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**Uncorking East Antarctica yields unstoppable sea-level rise**  
*The melting of a rather small ice volume on East Antarctica's shore could trigger a persistent ice discharge into the ocean, resulting in unstoppable sea-level rise for thousands of years to come.*

This is shown in a study now published in Nature Climate Change by scientists from the Potsdam Institute for Climate Impact Research (PIK). The findings are based on computer simulations of the Antarctic ice flow using improved data of the ground profile underneath the ice sheet.

"East Antarctica's Wilkes Basin is like a bottle on a slant," says lead-author Matthias Mengel, "once uncorked, it empties out." The basin is the largest region of marine ice on rocky ground in East Antarctica.

Currently a rim of ice at the coast holds the ice behind in place: like a cork holding back the content of a bottle. While the air over Antarctica remains cold, warming oceans can cause ice loss on the coast. Ice melting could make this relatively small cork disappear – once lost, this would trigger a long term sea-level rise of 300-400 centimeters. "The full sea-level rise would ultimately be up to 80 times bigger than the initial melting of the ice cork," says co-author Anders Levermann.

"Until recently, only West Antarctica was considered unstable, but now we know that its ten times bigger counterpart in the East might also be at risk," says Levermann, who is head of PIK's research area Global Adaptation Strategies and a lead-author of the sea-level change chapter of the most recent scientific assessment report by the Intergovernmental Panel on Climate Change, IPCC. This report, published in late September, projects Antarctica's total sea level contribution to be up to 16 centimeters within this century.

"If half of that ice loss occurred in the ice-cork region, then the discharge would begin. We have probably overestimated the stability of East Antarctica so far," says Levermann.

**Emitting greenhouse-gases could start uncontrollable ice-melt**

Melting would make the grounding line retreat – this is where the ice on the continent meets the sea and starts to float. The rocky ground beneath the ice forms a huge inland sloping valley below sea-level. When the grounding line retreats from its current position on a ridge into the valley, the rim of the ice facing the ocean becomes higher than before. More ice is then pushed into the sea, eventually breaking off and melting. And the warmer it gets, the faster this happens.

Complete ice discharge from the affected region in East Antarctica takes five thousand to ten thousand years in the simulations. However, once started, the discharge would slowly but relentlessly continue until the whole basin is empty, even if climate warming stopped. "This is the underlying issue here", says Matthias Mengel. "By emitting more and more greenhouse gases we might trigger responses now that we may not be able to stop in the future." Such extensive sea level rise would change the face of planet Earth – coastal cities such as Mumbai, Tokyo or New York are likely to be at risk.

*Article: Mengel, M., Levermann, A. (2014): Ice plug prevents irreversible discharge from East Antarctica. Nature Climate Change (online) [DOI: 10.1038/NCLIMATE2226]*

*Weblink to the article:*

<http://www.nature.com/nclimate/journal/vaop/ncurrent/full/nclimate2226.html>

*Related article: Levermann, A., Bamber, J., Drijfhout, S., Ganopolski, A., Haerberli, W., Harris, N.R.P., Huss, M., Krüger, K., Lenton, T., Lindsay, R.W., Notz, D., Wadhams, P., Weber, S. (2012): [Potential climatic transitions with profound impact on Europe - Review of the current state of six 'tipping elements of the climate system'](#). Climatic Change 110 (2012), 845-878, [DOI 10.1007/s10584-011-0126-5]*

<http://bit.ly/1hGHHGy>

**Soy sauce molecule may unlock drug therapy for HIV patients**  
**Compounds can be 70 times more potent than Tenofovir, a first-line HIV drug**

COLUMBIA, Mo. – For HIV patients being treated with anti-AIDS medications, resistance to drug therapy regimens is commonplace. Often, patients develop resistance to first-line drug therapies, such as Tenofovir, and are forced to adopt more potent medications. Virologists at the University of Missouri now are testing the next generation of medications that stop HIV from spreading, and are using a molecule related to flavor enhancers found in soy sauce, to develop compounds that are more potent than Tenofovir.

"Patients who are treated for HIV infections with Tenofovir, eventually develop resistance to the drugs that prevent an effective or successful defense against the virus," said Stefan Sarafianos, associate professor of molecular microbiology and immunology in the University of Missouri School of Medicine, and a virologist at the Bond Life Sciences Center at MU. "EFdA, the molecule we are studying, is less likely to cause resistance in HIV patients because it is more readily activated and is less quickly broken down by the body as similar existing drugs."

In 2001, a Japanese soy sauce company inadvertently discovered the EFdA molecule while trying to enhance the flavor of their product. The flavor enhancer is part of the family of compounds called "nucleoside analogues" which is very similar to existing drugs for the treatment of HIV and other viruses. EFdA

samples were sent for further testing, which confirmed EFdA's potential usefulness against HIV and started more than a decade of research.

EFdA, along with eight existing HIV drugs, is part of the class of compounds called nucleoside reverse transcriptase inhibitors (NRTIs). NRTIs "hijack" the HIV replicating process by "tricking" building blocks inside the virus. Since EFdA appears similar to those building blocks, the virus is misled into using the imposter, which prevents HIV replication and halts the spread of the virus.

