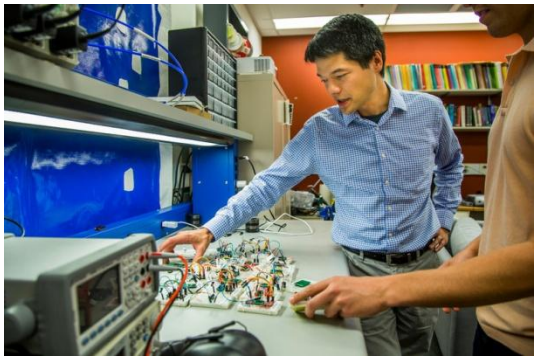
http://nyti.ms/2n2pyQk

It's Possible to Hack a Phone With Sound Waves, Researchers Show

A security loophole that might seem harmless points to the broader risks that come with technology

By JOHN MARKOFF MARCH 14, 2017

SAN FRANCISCO — A security loophole that would allow someone to add extra steps to the counter on your Fitbit monitor might seem harmless. But researchers say it points to the broader risks that come with technology's embedding into the nooks of our lives.



Kevin Fu and other researchers have found a way to take control of or influence devices using a standard component in cellphones and other gadgets. **Joseph Xu/University of Michigan**

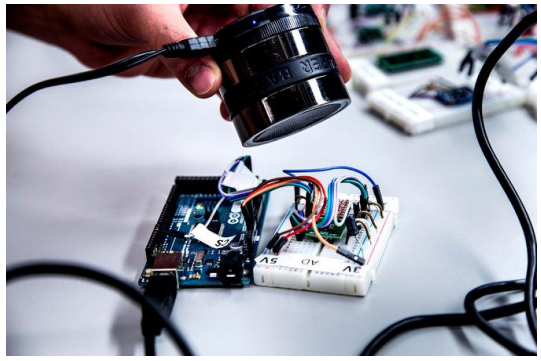
On Tuesday, a group of computer security researchers at the University of Michigan and the University of South Carolina will demonstrate that they have found a vulnerability that allows them to take control of or surreptitiously influence devices through the tiny accelerometers that are standard components in consumer products like smartphones, fitness monitors and even automobiles.

In their paper, the researchers describe how they added fake steps to a Fitbit fitness monitor and played a "malicious" music file from the speaker of a smartphone to control the phone's accelerometer. That allowed them to interfere with software that relies on the smartphone, like an app used to pilot a radio-controlled toy car.

"It's like the opera singer who hits the note to break a wine glass, only in our case, we can spell out words" and enter commands rather than just shut down the phone, said Kevin Fu, an author of the paper, who is also an associate professor of electrical engineering and computer science at the University of Michigan and the chief executive of Virta Labs, a company that focuses on cybersecurity in health care. "You can think of it as a musical virus."

The flaw, which the researchers found in more than half of the 20 commercial brands from five chip makers they tested, illustrates the security challenges that have emerged as robots and other kinds of digital appliances have begun to move around in the world.

With dozens of start-ups and large transportation companies pushing to develop self-driving cars and trucks, undetected vulnerabilities that might allow an attacker to remotely control vehicles are an unnerving possibility.



A speaker can make tones that fool a sensor and cause a microprocessor to accept the sensor readings. **Joseph Xu/University of Michigan**

Still, computer security researchers said the discovery was not a sky-is-falling bug but rather a revealing window into the cybersecurity challenges inherent in complex systems in which analog and digital components can interact in unexpected ways.

"The whole world of security is about unintended interactions," said Paul Kocher, a cryptographer and a former executive at the chip company Rambus.

Accelerometers are instruments that measure acceleration and are frequently manufactured as silicon chip-based devices known as microelectromechanical systems, or MEMS. Accelerometers are used for navigating, for determining the orientation of a tablet computer and for measuring distance traveled in fitness monitors such as Fitbits.

In the case of the toy car, the researchers did not actually compromise the car's microprocessor, but they controlled the car by forcing the accelerometer to produce false readings. They exploited the fact that a smartphone application relies on the accelerometer to control the car.

While toy cars might seem like trivial examples, there are other, darker possibilities. If an accelerometer was designed to control the automation of insulin dosage in a diabetic patient, for example, that might make it possible to tamper with the system that controlled the correct dosage.

Dr. Fu has researched the cybersecurity risks of medical devices, including a demonstration of the potential to wirelessly introduce fatal heart rhythms into a pacemaker.

He said the current research was inspired by a discussion in his group about a previous study in which drones were disabled with music. He added that earlier research demonstrated denial-of-service attacks that used sound to disable accelerometers.

In 2014, security researchers at Stanford University demonstrated how an accelerometer could be used surreptitiously as a rudimentary microphone, for example. And in 2011, a group from the Massachusetts Institute of Technology and the Georgia Institute of Technology demonstrated the use of an accelerometer in a smartphone to decode roughly 80 percent of the words being typed on a nearby computer keyboard by capturing vibrations from the keyboard.

In the case of the research by the University of Michigan and the University of South Carolina, scientists stopped the accelerometer from functioning and changed its behavior.

In testing 20 accelerometer models from five manufacturers, they affected the information or output from 75 percent of the devices tested and controlled the output in 65 percent of the devices.

The Department of Homeland Security was expected to issue a security advisory alert Tuesday for chips produced by the semiconductor companies documented in the paper, Dr. Fu said. The five chip makers were Analog Devices, Bosch, InvenSense, Murata Manufacturing and STMicroelectronics.

The paper, which will be presented at the IEEE European Symposium on Security and Privacy in Paris next month, also documents hardware and software changes manufacturers could make to protect against the flaws the researchers discovered.

http://bit.ly/2mSbjNA

Experimental Ebola vaccine regimen induced durable immune response, study finds

Antibodies to Ebola present in all participants one year after vaccination

WHAT: A two-vaccine regimen intended to protect against Ebola virus disease induced an immune response that persisted for approximately one year in healthy adult volunteers, according to results from a Phase 1 clinical trial published in the March 14th issue of the Journal of the American Medical Association. The investigational vaccines included Ad26.ZEBOV, developed by Janssen Vaccines & Prevention B.V., one of the Janssen Pharmaceutical Companies of Johnson & Johnson, and MVA-BN-Filo, developed by Bavarian Nordic. The National Institute of Allergy and Infectious Diseases (NIAID), part of the National Institutes of Health (NIH), supported the development and testing of the experimental

vaccines, beginning with early non-clinical and manufacturing process development.

Both of the vaccines in the regimen use harmless viral vectors, or carriers, to deliver proteins of the Ebola virus, which prompt an immune response. Ad26.ZEBOV uses a modified adenovirus vector to express proteins from Zaire ebolavirus, which was the species responsible for the 2014-2015 outbreak in West Africa. MVA-BN-Filo uses a modified vaccinia virus Ankara vector to express proteins from various species of Ebola virus, as well as the related Marburg virus.

The Phase 1 trial enrolled healthy participants ages 18-50 years in the United Kingdom and was conducted by the Oxford Vaccine Group at the University of Oxford. Participants were selected randomly to receive either the two-vaccine regimen or placebo (saltwater injections). Previously reported initial results showed the two-vaccine regimen is safe, well-tolerated and induced immune responses in participants eight months after immunization.

Of the 75 participants who received the vaccine regimen, 64 remained in the study for a follow-up visit on day 360. No serious vaccine-associated adverse events were observed, and all 64 participants maintained antibodies to Ebola virus at day 360. The researchers note additional research is necessary to assess the durability of immunity beyond one year and the immune response to booster doses of vaccine.

ARTICLE:

R Winslow et al. Immune Responses to Novel Adenovirus Type 26 and Modified Vaccinia Virus Ankara-Vectored Ebola Vaccines at 1 Year. JAMA DOI: 10.1001/jama.2016.20644 (2017).

http://bbc.in/2mSFhRt

'Oldest plants on Earth' discovered

The origins of plants may go back hundreds of millions of years earlier than previously thought, according to fossil evidence.

By Helen Briggs BBC News

Ancient rocks from India suggest plants resembling red algae lived 1.6 billion years ago in what was then shallow sea. The discovery may overturn ideas of when relatively advanced life evolved, say scientists in Sweden. They identified parts of chloroplasts, structures within plant cells involved in photosynthesis.

The earliest signs of life on Earth are at least 3.5 billion years old.

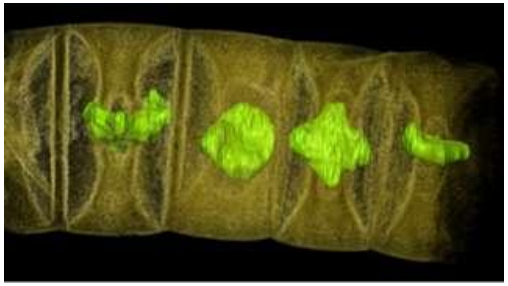


Image copyright Stefan Bengtson

The first single-celled microscopic life forms evolved into larger multi-cellular eukaryotic organisms (made up of cells containing a nucleus and other structures within a membrane).

Therese Sallstedt of the Swedish Museum of Natural History discovered some of the fossils. She described them as "the oldest fossil plants that we know of on Earth in the form of 1.6 billion year old red algae".

"They show us that advanced life in the form of eukaryotes (like plants, fungi and us humans/animals) have a much deeper history on Earth than what we previously have thought," she told BBC News.

Tree of life

The scientists found thread-like fossils and more complex "fleshy" colonies in sedimentary rock from central India. Both have characteristics of modern red algae, a type of seaweed.

Co-researcher Prof Stefan Bengtson of the Swedish Museum of Natural History added: "You cannot be 100% sure about material this ancient, as there is no DNA remaining, but the characters agree quite well with the morphology and structure of red algae."

The oldest known red algae before the present discovery date back 1.2 billion years. The Indian fossils are 400 million years older, suggesting that the early branches of the tree of life began much earlier than previously thought.

Claims of ancient life are always controversial. Without DNA evidence, confirmation must rest on whether more fossils can be

found. There is also debate over whether red algae belong in the plant kingdom or in a class of their own.

Modern red algae is perhaps best known for two commercial products - gelatinous texturing agents used in making ice cream - and nori - the seaweed used to wrap sushi.

The research is published in the journal, PLOS Biology.

http://bit.ly/2mSjq5f

Bacteria-Enriched Lotion Battles Skin Infections

Pilot study shows possibility of putting good bacteria into a lotion and spreading that lotion onto the skin to fight off bad bacteria

By Jenna Flannigan, Live Science Contributor

Human skin is home to a multitude of microbes, including some that are helpful and some that could potentially be harmful. Now, a small pilot study shows that it might be possible to harness the good bacteria, put them into a lotion and then spread that lotion onto the skin to fight off the bad bacteria.

In the study, researchers took bacterial samples from patients' skin, picked out certain species and cultured them in a lab, and then put these bacteria into a lotion. They found that, for five patients with a skin condition, the bacteria-rich lotion protected them against infections by destroying harmful germs on their skin.

The findings show that "bacteria have a very important role to play in our immune defense," Dr. Richard Gallo, chair of the Department of Dermatology at the University of California, San Diego, and one of the study authors, told Live Science. The study was published Feb. 22 in the journal Science Translational Medicine.

In the study, the researchers looked at patients with eczema, a condition which causes itchy, red, inflamed skin. In some people, the condition is chronic, which means that it recurs again and again.

Eczema also makes patients more susceptible to staph infections. ("Staph" is short for the bacterial group known as Staphylococcus.)

The researchers found that the patients who had persistent eczema tended to be deficient in the friendly bacteria that kill a type of bacteria called Staphylococcus aureus. In contrast, people without eczema have an abundance of the helpful bacteria, according to the study.

The researchers analyzed the friendly strains of bacteria, which are also forms of staph, but types that do not cause harm. They found that these bacteria produce two natural antibiotic agents, known as "antimicrobial peptides," according to the study. The human body also makes these substances, but the new study suggests that the bacteria on the skin do a better job of producing them, the researchers said.

In experiments using pig skin and mice, the researchers found that the "good" bacteria strongly inhibited the growth of several harmful staph strains, including methicillin-resistant Staphylococcus aureus, or MRSA, a pathogen that is resistant to multiple types of antibiotics.

What's more, the animal experiments showed that even as pathogens were killed off, other colonies of bacteria continued to thrive. Conversely, traditional antibiotics tend to destroy both helpful and harmful germs all at once, potentially weakening people's immune response.

The five patients in the study had only tiny amounts of the good bacteria on their skin, and all tested positive for the type of staph that causes infections. The researchers swabbed the patients' skin, and cultured the few friendly strains of bacteria they could find. Then, they grew more of these bacteria in the lab, and added the microbes to a lotion.

To test whether the good bacteria would kill harmful bacteria, and help prevent infections, the researchers had the patients apply their own personalized microbe-containing lotion to one arm and regular moisturizer to the other. After 24 hours, in all five patients, only the arms treated with the microbe-containing lotion showed near total improvement, and in two patients, the staph pathogens were destroyed entirely.

While the results of this test were promising, questions remain.

First, the study was very small, so the results need to be confirmed in larger studies. In addition, although the lotion prevented skin infections, it's not clear whether it would improve the patients' skin condition, Gallo said.

Researchers also don't know if the patients were "cured" of the staph pathogen, or if they would need to keep reapplying the lotion in the future.

For this study, the bacteria in the lotions came from the patients' own skin, but that's already changing. "The next-stage trial that is underway now is to just use one universal strain and apply it to everyone," Gallo said.

http://bit.ly/2nviBoz

Researcher invents lip motion password technology

New technology utilises a person's lip motions to create a password

The use of biometric data such as fingerprints to unlock mobile devices and verify identity at immigration and customs counters are used around the world. Despite its wide application, once the scan is stolen or hacked, the owner can't change his/her fingerprints and has to look for another identity security system. With this in mind, a scholar at HKBU has invented a new technology for lip motion password recognition, which utilises a person's lip motions to create a password. This system verifies a person's identity by simultaneously matching the password content with the underlying behavioural characteristics of lip movement. Nobody can mimic a user's lip movement when speaking the password, which can be changed at any time.

HKBU's Science Professor Cheung Yiu-ming, in charge of the research, said the new technique has a number of advantages over conventional security access control methods:

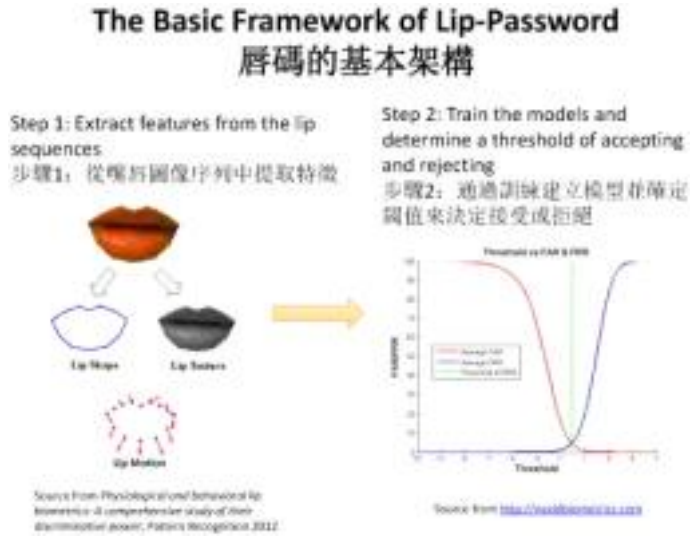
(1) The dynamic characteristics of lip motions are resistant to mimicry, so a lip password can be used singly for speaker verification, as it is able to detect and reject a wrong password uttered by the user or the correct password spoken by an imposter;

(2) Verification based on a combination of lip motions and password content ensures that access control is doubly secure;

(3) Compared with traditional voice-based authentication, the acquisition and analysis of lip movements is less susceptible to background noise and distance, moreover, it can even be used by a speech-impaired person;

(4) A user can reset the lip password in a timely manner to strengthen security;

(5) There is no language boundary; in other words, a person from any country can use this lip password verification system.



A diagram shows the basic concept of lip motion password and how it works.
Hong Kong Baptist University

Professor Cheung said: "The same password spoken by two persons is different and a learning system can distinguish them." The study adopted a computational learning model that extracts the visual features of lip shape, texture and movement to characterise lip sequence. Samples of lip sequence are collected and analysed to train the models and determine the threshold of accepting and rejecting a spoken password.

The potential application of this new patented technology includes, but is not limited to, financial transaction authentication including electronic payment using mobile devices, transactions at ATM machines, and credit card user passwords. It can also be applied to enhance the security access control system currently used in entrances of companies or private premises.

In addition, lip password can be used together with other biometrics to enhance the security level of systems. For instance, lip password can be combined with face recognition, whereby the problem of spoofing face recognition with 3-D masks in personal identity verification would be solved.

http://bit.ly/2nvkj9F

Topical curcumin gel effective in treating burns and scalds

What is the effect of Topical Curcumin Gel for treating burns and scalds?

In a recent research paper, published in the open access journal BioDiscovery, Dr. Madalene Heng, Clinical Professor of Dermatology at the David Geffen School of Medicine, stresses that use of topical curcumin gel for treating skin problems, like burns and scalds, is very different, and appears to work more effectively, when compared to taking curcumin tablets by mouth for other conditions.

"Curcumin gel appears to work much better when used on the skin because the gel preparation allows curcumin to penetrate the skin, inhibit phosphorylase kinase and reduce inflammation," explains Dr Heng.

In this report, use of curcumin after burns and scalds were found to reduce the severity of the injury, lessen pain and inflammation, and improve healing with less than expected scarring, or even no scarring, of the affected skin. Dr. Heng reports her experience using curcumin gel on such injuries using three examples of patients treated after burns and scalds, and provides a detailed explanation why topical curcumin may work on such injuries.

Curcumin is an ingredient found in the common spice turmeric. Turmeric has been used as a spice for centuries in many Eastern countries and gives well known dishes, such as curry, their typical yellow-gold color. The spice has also been used for cosmetic and medical purposes for just as long in these countries.

In recent years, the medicinal value of curcumin has been the subject of intense scientific studies, with publication numbering in the thousands, looking into the possible beneficial effects of this natural product on many kinds of affliction in humans.

This study published reports that topical curcumin gel applied soon after mild to moderate burns and scalds appears to be remarkably effective in relieving symptoms and improved healing of the affected skin.

"When taken by mouth, curcumin is very poorly absorbed into the body, and may not work as well," notes Dr. Heng. "Nonetheless, our tests have shown that when the substance is used in a topical gel, the effect is notable."

The author of the study believes that the effectiveness of curcumin gel on the skin - or topical curcumin - is related to its potent anti-inflammatory activity. Based on studies that she has done both in the laboratory and in patients over 25 years, the key to curcumin's effectiveness on burns and scalds is that it is a natural inhibitor of an enzyme called phosphorylase kinase.

This enzyme in humans has many important functions, including its involvement in wound healing. Wound healing is the vital process that enables healing of tissues after injury.

The process goes through a sequence of acute and chronic inflammatory events, during which there is redness, swelling, pain and then healing, often with scarring in the case of burns and scalds of the skin. The sequence is started by the release of phosphorylase kinase about 5 mins after injury, which activates over 200 genes that are involved in wound healing.

Dr. Heng uses curcumin gel for burns, scalds and other skin conditions as complementary treatment, in addition to standard treatment usually recommended for such conditions.

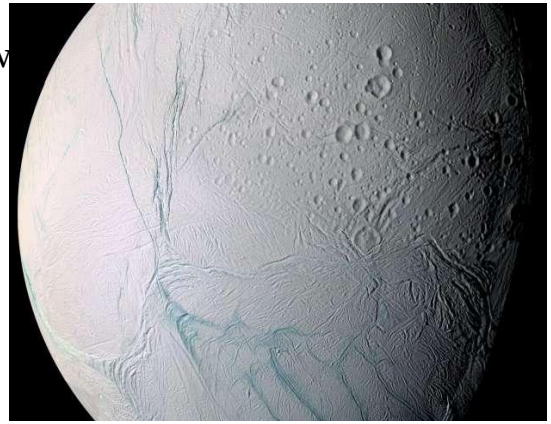
Heng M (2017) Phosphorylase Kinase Inhibition Therapy in Burns and Scalds. BioDiscovery 20: e11207. https://doi.org/10.3897/biodiscovery.20.e11207

http://bit.ly/2ndrCFn

Enceladus' south pole is warm under the frost

New study shows that the moon is warmer than expected just a few metres below its icy surface

Over the past decade, the international Cassini mission has revealed intense activity at the southern pole of Saturn's icy moon, Enceladus, with warm fractures venting water-rich jets that hint at an underground sea. A new study, based on microwave observations of this region, shows that the moon is warmer than expected just a few metres below its icy surface. This suggests that heat is produced over a broad area in this polar region and transported under the crust, and that Enceladus' reservoir of liquid water might be lurking only a few kilometres beneath.



As it swooped past the south pole of Saturn's moon Enceladus on 14 July 2005, Cassini acquired high-resolution views of this puzzling ice world. From afar, Enceladus exhibits a bizarre mixture of softened craters and complex, fractured terrains. This large mosaic of 21 images has been arranged to provide a full-disc view of the anti-Saturn hemisphere on Enceladus. This mosaic is a false-colour view highlighting the long fractures – tiger stripes – in blue.
NASA/JPL/Space Science Institute

In 2005, observations by the NASA/ESA/ASI Cassini mission revealed plumes of water vapour and ice spraying into space from the south pole of Enceladus, the sixth-largest moon of Saturn. These jets originate from the so-called 'tiger stripes' – four warm fractures in the moon's icy surface. The salty composition of these jets points to an underground sea of liquid water that might interact with Enceladus' rocky core, similar to the sub-surface ocean that is thought to exist on Jupiter's moon, Europa.

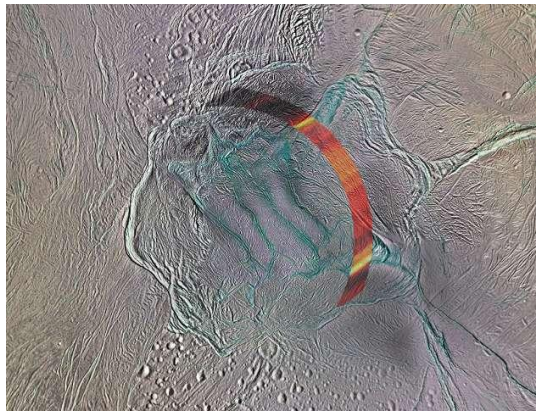
Many of Cassini's flybys of Enceladus have been dedicated to understanding the structure of the interior of this fascinating body and its potentially habitable water reservoir. Now, a study based on data collected during a close flyby in 2011 indicates that the moon's hidden sea might be closer to the surface than previously thought.

"During this flyby, we obtained the first and, unfortunately, only high-resolution observations of Enceladus' south pole at microwave wavelengths," says Alice Le Gall from Laboratoire Atmosphères, Milieux, Observations Spatiales (LATMOS), and Université Versailles Saint-Quentin (UVSQ), France. Alice is an associate member of the Cassini RADAR instrument team and the lead scientist of the new study, published today in *Nature Astronomy*.

"These observations provide a unique insight into what is going on beneath the surface. They show that the first few metres below the surface of the area that we investigated, although at a glacial 50-60 K, are much warmer than we had expected: likely up to 20 K warmer in some places," she adds

"This cannot be explained only as a result of the Sun's illumination and, to a lesser extent, Saturn's heating so there must be an additional source of heat."

The detected heat appears to be lying under a much colder layer of frost, as no similar anomaly was found in infrared observations of the same region – these probe the temperature of the surface but are not sensitive to what is underneath.



The South Pole region on Enceladus, the sixth-largest moon of Saturn, imaged by the Imaging Science Subsystem (ISS) on the international Cassini mission.
NASA/JPL-Caltech/Space Science Institute; Acknowledgement: A. Lucas

The observations used by Alice and her collaborators cover a narrow, arc-shaped swathe of the southern polar region, about 500 km long and 25 km wide, and located just 30 km to 50 km north of the tiger-

stripe fractures. Because of operational constraints of the 2011 flyby, it was not possible to obtain microwave observations of the active fractures themselves. This had the benefit of allowing the scientists to observe that the thermally anomalous terrains of Enceladus extend well beyond the tiger stripes.

"The thermal anomaly we see at microwave wavelengths is especially pronounced over three fractures that are not unlike the tiger stripes, except that they don't seem to be the source of jets at the moment," Alice says.

These seemingly dormant fractures lying above the warm, underground sea point to a dynamic character of Enceladus' geology: the moon may have experienced several episodes of activity at different locations during its past history.

Even if the observations cover only a small patch of the southern polar terrains, it is likely that the entire region is warm underneath and Enceladus' ocean could be a mere 2 km under the icy surface. The finding agrees well with the results of a recent study, led by Ondrej Cadek and published in 2016, which estimated the thickness of the crust on Enceladus. With an average depth of 18–22 km, the ice shell appears to reduce to less than 5 km at the south pole.

Alice and her collaborators think that the underground heating source is linked to the tidal cycle of the moon along its eccentric orbit around Saturn. This induces stress compressions and deformations on the crust, leading to the formation of faults and fractures while at the same time heating up the sub-surface layers. In this scenario, the thinner icy crust in the south pole region is subject to a larger tidal deformation that, in turn, releases more heat and contributes to maintaining the underground water in liquid form.

"This discovery opens new perspectives to investigate the emergence of habitable conditions on the icy moons of the gas giant planets," says Nicolas Altobelli, ESA's Project Scientist for Cassini–Huygens.

"If Enceladus' underground sea is really as close to the surface as this study indicates, then a future mission to this moon carrying an ice-penetrating radar sounding instrument might be able to detect it."

More information: *A. Le Gall et al. Thermally anomalous features in the subsurface of Enceladus's south polar terrain, Nature Astronomy (2017).* DOI: 10.1038/s41550-017-0063

http://bit.ly/2nvPQs5

Did humans create the Sahara desert?

New research challenges the idea that changes in the Earth's orbit triggered Sahara desertification

New research investigating the transition of the Sahara from a lush, green landscape 10,000 years ago to the arid conditions found today, suggests that humans may have played an active role in its desertification.

The desertification of the Sahara has long been a target for scientists trying to understand climate and ecological tipping points. A new paper published in Frontiers in Earth Science by archeologist Dr. David Wright, from Seoul National University, challenges the conclusions of most studies done to date that point to changes in the Earth's orbit or natural changes in vegetation as the major driving forces.

"In East Asia there are long established theories of how Neolithic populations changed the landscape so profoundly that monsoons stopped penetrating so far inland", explains Wright, also noting in his paper that evidence of human-driven ecological and climatic change has been documented in Europe, North America and New Zealand. Wright believed that similar scenarios could also apply to the Sahara.

To test his hypothesis, Wright reviewed archaeological evidence documenting the first appearances of pastoralism across the Saharan region, and compared this with records showing the spread of scrub vegetation, an indicator of an ecological shift towards desert-like conditions. The findings confirmed his thoughts; beginning approximately 8,000 years ago in the regions surrounding the Nile

River, pastoral communities began to appear and spread westward, in each case at the same time as an increase in scrub vegetation.

Growing agricultural addiction had a severe effect on the region's ecology. As more vegetation was removed by the introduction of livestock, it increased the albedo (the amount of sunlight that reflects off the earth's surface) of the land, which in turn influenced atmospheric conditions sufficiently to reduce monsoon rainfall. The weakening monsoons caused further desertification and vegetation loss, promoting a feedback loop which eventually spread over the entirety of the modern Sahara.

There is much work still to do to fill in the gaps, but Wright believes that a wealth of information lies hidden beneath the surface: "There were lakes everywhere in the Sahara at this time, and they will have the records of the changing vegetation. We need to drill down into these former lake beds to get the vegetation records, look at the archaeology, and see what people were doing there. It is very difficult to model the effect of vegetation on climate systems. It is our job as archaeologists and ecologists to go out and get the data, to help to make more sophisticated models".

Despite taking place several thousands of years ago, the implications of humans being responsible for environmental and climatic degradation are easy to see. With approximately 15% of the world's population living in desert regions, Wright stresses the importance of his findings: "the implications for how we change ecological systems have a direct impact on whether humans will be able to survive indefinitely in arid environments".

http://bit.ly/2nJf0Cu

Bowel cancer medication could help combat early-onset Parkinson's disease

Medical Research Council-funded University of Leicester study shows folinic acid can protect neurons in fruit flies

People with certain forms of early-onset Parkinson's disease could potentially benefit from taking a medication used to treat certain forms of cancer, according to new research by University of Leicester scientists and funded by the Medical Research Council.

The study, which has been published in Science Matters, suggests that folinic acid, which is used in medications to treat bowel cancer, can also protect neurons associated with Parkinson's disease in fruit flies.

Dr Miguel Martins from the MRC Toxicology Unit at the University of Leicester explained: "Parkinson's disease is a disabling disorder for which no cure is yet available; further, after dopaminergic neurons are lost, only a few palliative treatment options for Parkinson's symptoms are available. Therefore, treatments that either prevent or delay the onset of the disease at an early stage are needed.

"Folinic acid is already approved and used for applications in the clinic as an adjuvant during chemotherapy and can be administered orally, as a dietary supplement, or intravenously.

"Thus, the drug safety risk is low, and drug development for repurposing folinic acid as a treatment for Parkinson's disease would be faster than for a novel drug.

"With this in mind, it seems worthwhile to further test the supplementation of folinic acid in clinical trials with human participants as a potential preventative or palliative therapeutic for PD and to expand the repertoire of treatment options."

The researchers studied fruit flies with faulty mitochondria caused by a mutation that mimics Parkinson's disease in humans. Lab experiments, like this, allow us to draw conclusions about the effect of folinic acid on neurons in fruit flies.

Previous research by the team has shown that folic acid protects neurons in models of Parkinson's disease. Folinic acid is related to folic acid but is metabolically more active.

In contrast to folic acid, folinic acid taken orally can penetrate into the human brain.

The paper, 'Folinic acid is neuroprotective in a fly model of Parkinson's disease associated with pink1 mutations', published in Science Matters, is available here: https://sciencematters.io/articles/201702000009

http://bit.ly/2mGf6uL

Spiders eat twice as much animal prey as humans do in a year

Spiders eat more prey than humans

By New Scientist staff and Press Association

Spiders devour up to 800 million tonnes of prey each year, making them some of the world's most voracious predators.

Most of their victims are insects but the largest tropical species occasionally make a meal of vertebrates such as frogs, lizards, fish and small mammals.

There are more than 45,000 species of spider living in all parts of the world with a collective weight of about 25 million tonnes.

Together they kill between 400 million and 800 million tonnes of prey annually, a team of Swiss and Swedish scientists has calculated.

In comparison, all the humans on Earth consume about 400 million tonnes of meat and fish each year, according to the United Nations Food and Agriculture Organisation.

The appetite of spiders even exceeds that of whales, which get through an estimated 280 million to 500 million tons of prey a year.

"Our calculations let us quantify for the first time on a global scale that spiders are major natural enemies of insects," says Martin Nyffeler, from the University of Basel in Switzerland. "In concert with other insectivorous animals such as ants and birds, they help to reduce the population densities of insects significantly."

"Spiders thus make an essential contribution to maintaining the ecological balance of nature," Nyffeler says.

Ninety per cent of spider prey consists of insects and springtails, small insect-like arthropods. The team showed that spiders killed many times more insects in forests and grasslands than in other habitats.

Their impact was lower in agricultural areas because intensively managed farmland is not favourable to spiders, the researchers said.

"We hope that these estimates and their significant magnitude raise public awareness and increase the level of appreciation for the important global role of spiders in terrestrial food webs," says Nyffeler.

Journal reference: The Science of Nature, DOI: 10.1007/s00114-017-1440-1

http://bit.ly/2mWJIsM

Brain-aging gene discovered

Genetic variant accelerates normal brain aging in older people by up to 12 years

NEW YORK, NY - Columbia University Medical Center (CUMC) researchers have discovered a common genetic variant that greatly impacts normal brain aging, starting at around age 65, and may modify the risk for neurodegenerative diseases. The findings could point toward a novel biomarker for the evaluation of anti-aging interventions and highlight potential new targets for the prevention or treatment of age-associated brain disorders such as Alzheimer's disease.

The study was published online today in the journal Cell Systems.

"If you look at a group of seniors, some will look older than their peers and some will look younger," said the study's co-leader Asa Abeliovich, PhD, professor of pathology and neurology in the Taub Institute for Alzheimer's Disease and the Aging Brain at CUMC. "The same differences in aging can be seen in the frontal cortex, the brain region responsible for higher mental processes. Our findings show that many of these differences are tied to variants of a gene called TMEM106B. People who have two 'bad' copies of this gene have a frontal cortex that, by various biological measures, appears 12 years older that those who have two normal copies."

Studies have identified individual genes that increase one's risk for various neurodegenerative disorders, such as apolipoprotein E (APOE) for Alzheimer's disease. "But those genes explain only a small part of these diseases," said study co-leader Herve Rhinn, PhD, assistant professor of pathology and cell biology in the Taub Institute. "By far, the major risk factor for neurodegenerative disease is aging. Something changes in the brain as you age that makes you more

susceptible to brain disease. That got us thinking, 'What, on a genetic level, is driving healthy brain aging?'"

In the current study, Drs. Abeliovich and Rhinn analyzed genetic data from autopsied human brain samples taken from 1,904 people without neurodegenerative disease. First, the researchers looked at the subjects' transcriptomes (the initial products of gene expression), compiling an average picture of the brain biology of people at a given age. Next, each person's transcriptome was compared to the average transcriptome of people at the same age, looking specifically at about 100 genes whose expression was found to increase or decrease with aging. From this comparison, the researchers derived a measure that they call differential aging: the difference between an individual's apparent (biological) age and his or her true (chronological) age. "This told us whether an individual's frontal cortex looked older or younger than expected," said Dr. Abeliovich.

The researchers then searched the genome of each individual, looking for genetic variants that were associated with an increase in differential age. "One variant stood out: TMEM106B," said Dr. Rhinn. "It's very common. About one-third of people have two copies and another third have one copy."

"TMEM106B begins to exert its effect once people reach age 65," said Dr. Abeliovich. "Until then, everybody's in the same boat, and then there's some yet-to-be-defined stress that kicks in. If you have two good copies of the gene, you respond well to that stress. If you have two bad copies, your brain ages quickly."

The researchers found a second variant--inside the progranulin gene--that contributes to brain aging, though less so than TMEM106B. Progranulin and TMEM106B are located on different chromosomes but are involved in the same signaling pathway. Both have also been associated with a rare neurodegenerative disease called frontotemporal dementia.

The study did not address what role the two genetic variants might have in neurodegenerative disease. "We were studying healthy individuals, so it is not about disease, per se," said Dr. Abeliovich. "But of course, it's in healthy tissue that you start to get disease. It appears that if you have these genetic variants, brain aging accelerates and that increases vulnerability to brain disease. And vice versa: if you have brain disease, the disease accelerates brain aging. It's a vicious cycle."

The study is titled, "Genetic determinants of aging in human brain."

The study was supported by grants from the National Institute of Aging (AG042317), the National Institute of Neurological Disorders and Stroke, and the Michael J. Fox Foundation for Parkinson's Research.

Dr. Abeliovich is a co-founder of and consultant for Alector. Dr. Rhinn is a consultant for Alector. The researchers declare no other financial conflicts of interest.

http://bit.ly/2mI8jAV

Undergoing hip replacement improves five-year quality of life

Patients with total hip replacement experience lasting improvements in quality of life at least five years after the procedure

Patients undergoing total hip replacement experience meaningful and lasting improvements in quality of life (QOL) through at least five years after the procedure, reports a study in the March 15 issue of The Journal of Bone & Joint Surgery. The journal is published by Wolters Kluwer.

As pain and functioning improve after hip replacement surgery, so do scores on the "Short Form-6D" (SF-6D)--a widely used measure of patient-perceived QOL, according to the new research by Dr. Michael A. Mont of the Cleveland Clinic and colleagues. They write, "The SF-6D is one of the few straightforward, easily obtainable methods that provide clinicians quantifiable insight into a patient's quality of life."

Measuring QOL Helps Show Benefits and Value of Total Hip Replacement

The researchers analyzed data on 188 patients, average age 69 years, who underwent total hip replacement (also called arthroplasty) at seven hospitals. Patients were evaluated with a standard QOL

assessment, called the "Short Form 36" (SF-36), from which the SF-6D scores were calculated.

The SF-6D provides scores for six QOL domains: vitality, pain, mental health, social and physical functioning, and role limitations. The SF-6D has been used to assess the health benefits and economic value of many different treatments. The results showed significant increases in the SF-6D score from before to after total hip replacement. Although scores peaked at one year, the improvement remained significant through five years' follow-up.

Furthermore, the gains in SF-6D score remained well above the cutoff point for a large "effect size"--indicating clinically relevant improvement in QOL. The SF-6D scores corresponded to lasting improvements on standard assessments of hip pain and motion as well as the ability to perform everyday activities.

At a time of increased focus on the economic sustainability of the healthcare system, it is important to document the value of healthcare interventions. Total hip replacement is an effective procedure for which demand is expected to increase in the future. The new study appears to be the first to show that the SF-6D--an easy-to-use QOL measure--confirms the positive patient-perceived impact of hip replacement surgery.

Like other QOL assessments, the SF-6D has some disadvantages. However, Dr. Mont and coauthors note, "The SF-6D provides clinicians with a method of quantifying patient satisfaction and perception of their own health." This is an important concept in assessing the value, or "utility," of the procedure.

Another key advantage is the ability to calculate the SF-6D score from the SF-36 assessment--one of the most widely used evaluations of mental and physical health after surgery. Dr. Mont and colleagues conclude: "Therefore, widely incorporating the SF-6D into future postoperative assessments is straightforward, and having these values readily available may make prospective cost-effectiveness analyses considerably easier."

Determining Health-Related Quality-of-Life Outcomes Using the SF-6D Following Total Hip Arthroplasty.
http://journals.lww.com/jbjsjournal/Fulltext/2017/03150/Determining_Health_Related_Quality_of_Life.7.aspx *(doi: 10.2106/JBJS.15.01351)*

http://bit.ly/2mqp0qC

Dissent in Science Is Essential--up to a Point

When discredited "outsider" theories inform government policy, we all pay a price

By Matthew Nolan on March 15, 2017

Disagreement is part and parcel of the human condition. This is no less true for the scientific research community, and in my field, neuroscience, opposing opinions abound on even the most fundamental concepts. In science, the opinion backed by the largest amount of credible evidence is held as the most legitimate. But if, at any point, new information arises that challenges the orthodoxy, it will be accepted provided it presents more credible evidence than the previous consensus. This is the fundamental, defining principle of scientific advancement, and science prides itself on this adherence to empiricism.

However, even this seemingly straightforward concept can create issues. The problem can be summarized as follows: what level of evidence do we require to fulfill a hypothesis, and at what point (if ever) does a hypothesis become unequivocal truth? Though these are not easy questions to answer, the de facto protocol of science is to gradually accept answers as their viability withstands the test of time.

That said, there will always be those who reject evidence accepted by the majority. This leads us to more pressing questions: to what extent should the scientific community engage with ideas that fall outside the mainstream? And, perhaps more pertinently, does a dissenting scientist have the right to be heard and validated irrespective of the potential weight of evidence contradicting their beliefs?

While some dissenting voices are relatively harmless (step forward, people that think the earth is flat), others have devastating consequences. An obvious example of the latter is in HIV/AIDS

denialism, whose proponents refuse to accept the overwhelming evidence that HIV causes AIDS and, to varying degrees also believe that diagnostic tests for HIV are inaccurate, the HIV/AIDS epidemic in Africa is a myth, and that anti-retroviral treatment makes the condition worse. Notably, several prominent denialists have extensive academic credentials, lending their views a veneer of legitimacy.

One prominent denialist was Christine Maggiore, founder of the alternative AIDS organization Alive & Well. Maggiore—who herself was HIV-positive—rejected conventional treatment and advocated a variety of holistic therapies—before dying from pneumonia with disseminated herpes, classic opportunistic infections arising from a compromised immune system associated with advanced AIDS. After she refusing anti-retrovirals throughout her pregnancy, her HIV-positive three-year-old daughter also died of infections resulting from AIDS. Tragically, these are not isolated cases of denialists dying for their beliefs.

When such extreme views become government policy, the potential for harm increases exponentially. Until 2008, Dr. Manto Tshabalala-Msimang, the health minister to the South African President Thabo Mbeki, advocated beetroot, lemon juice and garlic as a treatment for HIV. Experts subsequently estimated that more than 300,000 people died as a result of the AIDS denialist policies of the Mbeki government.

The growing "anti-vaxxer" movement is similarly corrosive, where rejecting scientific and medical evidence on the efficacy and safety of vaccinations contributes to demonstrable outbreaks of otherwise wholly preventable diseases. Of course, making such decisions for your own health are one thing, but I would argue that new realms of injustice are broken when you force your own ignorance on to an innocent child.

The damaging effects of a vocal minority are not confined to medicine. Donald Trump has front-lined scientific dissent through cabinet appointments (some of whom also have established scientific backgrounds) and their remarks on the nature of climate change. If these position-holders influence US climate policy as feared, the effects will undoubtedly be both devastating and irreversible. Ignoring the majority expert opinion in this way has been labeled as being part of a "post-truth" society, where the voices of world-leading experts are swept aside amongst the cacophony of populist opinion. Regardless, what unites the above examples is that ultimately, it is the public who pay the price when marginalized science informs policy. History reminds us this is unsafe territory.

One issue is that when the mainstream view turns out to be wrong, it jades the public perception of science whilst also seemingly validating the pursuits of the contrarians. Nutrition is a prime example of this phenomenon. In 1972 scientist John Yudkin was the author of a book that described how sugar, not fat, was the primary culprit for rising levels of obesity. He was marginalized by the mainstream nutrition community during his lifetime. But Yudkin was right, and now, 40 years later, the central role that sugar plays in expanding our waistlines is now widely acknowledged by nutritionists. Despite this knowledge, and in response to demand, food companies continue to capitalize on consumer fears with "low-fat" alternatives.

The right to dissent in enshrined in the scientific constitution. Arguably, it is the duty of scientists to dispute the status quo. Hypotheses are designed to be challenged, and history is littered with examples of long-held beliefs being re-written on the basis of fresh evidence. This continual revision of the orthodoxy is, in general, a good thing for science and more fundamentally, the development of mankind. The inherent fallibility of science—which on the surface can seem its biggest weakness—is actually its biggest strength.

However, at the extreme end of the spectrum are those dissenters who style themselves as research "white knights," pariahs of the establishment working to uncover unpopular truths, and crying oppression when their views are debunked. This is more commonly

known as playing the victim card. The spirit of Galileo, the most famous contrarian in history, is regularly invoked.

But in reality, such dissenters rely on their right to dissent, rather than the validity of their opinions, to be heard. Both the public and scientists must be wary of lending credence to unsubstantiated (and potentially damaging) beliefs on the principle of someone's right to disagree.

http://bit.ly/2mqDHu9

O my! Paper strip test determines blood type in just 30 seconds

A quick test could allow donor blood to be matched to patients in seconds

By Alice Klein

A, B, AB or O: Do you know which blood type you are? A quick and easy test might soon be able to tell you and allow hospitals to rapidly administer the right blood in an emergency.

Your blood type is essential information if you ever need a blood transfusion, because mismatched blood can send the immune system into meltdown, and sometimes lead to death. But it can take 10 or 20 minutes to verify someone's blood type by conventional methods – not including the time it takes to transport the blood to a lab.

For this reason, most emergency departments stock only type O blood, which can safely be given to anyone because it lacks the antigens that trigger immune reactions. However, this places pressure on supplies of O blood. "The demand is already extremely high and it's getting higher," says Janet Wong at the Australian Red Cross Blood Service, which collects blood donations in Australia.

Now Hong Zhang at the Third Military Medical University in China and his colleagues have developed a paper-based test that could be quickly performed at a patient's bedside without specialised training or equipment. It would allow hospitals to give patients matching blood – whether it be A, B, AB or O, and make it more viable to keep supplies of more blood types.

Looks good on paper

The test relies on the antigens that are present on the surface of red blood cells. Type A blood contains red blood cells with A antigens, B with B antigens, AB with both, and O with neither. The presence of a separate D antigen determines whether the blood type is "positive" or "negative", also known as rhesus positive or negative.

Blood also contains antibodies, which find and destroy invaders. If they detect antigens from foreign red blood cells, they can mount a catastrophic immune response.

Zhang's paper strip is impregnated with a matrix of antibodies and dye, and when a drop of blood is applied, squares of colour develop as the blood spreads across the strip and reacts with the antibodies. For each antigen – A, B and D – a teal square shows up if it is a present, or a brown square if it is not. When trialled on 3550 human blood samples, the low-cost strip was more than 99.9 per cent accurate, and only took 30 seconds to complete.

Zhang says the paper strip could be used in war zones or remote areas where there are no labs to test patients' blood types. O blood is currently used in these contexts, but supplies are limited.

Wong says the test could alleviate reliance on O blood in emergency settings, but it might still be wise to keep big stores of O blood, "The benefit of only stocking O blood is that you can't accidentally give the wrong type," she says. A paper test might determine the correct blood to give, but human error could still lead to a mix-up – particularly in high pressure scenarios – she adds.

Zhang says the paper test could be developed into a cost-effective and robust universal blood-grouping platform. "We are expecting that we can see this product in the market within 1-2 years," he says.

Journal reference: Science Translational Medicine, DOI: 10.1126/scitranslmed.aaf9209

http://bit.ly/2nRN5kd

Complex Life Could Be Vastly Older Than Thought

New algae fossil discovery may reset the evolutionary time line

By Bret Stetka on March 15, 2017

It was around 1.6 billion years ago that a community of small, bright red, plantlike life-forms, flitting around in a shallow pool of prehistoric water, were etched into stone until the end of time. Or at least until a team of Swedish researchers chipped their fossilized remnants out of a sedimentary rock formation in central India.

This photo shows a light microscope image of cellular tissue from a "fleshy" specimen resembling red algae, named Ramathallus lobatus. **Therese Sallstedt, Swedish Museum of Natural History**

Research published this week in PLoS Biology suggests this collection of ancient, newly analyzed fossils—unearthed a few years back—are in all likelihood red algae. If that proves true, it would imply that complex, multicellular life evolved a lot earlier than previously thought—and that the evolutionary family tree of life on Earth might need a major pruning.

Earth's first traces of life probably showed up around 3.5 billion years ago, a billion years or so after our planet formed. Just when these simple, single-celled organisms—classified as "prokaryotes" due to their lack of a nucleus—evolved into multicellular, nucleated forms called "eukaryotes" is a matter of debate. Alga, a eukaryote, is thought to be one of the oldest forms of complex life. And given that previous fossil finds had dated red algae back just 1.2 billion years, the new discovery could reset the evolutionary time line by nearly half a billion years.

The apparent red alga was found embedded in fossilized sheets of cyanobacteria, widely believed to be the first oxygen-producing life-forms to have arisen and a precursor all to algae and plants. (Although not all algae are considered "plants" per current classification, they are all considered plantlike because they use photosynthesis to produce energy). By dissolving surrounding rock with acetic acid—a common method used in excavating fossils—the new paper's authors unearthed what appear to be two forms of red alga: a tubular strain resembling a segmented pool noodle and a fleshier variety composed of multilayered collections of cells.

The authors used a technique called synchrotron-based x-ray tomographic microscopy to construct a three-dimensional model of the fossils, and to identify internal cellular structures that the organisms probably used for energy production. Radioactive dating was used to confirm the fossils' age. "The new fossils provide tangible evidence that advanced multicellularity, at least in plants, appeared much earlier than previously thought," says Stefan Bengtson, senior author of the new paper and professor emeritus of paleozoology at the Swedish Museum of Natural History. "They suggest that the timing of early eukaryotes may have to be drastically revised."

Without the presence of DNA—which does not hang around in samples so staggeringly old—it is impossible to confirm the new fossils are bygone red algae. Bengtson admits as much. But he also believes the fossils' structures bear a strong resemblance to that of red alga.

Paul Strother, a Boston College biologist who studies the evolution of algae and plants, and who was not involved in the new research, is not sold. "If these are real…they still do not show any sort of cell differentiation. All the cells are basically the same, and these forms do not represent complex multicellularity," he says.

University of Wisconsin–Eau Claire biology chair, Wilson Taylor, who was also uninvolved in the work, points out that even if the new samples are really algae, the search for the origins of complex life still has a long way to go. "If a red alga really had evolved by this time…this implies a prior period of eukaryotic evolution of some length," he says. "How long before the 1.6-billion-year horizon eukaryotes arose, based on that early occurrence, is anyone's guess." Taylor explains that eukaryotes—which comprise virtually all nonmicroscopic life on Earth—likely arose when one prokaryote

engulfed another and found some symbiotic benefit that kept the relationship going. But how long it took this vital communion to take hold in the evolutionary process is unknown.

As Bengtson points out, whereas red algae are not a direct precursor to plants—that honor belongs to green algal ancestors—they do closely descend from one common ancestor of all plants on Earth today. Assuming the new findings are true, a major question now facing paleobotanists is why it took another billion years for larger, more complex organisms to flourish.

It was not until between 600 million and 500 million years ago that higher plants and animals began evolving. Submarine algae, bobbing amid blankets of microbes, gradually gave way to what we know as plants. Plants made their way to shore, shaping a new landscape that would come to include complex fungi and, eventually, terrestrial animals.

Bengtson hopes to further study early algal populations in order to better pinpoint where and when they arose, and why they lingered in the sea for so long. Although evolution's tributaries will no doubt continue to be modified—some rerouted, others reset—scientists can assume they are closing in on an accurate lineage of life on Earth; and that our photosynthetic, oxygen-producing co-dwellers, on which our lives rely, have speckled the planet we call home for much of its existence.

http://bit.ly/2nRD2f1

Malaysia raises alert as bird flu virus hits more birds

Malaysian authorities raised an alert for a northeastern state after the virulent H5N1 bird flu virus was found to have spread to poultry in more villages.

Some 24,000 birds have been culled in Kelantan state since the H5N1 strain was reported March 6 after several backyard poultry died.

Che Abdullah Mat Nawi, who heads Kelantan's agricultural committee, said Thursday that free-range chickens reared by residents in 20 villages have been affected. He said the state is getting more manpower and resources to survey a wider area to contain the spread of the virus.

"The situation is getting more serious but it is not yet an emergency situation as there is no transmission of the virus from bird to human. We have raised the alert so that we can pool resources from different departments" to curb the spread of the virus, he told The Associated Press. Che Abdullah said commercial bird farms in the state were not affected by the virus.

The H5N1 strain is highly contagious and linked by the World Health Organization to hundreds of deaths worldwide over the last decade.

Health Director-General Noor Hisham Abdullah said health officials have screened more than 3,500 villagers in the affected areas. More than a dozen people showing bird flu symptoms tested negative for the virus, he said.

Veterinary officials told local media that the virus could have been due to cockfighting activities involving roosters from neighboring countries. Che Abdullah said officials believed the virus was imported but have not determined the cause of the outbreak.

http://bit.ly/2mX5BIx

Cause of obsessive-compulsive disorder discovered

absence of a single protein can trigger an excessive grooming behaviour

Some people have an extreme fear of dirt or bacteria. As a result, they may develop a habit of compulsive washing and repeatedly cleaning their hands or body. They are trapped in a vicious circle, as the fear of new contamination returns quickly after washing. Sufferers see no way out. They are even incapable of changing their behaviour when the excessive washing has led to skin irritation or damage.

Around two percent of the general population suffer from some kind of obsessive-compulsive disorder (OCD) at least once in their life. The disorder is characterised by persistent intrusive thoughts which the sufferers try to compensate for by repetitive ritualized behaviour.

Like depression, eating disorders and other mental diseases, OCD is treated with antidepressants. However, the drugs are non-specific, that is they are not tailored to the respective disease. Therefore, scientists have been looking for new and better targeted therapies that have fewer side effects.

Missing protein triggers urge to wash

Professor Kai Schuh from the Institute of Physiology at the Julius-Maximilians-Universität (JMU) Würzburg (Germany) and his team explore the underlying causes of obsessive-compulsive disorder in collaboration with the JMU's Departments of Psychiatry and Neurology.

"We were able to show in mouse models that the absence of the protein SPRED2 alone can trigger an excessive grooming behaviour," Schuh says. He believes that this finding is crucial as no clear trigger for this type of disorder has been identified until now. Previous research pointed to multiple factors being responsible for developing OCD.

Occurring in all cells of the body, the protein SPRED2 is found in particularly high concentrations in regions of the brain, namely in the basal ganglia and the amygdala. Normally, the protein inhibits an important signal pathway of the cell, the so-called Ras/ERK-MAP kinase cascade. When it is missing, this signal pathway is more active than usual.

Hyperactive signal cascade in the brain

"It is primarily the brain-specific initiator of the signal pathway, the receptor tyrosine kinase TrkB, that is excessively active and causes the overshooting reaction of the downstream components", biologist Dr. Melanie Ullrich explains.

Administering an inhibitor to attenuate the overactive signal cascade in the animal model improves the obsessive-compulsive symptoms. Moreover, the JMU research team was able to treat the OCD with an antidepressant, similarly to standard therapy in humans. Their detailed results have been published in the journal Molecular Psychiatry.

New targets for therapies pinpointed

"Our study delivers a valuable new model that allows the disease mechanisms to be investigated and new therapy options for obsessive-compulsive disorders to be tested," Professor Schuh says.

The recently discovered link between OCDs and the Ras/ERK-MAP kinase cascade also opens up new targets for therapy. Drugs that inhibit this cascade are already available and some of them are approved for human treatment.

According to Melanie Ullrich, these are cancer drugs, as overactivation of the Ras/ERK-MAP kinase cascade is also a frequent trigger of cancer: "So we are wondering whether such drugs could also be effective in the treatment of obsessive-compulsive disorders and whether they are beneficial in terms of side effects."

http://to.pbs.org/2mqRK2I

Remnants of Earth's Original Crust Found in Canada

Two geologists studying North America's oldest rocks have uncovered ancient minerals that are remnants of the Earth's original crust which first formed more than 4.2 billion years ago.

Posted by Annette Choi on Thu, 16 Mar 2017

These rocks appear to preserve the signature of an early Earth that presumably took shape within the first few hundred million years of Earth's history.

Jonathan O'Neil and Richard Carlson uncovered the samples on a trek to the northeastern part of Canada to study the Canadian Shield formation, a large area of exposed continental crust underlying, centered on Hudson Bay, which was already known to contain some of the oldest parts of North America. O'Neil calls it the core or nucleus of the North American continent. "That spot on the shore of Hudson Bay has this older flavor to it, this older chemical signature."

To O'Neil, an assistant professor of geology at the University of Ottawa, rocks are like books that allow geologists to study their compositions and to learn about the conditions in which they form.

But as far as rock records go, the first billion years of the Earth's history is almost completely unrepresented.

"We're missing basically all the crust that was present about 4.4 billion years ago. The question we're after with our study is: what happened to it?" said Carlson, director of the Carnegie Institution of Washington. "Part of the goal of this was simply to see how much crust was present before and see what that material was."

A view of 2.7 billion-year-old continental crust produced by the recycling of more than 4.2 billion-year-old rocks.

While most of the samples are made up of a 2.7 billion-year-old granite, O'Neil said these rocks were likely formed by the recycling of a much older crust. "The Earth is very, very good at recycling itself. It constantly recycles and remelts and reworks its own crust," O'Neil said. He and Carlson arrived at their conclusion by determining the age of the samples using isotopic dating and then adding on the estimate of how long it would have taken for the recycled bits to have originally formed.

O'Neil and Carlson's estimate relies on the theory that granite forms through the reprocessing of older rocks. "That is a possibility that they form that way, but that is not the only way you can form these rocks," said Oliver Jagoutz, an associate professor of geology at the Massachusetts Institute of Technology. "Their interpretation really strongly depends on their assumption that that is the way these granites form.

The nature of Earth's first crust has largely remained a mystery because there simply aren't very many rocks that have survived the processes that can erase their signature from the geologic record. Crust is often forced back into the Earth's interior, which then melts it down, the geologic equivalent of sending silver jewelry back into the forge. That makes it challenging for geologists to reconstruct how the original looked.

These new findings give geologists an insight into the evolution of the oldest elements of Earth's outer layer and how it has come to form North America. "We're recycling extremely, extremely old crust to form our stable continent," O'Neil said.

http://bit.ly/2mIQWjr

Honest mistakes by young scientists shouldn't doom their careers

Pressure for postdocs to publish is intense, as young researchers vie for the few top academic lab jobs

By Ivan Oransky @ivanoransky and Adam Marcus @armarcus

The years spent as a grad student and postdoc are among the most trying times for any scientist. The pressure to publish is intense, as young researchers vie for the few jobs at the heads of academic labs.

Those high stakes and the pressure-cooker atmosphere make mistakes — and sometimes the willingness to cut corners and commit fraud — more likely.

Unfortunately, both can be career killers, if two recent cases are taken as examples. And although fraudsters aren't welcome, the loss of the innocent overwhelmed is taking a toll on science.

Case one: Sergio Gonzalez, a postdoctoral researcher at the Institute for Neurosciences of Montpellier, France. He was hitting the job market in 2015, and he knew he needed a paper in a top journal to stand out in that market.

So he was relieved — elated, perhaps — when editors at the Journal of Clinical Investigation, one of the world's most prestigious journals, told him they'd be publishing one of his papers. Having an article accepted there would carry a lot of weight on a job application — and in France's system, success on that application meant a permanent job.

But the paper was flawed — deeply, it turns out. First, a commenter on PubPeer, an anonymous post-publication peer review site, flagged a suspicious-looking figure. Next came a correction in the journal,

more comments on PubPeer, an expression of concern from the editors, an institutional review of Gonzalez's work, and finally, this month, a retraction. Along the way, Gonzalez lost the opportunity for the job he so wanted.

Around that same time, across the Atlantic, another grad student was also on the academic job market. Michael LaCour was a promising graduate student in political science at the University of California, Los Angeles, who managed to publish a headline-grabbing paper in Science, one of the world's top journals, about attitudes toward gay marriage. Soon after that, he landed a job at Princeton. But then his paper — and academic career — unraveled after two other graduate students at different institutions starting asking questions that would eventually make it clear LaCour had made up the data. The paper was retracted — and so was his job offer.

LaCour's story and Gonzalez's take different paths from there. While LaCour, it seems, faked his data, through it all, Gonzalez and his supervisor, Nicolas Tricaud, maintained that the postdoc was innocent of misconduct. Any errors, Tricaud insisted, were the result of Gonzalez's haste and stress over his impending job search and his desire to land a plum spot.

The university seemed to agree that Gonzalez was honest but sloppy. According to the JCI's retraction notice: "The institutional review found no evidence of intention to falsify results and concluded that errors were made due to negligence during the assembly of figures. The institutional review panel did not question in any way the authenticity of the published results."

We may feel relieved that LaCour seems unlikely to return to the ivory tower. But the loss of Gonzalez, who did not win a coveted spot in a laboratory, seems by all accounts to have been a blow to science. Tricaud says the budding researcher has dropped out of academia — a shame considering his willingness to "work like hell" on the project.

It's not just France where postdocs feel this pressure. In the US, only about 15 percent of postdocs can expect to land faculty jobs, according to one estimate. Meanwhile, the rate of unemployment among this group jumped from 4 percent in 2008 to 10 percent in 2012.

Part of the problem is that academic mentors tend to emphasize careers in academia, rather than all of the other doors a PhD can open. So when someone like Gonzalez is shut out of the academy, they feel the failure even more acutely. One solution would be for such mentors to embrace so-called "alternative careers," whether in industry, public service, or elsewhere — which aren't really "alternative" anymore, given that most PhDs end up in them.

But if senior faculty and administrators don't want to drive young scientists from the field — even the honest ones — they'd best figure out a way to let publish or perish itself perish.

http://bit.ly/2nA3xt8

Osteoporosis drug found safe in long-term trial

A new study provides reassuring information about the short-term and long-term safety of denosumab, a monoclonal antibody that is used to treat postmenopausal osteoporosis.

Adverse events that had been noted in a pivotal clinical trial in women age 60 to 90 years old treated for 3 years showed no tendency to increase after a further 3 years of treatment, the study showed.

In addition, women who crossed over from 3 years of placebo to 3 years of denosumab experienced no increase in adverse effects compared with women treated for the initial 3 years.

"All of this is consistent with an excellent safety and tolerability profile for denosumab treatment for osteoporosis," said Dr. Nelson Watts, lead author of the study results published in Journal of Bone and Mineral Research. The authors noted that, especially in older women on long-term treatment, many if not all adverse events could be called "life events" -- things that would have happened whether or not the person was participating in a clinical trial.

http://bit.ly/2n3J2DK

New 'gene silencer' drug reduce cholesterol by over 50 percent

The findings come from the largest trial yet to test the safety and effectiveness of this kind of therapy.

The technique, known as RNA interference (RNAi) therapy, essentially 'switches off' one of the genes responsible for elevated cholesterol.

Researchers from Imperial College London and their colleagues, who conducted the trial, say the twice-a-year treatment could be safely given with or without statins, depending on individual patient needs. Eventually, inclisiran could help to reduce the risk of heart attacks and stroke related to high cholesterol.

"These initial results are hugely exciting for patients and clinicians," said Professor Kausik Ray, lead author of the study from the School of Public Health at Imperial.

"We appear to have found a versatile, easy-to-take, safe, treatment that provides sustained lowering of cholesterol levels and is therefore likely to reduce the risk of cardiovascular disease, heart attacks, and stroke. These reductions are over and above what can be already be achieved with statins alone or statins plus ezetemibe, another class of cholesterol-lowering drug.

Elevated levels of low-density lipoprotein (LDL) cholesterol can lead to cardiovascular disease and blood vessel blockage, leading to an increased risk heart attacks and stroke in patients.

Statins are currently the standard treatment for high cholesterol, combined with exercise and healthy diet, as they reduce levels in the blood and therefore help to prevent heart attacks and stroke.

However, many patients are unable to tolerate the highest doses and they need to be taken consistently. Forgetting to take them or taking them infrequently reduces the expected benefit from these treatments. Also, in some patients cholesterol levels can remain high despite being given the maximum doses of statins.

Now, this new phase 2 clinical trial has confirmed the effectiveness of injecting inclisiran for reducing cholesterol that can be taken alone or potentially combined with statins for maximum effect.

In the study, researchers gave 497 patients with high cholesterol and at high risk of cardiovascular disease either inclisiran at varying doses, or placebo. Seventy-three per cent of these patients were already taking statins, and 31 per cent were taking ezetimibe. Participants, who were recruited from Canada, USA, Germany, Netherlands, and the UK, were excluded if they were taking monoclonal antibodies for cholesterol lowering.

Patients were given different doses of inclisiran or placebo via subcutaneous injection, either via a single dose, or via a dose on day one and another at three months. They were followed up regularly for a subsequent eight months and tested for blood cholesterol and side effects.

The researchers found that just one month after receiving a single treatment of inclisiran, participants' LDL cholesterol levels had reduced by up to 51 per cent.

In those on a single dose of 300 mg, cholesterol levels were reduced by 42 per cent at six months. In the matched placebo group, cholesterol levels had increased by two per cent within that time frame.

In those on two doses of 300 mg, cholesterol levels were reduced by up to 53 per cent at six months. Moreover, cholesterol levels had gone down for all patients in this group, and 48 per cent of them had achieved cholesterol levels (below 50 mL/dL).

In all patients, cholesterol levels stayed lower for at least eight months. No extra side effects were seen in the study group compared to the placebo group.

The study will now follow up patients for a further four months (one year total follow up). The results from this trial, known as ORION-1, are published in the New England Journal of Medicine, and are presented today at the American College of Cardiology's 66th Annual Scientific Session in Washington.

The authors say the results show the drug acts quickly to reduce cholesterol levels by as early as two weeks post-injection, while also giving a prolonged effect when given in two doses over a year. Therefore, the next step is to conduct an extended study, using more patients and for a longer period of time, to determine whether these reductions in cholesterol translate into a reduction in heart attacks and strokes. Professor Ray said: "We are keen to enter the next phase of development to assess long-term safety and to see how this novel approach might translate into improvements in patient health."

Aside from its effectiveness, the authors point out that because inclisiran acts on a different biological pathway to statins, the two drugs would likely be combined for the best results. Professor Ray said: "Even the single dose of inclisiran appears to lower cholesterol by 35-40% at eight months. We could essentially experiment with how often to give the drug based on levels of cardiovascular risk for each patient. Lower risk patients could in theory have once yearly injections whereas higher risk patients might have two injections a year."

The authors emphasise that because this is an early-phase study, and because this is one of the first clinical studies on this type of drug, more research is needed before it can go to market.

He added: "The effectiveness of statins and other cholesterol-lowering treatments such as monoclonal antibodies relies on patients' ability to take them consistently. Therefore, giving inclisiran up to twice yearly at a GP surgery, much in the same way flu vaccinations are provided, might be more effective."

"We believe that these clinical visits might only be twice a year at most, so ultimately, they are more convenient and more effective for patients and their health."

Inclisiran is being developed by Alnylam Pharmaceuticals and The Medicines Company. This study was funded by The Medicines Company, and performed by the sponsors and World Wide Clinical Trials (Nottingham, UK).

http://bit.ly/2mFfLMe

The Lancet: Indigenous South American group has healthiest arteries of all populations yet studied, providing clues to healthy lifestyle

Study estimates that an 80-year-old from the Tsimane group had the same vascular age as an American in their mid-50s

The Tsimane people - a forager-horticulturalist population of the Bolivian Amazon - have the lowest reported levels of vascular ageing for any population, with coronary atherosclerosis (hardening of the arteries) being five times less common than in the US, according to a study published in The Lancet and being presented at the American College of Cardiology conference.

The researchers propose that the loss of subsistence diets and lifestyles in contemporary society could be classed as a new risk factor for heart disease. The main risk factors are age, smoking, high cholesterol, high blood pressure, physical inactivity, obesity and diabetes.

"Our study shows that the Tsimane indigenous South Americans have the lowest prevalence of coronary atherosclerosis of any population yet studied," said senior anthropology author, Professor Hillard Kaplan, University of New Mexico, USA. "Their lifestyle suggests that a diet low in saturated fats and high in non-processed fibre-rich carbohydrates, along with wild game and fish, not smoking and being active throughout the day could help prevent hardening in the arteries of the heart. The loss of subsistence diets and lifestyles could be classed as a new risk factor for vascular ageing and we believe that components of this way of life could benefit contemporary sedentary populations." [1]

Although the Tsimane lifestyle is very different from that of contemporary society, certain elements of it are transferable and could help to reduce risk of heart disease.

While industrial populations are sedentary for more than half of their waking hours (54%), the Tsimane spend only 10% of their daytime being inactive. They live a subsistence lifestyle that involves hunting,

gathering, fishing and farming, where men spend an average of 6-7 hours of their day being physically active and women spend 4-6 hours. Their diet is largely carbohydrate-based (72%) and includes non-processed carbohydrates which are high in fibre such as rice, plantain, manioc, corn, nuts and fruits. Protein constitutes 14% of their diet and comes from animal meat. The diet is very low in fat with fat compromising only 14% of the diet - equivalent to an estimated 38 grams of fat each day, including 11g saturated fat and no trans fats. In addition, smoking was rare in the population.

In the observational study, the researchers visited 85 Tsimane villages between 2014 and 2015. They measured the participants' risk of heart disease by taking CT scans of the hearts of 705 adults (aged 40-94 years old) to measure the extent of hardening of the coronary arteries, as well as measuring weight, age, heart rate, blood pressure, cholesterol, blood glucose and inflammation.

Based on their CT scan, almost nine in 10 of the Tsimane people (596 of 705 people, 85%) had no risk of heart disease, 89 (13%) had low risk and only 20 people (3%) had moderate or high risk. These findings also continued into old age, where almost two-thirds (65%, 31 of 48) of those aged over 75 years old had almost no risk and 8% (4 of 48) had moderate or high risk. These results are the lowest reported levels of vascular ageing of any population recorded to date.

By comparison, a US study of 6814 people (aged 45 to 84) found that only 14% of Americans had a CT scan that suggested no risk of heart disease and half (50%) had a moderate or high risk - a five-fold higher prevalence than in the Tsimane population.

In the Tsimane population, heart rate, blood pressure, cholesterol, and blood glucose were also low, potentially as a result of their lifestyle. The researchers also note that the low risk of coronary atherosclerosis was identified despite there being elevated levels of inflammation in half of the Tsimane population (51%, 360 of 705 people).

"Conventional thinking is that inflammation increases the risk of heart disease," said Professor Randall Thompson, cardiologist at Saint Luke's Mid America Heart Institute, USA. "However, the inflammation common to the Tsimane was not associated with increased risk of heart disease, and may instead be the result of high rates of infections." [1]

Because the study is observational it cannot confirm how the Tsimane population is protected from vascular ageing, or which part of their lifestyle (diet, physical activity or smoking) is most protective. The researchers suggest it is more likely to be a result of their lifestyle than genetics, because of a gradual increase in cholesterol levels coinciding with a rapidly changing lifestyle.

"Over the last five years, new roads and the introduction of motorised canoes have dramatically increased access to the nearby market town to buy sugar and cooking oil," said Dr Ben Trumble, Arizona State University, USA. "This is ushering in major economic and nutritional changes for the Tsimane people." [1]

The researchers did not study whether coronary artery hardening in the Tsimane population impacted on their health, but note that deaths from heart attacks are very uncommon in the population so it is likely that their low levels of atherosclerosis and heart disease are associated. The researchers are investigating this in further research.

"This study suggests that coronary atherosclerosis could be avoided if people adopted some elements of the Tsimane lifestyle, such as keeping their LDL cholesterol, blood pressure and blood sugar very low, not smoking and being physically active," said senior cardiology author Dr Gregory S. Thomas, Long Beach Memorial Medical Centre, USA. "Most of the Tsimane are able to live their entire life without developing any coronary atherosclerosis. This has never been seen in any prior research. While difficult to achieve in the industrialized world, we can adopt some aspects of their lifestyle to potentially forestall a condition we thought would eventually effect almost all of us." [1]

NOTES TO EDITORS

The study was funded by the US National Institute of Aging and the National Institutes of Health. It was conducted by scientists from University of New Mexico, Saint Luke's Mid

America Heart Institute, University of Missouri, Arizona State University, Ascension Healthcare, Al Azhar University, Max Planck Institute for Evolutionary Anthropology, National Museum of Natural History, Smithsonian Institution, Dartmouth College, Newport Diagnostic Centre, South Coast Radiological Medical Group, Institute for Advanced Study Toulouse, University of Nevada, University of California, Miller Women's and Children's Hospital Long Beach, Universidad Peruana Cayetano Heredia, University of Washington, University of California Santa Barbara, Weill Cornell Medical College, New York-Presbyterian Hospital, Icahn School of Medicine at Mount Sinai, USC Leonard Davis School of Gerontology, University of Southern California, University of California Irvine and Long Beach Memorial.

http://bit.ly/2mXs0FG

What does that sentence say?

New research shows late bilinguals are sensitive to unique aspects of second language

RIVERSIDE, Calif. - Imagine coming across a sentence in English that reads like this: "Mary apple eats her delicious." For most native-English speakers, the sentence would likely strike you as odd because it doesn't seem to be structured in an order that immediately gets the message across.

It has always been thought that, when adults learn a second language, they face this problem because the grammar of other languages doesn't necessarily match their native language. But, a new study reveals that adults are capable of learning and processing a new language in a way that resembles native speaker language use.

"Learning a second language as an adult is a difficult task," said UCR affiliate psychology professor Elenora Rossi, who was on the research team. "For years, scientists have believed that only the brains of very young children were pliable enough to allow for successful learning of a second language, while that was thought to be impossible for adults."

In the past two decades, the advance of testing methodologies and revolutionary neuroimaging methods have allowed language processing to be studied in real-time in a non-invasive way, opening the doors to a better understanding of how our brains process linguistic information in two languages.

In the study, the team looked at how native English speakers, who learned Spanish as a second language as adults, understood sentences in Spanish that contained subtle aspects of Spanish grammar that do not exist in English. Participants in the study were already advanced in Spanish, but not native speakers. The goal was to test them on aspects of Spanish that are typically difficult to learn because they don't exist in the structure of English grammar. Errors were purposely introduced and participants were asked whether they could detect the errors.

"Counter to the long-standing assumption that learning a second language and becoming bilingual past early childhood is impossible, we found that English speakers who learned Spanish as adults were able to understand these special aspects of Spanish," said Judith Kroll, a UCR psychology professor who was also on the research team. "The results suggest that adults are capable of learning and processing a new language in a way that resembles native speaker language use."

The research team also included Pennsylvania State University faculty members Michele Diaz, psychology professor, and Paola Dussia, professor of Spanish, Italian and Portuguese. The authors of the paper, published in Frontiers in Psychology, are part of a larger research effort between UCR and Penn State to study the bilingual mind and brain. The research is conducted in collaboration, and supported by a National Science Foundation Partnerships for International Research and Education grant. Future research by the team will target understanding how an intensive but short period of new language learning may shape adult minds.

http://bit.ly/2mXEGwl

These 3 Superbugs Pose the Greatest Threat to Human Health

WHO releases its first-ever list of "priority pathogens"

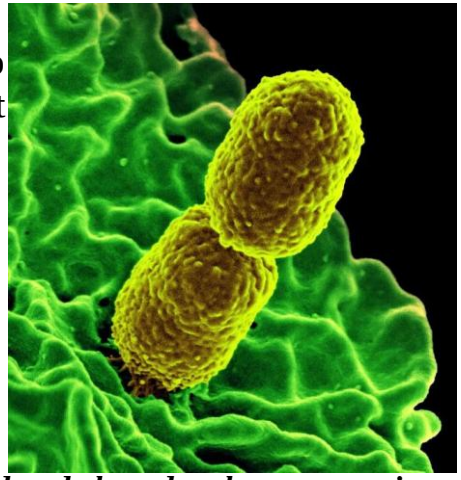
By Stephanie Bucklin, Live Science Contributor

The World Health Organization is issuing a warning about a group of deadly bacteria: Recently, the WHO released its first-ever listof "priority pathogens," a list of antibiotic-resistant bacteria that the organization says pose the greatest threat to human health.

The list is divided into three categories: critical-, high- and medium-priority. Three pathogens made it into the critical-priority group.

These bacteria are resistant to multiple antibiotics and pose a high risk to people in hospitals and nursing homes, the WHO says.

Multidrug-resistant bacteria, sometimes called "superbugs," are a critical priority because infections with these germs can be deadly, according to the WHO. For example, people who get infections from a type of multidrug-resistant bacterium called methicillin-resistant *Staphylococcus aureus* (MRSA) have a 64 percent greater chance of dying than people who contract the same infection in its nonresistant form, according to the WHO.



***This image depicts two mustard-colored, rod-shaped carbapenem-resistant Klebsiella pneumoniae (CRKP) bacteria interacting with a green-colored, human white blood cells.*National Institute of Allergy and Infectious Diseases (NIAID)**

All of the top three pathogens on the list are resistant to a group of antibiotics called carbapenems. These antibiotics are sometimes referred to as "last resort" medications, because if they don't work, very few options are left.

"It is important the WHO take this on, because with travel and now widespread communication, an antibiotic-resistant organism … is going to get around the world pretty quickly," said Dr. Kenrad Nelson, a professor of infectious-disease epidemiology at the Johns Hopkins Bloomberg School of Public Health. Nelson was not involved in compiling the WHO's list.

Overall, the WHO's list is good, Nelson told Live Science. He noted, however, that he would have included the pathogen *Clostridium difficile* on the list. *C. diff* can occur in patients who receive antibiotics and is difficult to treat and get rid of completely, he said.

Here are the top three germs the WHO is worried about:

Carbapenem-resistant *Acinetobacter baumannii*

This bacterium can cause pneumonia, serious blood infections and other conditions, according to the Centers for Disease Control and Prevention.

*A. baumannii*occurs primarily in hospitalized patients. It spreads through either person-to-person contact, or contact with a contaminated surface, the CDC says. Although the pathogen doesn't pose a big threat to healthy people, it's very dangerous for patients with compromised immune systems or chronic diseases, the CDC says. Outbreaks of *A. baumannii* typically take place in hospital settings such as intensive care units (ICUs) or long-term health care facilities with sick patients, such as nursing homes, according to the CDC.

It's unclear how common this pathogen is in many countries around the world; however, *A. baumannii* is estimated to cause between 2 and 10 percent of multidrug-resistant bacterial infections in ICUs in Europe and the U.S., according to the WHO.

Carbapenem-resistant *Pseudomonas aeruginosa*

P. aeruginosa infections most often occur in the hospital. For patients with *P. aeruginosa* infections,pneumonia or infections following surgery can become extremely dangerous, and even life-threatening**.** But these bacteria can also live in hot tubs and swimming pools, and have been linked to serious ear infections and skin rashes, according to the CDC.

*P. aeruginosa*infections occur most often in hospitals; patients can become infected with the bacteria from contact with a breathing machine or a catheter, or through a surgical wound, according to the CDC. The infection is most dangerous to those with weakened immune systems.

The CDC estimates that about 51,000 *P. aeruginosa* infections occur in health care settings in the U.S. each year; of these infections, more than 6,000 are from multidrug-resistant forms of the bacteria. About 400 deaths in the U.S. per year are linked to this infection, the CDC says.

Carbapenem-resistant *Enterobacteriaceae*

Infections with carbapenem-resistant *Enterobacteriaceae* (CRE)most often occur in hospitals or long-term health care settings, the CDC says. Similar to *A. baumannii*, CRE usually does not pose a risk to healthy people; rather, it is most dangerous to people with compromised immune systems, according to the CDC.

CRE can spread through person-to-person contact or through medical devices such as ventilators, the CDC says. In a 2015 study published in the journal JAMA, researchers found that CRE affected approximately 3 in 100,000 people in the U.S. Of the 599 cases studied, 51 patients died.

Other concerning germs

In the other two categories on the priority-pathogens list, the WHO included germs that are resistant to certain antibiotics and those that cause diseases including gonorrhea and *Salmonella food poisoning*.

Six pathogens were included in the high-priority category, and three pathogens were listed in the medium-priority category. The six high-priority pathogens are: *Enterococcus faecium*, vancomycin-resistant; *Staphylococcus aureus*, methicillin-resistant, vancomycin-intermediate and resistant; *Helicobacter pylori*, clarithromycin-resistant; *Campylobacter* spp., fluoroquinolone-resistant; *Salmonellae*, fluoroquinolone-resistant; and *Neisseria gonorrhoeae*, cephalosporin-resistant, fluoroquinolone-resistant. The three medium-priority pathogens are: *Streptococcus pneumoniae*, penicillin-non-susceptible; *Haemophilus influenzae*, ampicillin-resistant; and *Shigella* spp., fluoroquinolone-resistant.

The WHO list was developed in collaboration with the Division of Infectious Diseases at the University of Tübingen in Germany. To determine which bacteria to include, researchers looked at a few factors, including how deadly the infections caused by the bacteria are, how resistant the bacteria are to existing antibiotics, how easily the bacteria spread, the number of treatment options available, and how preventable infections caused by the bacteria are, according to the WHO.

One of the main goals of the list is to drive more research into the development of new antibiotics and inspire governments to invest in this research and development, WHO officials said. In addition, better prevention and the appropriate use of existing antibiotics are required in order to adequately address this threat, they added.

Indeed, "one issue is that one of the things that promotes antibiotic resistance is use of an antibiotic," Nelson said. "In general, antibiotics tend to be overused, and that's one of the things that leads to resistance."

http://bit.ly/2mJqBbv

UN body urges China to act as bird flu deaths spike

The UN's food agency on Friday urged China to step up efforts to contain and eliminate a strain of bird flu which has killed scores of people this year.

The Food and Agriculture Organization (FAO) warned that countries neighbouring China were at "high risk" of exposure to the H7N9 strain, which has recently mutated to become far more deadly for chicken than it had been.

The agency also warned that wild birds could carry the strain of the virus to Europe and the Americas, adding that it was baffled as to why China's efforts to contain the outbreak had not worked as well as anticipated.

The FAO's statement came after China reported last month that 79 people had died in January alone, the deadliest H7N9 outbreak since the strain first appeared in humans in 2013.

Nearly one in three people who contract H7N9 die from it.

FAO said the recent surge in cases in eastern and southern parts of China meant the virus had caused more reported human cases than all other types of avian influenza viruses, such as H5N1 and H5N6, combined.

Vincent Martin, the FAO's representative in China, said efforts to contain the outbreak needed to focus on eliminating the strain at its source. "Targeted surveillance to detect the disease and clean infected

farms and live bird markets, intervening at critical points along the poultry value chain—from farm to table—is required," he said.

"There should be incentives for everybody involved in poultry production and marketing to enforce disease control."

The agency recognised that China had invested heavily in surveillance of live bird markets and poultry farms while noting that monitoring has "proven particularly challenging as until recently (the strain) has shown no or few signs of disease in chickens."

The organisation said new evidence from Guangdong in southern China pointed to H7N9 having mutated to become much deadlier for chickens while retaining its capacity to make humans severely ill.

This could make it easier to spot outbreaks, as infected chickens are typically dying within 48 hours of infection, but it also underscores the potentially huge economic implications of the mutation, FAO said.

The FAO emphasised that there was no risk of humans catching the potentially deadly influenza strain by eating chicken.

China has suspended trade in live poultry in several cities, urged consumers to switch to frozen chicken, enforced stricter hygiene standards in fresh food markets, and culled affected flocks.

"With all the efforts taken by China and partners, there is a pressing need to understand why these measures have not worked as well as expected," the FAO said.

http://nyti.ms/2n3Un6I

Trump Plan Eliminates a Global Sentinel Against Disease, Experts Warn

Trump administration's proposed cuts to the N.I.H. would make America vulnerable again

By DONALD G. McNEIL Jr. MARCH 17, 2017

Nobody in the United States has ever died from an intercontinental missile strike. Over the past 50 years, hundreds of billions of dollars have been spent on silos, submarines, bombers and satellites to ensure that does not happen.

During the same period, about 1.3 million Americans have died from intercontinental virus strikes. The toll includes one American dead of Ebola, 2,000 dead of West Nile virus, 700,000 dead of AIDS, and 1.2 million dead of flu — a virus that returns from abroad each winter.

The federal budget to stop these threats is infinitely smaller than the Pentagon's, and the Trump administration's proposed cuts to the National Institutes of Health, and particularly its plan to eliminate the Fogarty International Center at the N.I.H., would, global health experts say, make America vulnerable again.

The Fogarty center, based in Bethesda, Md., was one of the few specific trims in President Trump's "skinny budget." It is an odd target: Eliminating it would save only $69 million. The administration did not explain why it was picked, leaving scientists to surmise that it was because the center's grants pay American doctors to train foreign ones. Mr. Trump has a well-known "America First" bent.

But most of those trainees focus on diseases that circle the globe, researchers point out, including flu, mosquito-borne viruses, vaccine-preventable diseases and bioterrorism agents.

The idea of eliminating the center "is just atrocious," said Dr. Daniel G. Bausch, a Tulane University virologist and the scientific program director at the American Society of Tropical Medicine and Hygiene. "It would have a severe impact not just on global health but on American health."

"Even if you don't care about your neighbors, if you see a fire across the street, your best bet to protect your house is not to just stand in your yard with a bucket of water," he added. "It's to help put it out."

New viral threats are constant. Pathogens like SARS, MERS, dengue and H7N9 avian flu have already probed America's defenses: Cases have reached these shores in people or in birds, but have not yet killed anyone.

The Zika virus, which is lethal to unborn babies, is still probing our limits; it is expected to return to this country this summer. Still in the wings are a host of other threats: The Nipah virus and Lassa fever, for

example, are considered so dangerous that the Bill and Melinda Gates Foundation and other donors recently announced a $500 million fund to jump-start the development of vaccines against them.

Rift Valley fever, Japanese encephalitis, Crimean-Congo hemorrhagic fever and many others lie in wait, and they are less remote than most Americans realize. Crimean-Congo fever, despite its exotic hybrid Russian-African name, circulates even in Spain. It killed someone there last year.

The early-warning system that protects America against viruses resembles the one that protects it against missiles. A network of laboratories around the world, known as World Health Organization reference labs, collects samples from disease outbreaks in local humans, animals and even insects. Researchers share the genetic sequences, track dangerous mutations, and ship virus samples on to more sophisticated labs that can turn them into vaccines.

Only a tiny number of these sentinel laboratories are in American hands. The Navy runs two in Egypt and Cambodia, for example. And the Centers for Disease Control and Prevention in Atlanta, of course, is one of the apex labs in the W.H.O. reference system, like the top labs in Paris, Moscow and Beijing.

But the system's furthest-flung sentries — the ones most likely to make first contact with a new viral foe — are those in the world's poorest countries. These labs are often the descendants of British, French, Dutch or Belgian facilities founded during the colonial era or those started by the Soviets.

Vital as they are to global health, they are usually underfunded and underequipped, and their personnel undertrained. The Fogarty center helps remedy that.

Dr. Bausch has a $50,000 Fogarty grant to plan what could turn into a $2 million to $3 million investment in Sierra Leone, one of the three West African countries where Ebola killed 11,000 people in 2014.

"That would buy lab equipment, train people to run it and to do contact-tracing during an outbreak," Dr. Bausch said. "The ability to do that is not a given in West Africa. If it had been, we wouldn't have had that massive Ebola outbreak."

The skills and equipment donated for one disease often help stop another. For example, early last year, Brazilian ultrasound specialists who had been trained under a Fogarty grant to spot brain abnormalities in Chagas disease victims were among the first to detect early signs of brain deformity in fetuses with Zika.

And Ebola was stopped in Nigeria in 2014 because emergency operations centers and case-detection teams that had been created to find polio victims were drafted to track Ebola cases instead.

Grants by the Pentagon let foreign militaries train with American forces, building alliances. The same is true of Fogarty grants.

When he worked in Pakistan, Bangladesh and Mongolia, said Dr. Sten H. Vermund, an AIDS expert who is now dean of the Yale School of Public Health, "I was able to offer Fogarty-supported training to my overseas partners, and they became the leading lights in H.I.V. when the epidemic hit."

"Cutting the tiny Fogarty budget is penny-wise and pound-foolish," he added. "In the old days, we'd float in, get our samples, and leave. We were accused, correctly, of scientific exploitation. Nowadays, you don't do research abroad if you don't offer the host country something."

Once tropical diseases emerge, "they know no borders," said Dr. Chris Beyrer, president of the International AIDS Society. "America is not hived off from the rest of the planet, and it's incredibly important to our biosecurity to have surveillance capability — which means partners in other countries. That's what Fogarty does."

http://bit.ly/2nC08tE

When things go wrong in an automated world, would we still know what to do?

Are we losing our skills as we hand more tasks to automated systems?

Peter Fisher

We live in a world that is both increasingly complex and automated. So just as we are having to deal with more complex problems, automation is leading to an atrophy of human skills that may leave us more vulnerable when responding to unexpected situations or when things go wrong.

Consider the final minutes of Air France Flight 447, which crashed into the Atlantic in May 2009 after leaving Rio de Janeiro, Brazil, for Paris, France.

Its flight recorder revealed utter confusion in the cockpit. The plane became tilted upwards at 15º with an automated voice repetitively calling "stall, stall". Yet the pilots were reeling, one exclaiming: "[…] we don't understand anything."

This is not the place to go into the ins and outs of that ill-fated flight, other than to note that any system designed to deal automatically with contingencies the majority of the time leaves a degraded skill base for the minority of situations the designers couldn't foresee.

Speaking to Vanity Fair, Nadine Sarter, an industrial engineer at the University of Michigan, recalls a conversation with five engineers involved in building a particular aircraft.

I started asking, 'Well, how does this or that work?' And they could not agree on the answers. So I was thinking, if these five engineers cannot agree, the poor pilot, if he ever encounters that particular situation … well, good luck.

In effect the complexity of judiciously flying highly intricate high-tech airliners has been outsourced to a robot, with flight engineers to all intents and purposes gone from cockpits. Only older pilots and ex air force pilots retain those detailed skills.

Back on terra firma, in an autonomous driving world there could be entire future generations with no practical experience whatsoever in driving and navigating a vehicle.

We're already seeing an indication of what can go wrong when humans leave control to autonomous systems.

An investigation into the fatal crash of a Tesla Model S with autopilot noted that the company provided information about "system limitations" to drivers. In that case, it's still up to drivers to pay attention.

But what chance would a person have of taking over any controls should things start to go wrong in their future fully autonomous vehicle. Would they even know how to spot the early signs of impending disaster?

Losing our way?

Driving this is a technological determinism that believes any and all innovation is intrinsically good. While emerging technologies may yet define what it is to be human, the challenge is to recognise the risk and what to do to make sure things don't go wrong. That's getting harder as we've been adding to complexity, especially with autonomous driving of suburban trains, air taxis and delivery drones.

System designers have been building bigger and more intertwined systems to share computer processing load even though this makes their creations prime candidates for breakdown. They are overlooking the fact that once everything is connected, problems can spread as readily as solutions, sometimes more so. The growing and immense complexity of an automated world poses similar risks.

Danger points

In hindsight, what is needed is an ability to cut networks free when there are failure points, or at least to seal off parts of a single network when there are failure points elsewhere within it.

This "islanding" is a feature of smart electricity grids providing scope to split the network into fragments that are able to self-sustain their internal power demand. Modelling has shown that fewer connections can lead to more security.

Could emergent complexity science help pinpoint where the danger points might lie in highly interconnected networks? Marten Scheffer and colleagues thought so. He had seen similarities between the behaviour of (his) natural systems and economic and financial systems.

His earlier work on lakes, coral reefs, seas, forests and grasslands, found that environments subject to gradual changes like climate, nutrient load and habitat loss can reach tipping points that flip them into a sometimes irreversible lower state.

Could bankers and economists grappling with the stability of financial markets learn from researchers in ecology, epidemiology and climatology to develop markers of the proximity to critical thresholds and system breakdown?

In February 2016 this all came together in the form of a paper on complexity theory and financial regulation co-authored by a wide range of experts including an economist, banker, physicist, climatologist, ecologist, zoologist, veterinarian and epidemiologist.

They recommended an online integration of data, methods and indicators, feeding into stress tests for global socioeconomic and financial systems in near-realtime. The former is similar to what's been achieved in dealing with other complex systems such as the weather.

We can begin to see how our example of an autonomous driving world folds over into questions of network stability. Imagine a highly interconnected network of autonomous vehicles.

There's a clear need to know how to detect and isolate any potential failure points in such a network, before things go wrong with potentially tragic consequences. This is more than just protecting driver and passenger from any system failure in a single autonomous vehicle.

It's time to think how we might use those multidisciplinary advances in understanding the stability of such large scale networks to avoid drastic consequences.

Peter Fisher does not work for, consult, own shares in or receive funding from any company or organisation that would benefit from this article, and has disclosed no relevant affiliations beyond the academic appointment above.

RMIT University and Victoria State Government provide funding as strategic partners of The Conversation AU.

http://bit.ly/2nIth3o

Do you really have high blood pressure?

Over half of family doctors in Canada still use manual devices to measure blood pressure, which may lead to misdiagnosis

Montreal - A study by researchers at the University of Montreal Hospital Research Centre (CRCHUM) shows that more than half of family doctors in Canada are still using manual devices to measure blood pressure, a dated technology that often leads to misdiagnosis.

"About 20% of people receiving treatment for hypertension don't actually have a problem and do not need medication. This is due mainly to the fact that their blood pressure was improperly measured," said lead author Janusz Kaczorowski, whose study was recently published in the journal Canadian Family Physician.

Kaczorowski, a medical sociologist, is a CRCHUM researcher and professor in the Department of Family and Emergency Medicine at Université de Montréal.

Getting one's blood pressure taken during a visit to the doctor is a routine procedure. It also provides crucial medical data. In Canada, one adult in five suffers from hypertension which represents the greatest global risk factor for death and disability.

Blood pressure is defined as the pressure that the blood exerts on artery walls. Measured in the arm artery, it is expressed in two numbers: the value when the heart contracts (systolic blood pressure) and the value when the heart relaxes between two contractions (diastolic blood pressure). Blood pressure is considered normal when systolic pressure is below 140 mmHg and diastolic pressure is below 90 mmHg. Above these values, a person is said to have high blood pressure (hypertension).

The technology behind tensiometers or sphygmomanometers, the devices used to measure blood pressure, has changed a great deal over the last 20 years. Today, automatic electronic measuring devices, known as oscillometric devices, are available. In 2016, The Canadian

Hypertension Education Program (CHEP) Guidelines recommended that electronic measurement is preferable to manual measurement.

An increasing number of medical clinics are equipped with automatic electronic devices. Yet in the spring of 2016, Kaczorowski's team conducted a survey among Canadian family doctors: 52% of the 769 respondents indicated that they used a manual tensiometer to measure blood pressure. Only 43% used an automatic device.

"Clinicians should use automatic devices," Kaczorowski said. "They are more expensive but more precise because they take several measurements. Manual measurement is acceptable if it's properly done, but that's often not the case. To take blood pressure the right way, a 12- to 15-minute period is required. We know that the average visit to a family doctor lasts 10 minutes. We have to rethink how patient visits are organized so that the patient can be left alone in a room while the measurement is taken."

Automated measurement has the advantage of eliminating what's known as white-coat syndrome, which refers to artificially high blood pressure resulting from the stress of being in a doctor's office and human interaction. The researchers believe that clinicians should adopt these devices to detect individuals likely to suffer from high blood pressure, in accordance with the Canadian guidelines.

In Canada, healthcare costs attributable to hypertension were evaluated at more than $13 billion in 2010. "If people who take high-blood-pressure medication had their blood pressure measured incorrectly, the financial implications are considerable, in addition to the side effects, which could be avoided," said Kaczorowski.

The CHEP Guidelines state that modifying health-related behaviours is an efficient way of preventing and treating high blood pressure and of reducing the risk of cardiovascular disease. It is possible to lower blood pressure through a healthy diet, regular physical activity, moderate alcohol consumption, reduced intake of dietary sodium, avoidance of exposure to tobacco products, and stress management. To find out more, please visit: http://www.hypertension.ca

About this study

The study How do family physicians measure blood pressure in routine clinical practice? A national survey of Canadian family physicians was published on March 14, 2017 in Canadian Family Physician. To find out more, the study is available at:

http://www.cfp.ca/content/63/3/e193.full.pdf+html

Source: University of Montreal Hospital Research Centre (CRCHUM)