In their latest study, Sarafianos and his colleagues, including researchers from the University of Pittsburgh and the National Institutes of Health, helped define how EFdA works on a molecular level.

Using virology techniques and nuclear magnetic resonance spectroscopy (NMR), they pieced together the exact structure and configuration of the molecule.

Compounds developed by Sarafianos and his team currently are being tested for usefulness as potential HIV-halting drugs with pharmaceutical company Merck.

"The structure of this compound is very important because it is a lock-and-key kind of mechanism that can be recognized by the target," Sarafianos said. "EFdA works extremely well on HIV that is not resistant to anti-AIDS drugs, it also works even better on HIV that's become resistant to Tenofovir."

*Grants from the National Institutes of Health funded this research which was published the Journals Retrovirology, Antimicrobial Agents and Chemotherapy and The International Journal of Pharmaceutics. Editor's Note: For a longer version of this story, please visit, <http://decodingscience.missouri.edu/2014/04/sarafianos-efda/>*

<http://bit.ly/1hGHNxL>

## **Study finds turtles are closer kin to birds, crocodiles than to lizards, snakes**

*What are turtles, and where did they come from?*

By Eric Gershon

Precise answers to these questions have long eluded scientists. But new research led by Daniel Field of Yale University and the Smithsonian Institution recasts the turtle's disputed evolutionary history, providing fresh evidence that the familiar reptiles are more closely related to birds and crocodiles than to lizards and snakes.

"These observations address one of the defining biological questions of the past decade, helping us illuminate the murkier reaches of reptile evolution," said Field, a doctoral candidate in geology and geophysics at Yale and a predoctoral fellow at the Smithsonian Institution's National Museum of Natural History.

"We show that turtles share a more recent common ancestor with birds and crocodilians - a group known as archosaurs - than with lizards and snakes." Field and collaborators report their findings May 5 in the journal *Evolution and Development*.

Reptiles comprise a vast animal group of more than 20,000 species. The interrelationships of some subgroups are well understood, the scientists said. Birds are most closely related to crocodilians among living reptiles, for example, while snakes, lizards, and New Zealand's tuatara form a natural group. But turtles' precise place has been unclear, in part due to conflicting research results.

For example, although a growing number of DNA sequence studies show a close evolutionary kinship between turtles and archosaurs (birds, crocodilians), these studies have sometimes been contradicted by anatomical studies and other research involving small biomolecules called microRNAs that indicate a closer relationship between turtles and lizards and snakes.

MicroRNAs are viewed by some scientists as especially good evolutionary markers.

Field and collaborators revisited a foundational microRNA study, applied updated criteria for microRNA identification, and came to a different conclusion. "Several studies purporting to investigate microRNAs misidentified other small RNA molecules as microRNAs," said Field.

"In our study, we collected new microRNA data from a variety of vertebrate animals and adhered to strict new guidelines for microRNA identification. When the experiment was redone, support for turtles as closer relatives of lizards and snakes turned out to be spurious, while support for turtles as closer relatives of birds and crocodilians was very strong."

In short, he said, microRNAs and DNA sequences now yield a common signal uniting turtles and archosaurs (birds and crocodilians).

"These results are exciting because, for the first time, we obtain a consistent evolutionary signal from different sources of molecular data regarding the evolutionary position of turtles," Field said.

*The paper is "Toward concision in reptile phylogeny: microRNAs support an archosaur, not lepidosaur, affinity for turtles."*

*Other authors on the paper are Jacques Gauthier, also of Yale, Ben King of the Mount Desert Island Biological Research Station, Davide Pisani of the University of Bristol, Tyler Lyson of the Smithsonian Institution, and Kevin Peterson of Dartmouth College.*

*Support for the research came from the Yale Peabody Museum, the Government of Alberta, the Canadian Natural Sciences and Engineering Research Council, and NASA.*

<http://bit.ly/IgsOhbz>

## Where DNA's copy machine pauses, cancer could be next

*Fragile sites that can be a breeding ground for human cancers appear in specific areas of the genome where the DNA-copying machinery is slowed or stalled*

DURHAM, N.C. - Each time a human cell divides, it must first make a copy of its 46 chromosomes to serve as an instruction manual for the new cell.

Normally, this process goes off without a hitch. But from time to time, the information isn't copied and collated properly, leaving gaps or breaks that the cell has to carefully combine back together.

Researchers have long recognized that some regions of the chromosome, called "fragile sites," are more prone to breakage and can be a breeding ground for human cancers.

But they have struggled to understand why these weak spots in the genetic code occur in the first place.

A comprehensive mapping of the fragile sites in yeast by a team of Duke researchers shows that fragile sites appear in specific areas of the genome where the DNA-copying machinery is slowed or stalled, either by certain sequences of DNA or by structural elements.

The study, which appears May 5 in Proceedings of the National Academy of Sciences, could give insight into the origins of many of the genetic abnormalities seen in solid tumors.

"Other studies have been limited to looking at fragile sites on specific genes or chromosomes," said Thomas D. Petes, Ph.D., the Minnie Geller professor of molecular genetics and microbiology at Duke University School of Medicine. "Ours is the first to examine thousands of these sites across the entire genome and ask what they might have in common."

The term "fragile sites" was first coined in the 1980s to describe the chromosome breaks that appeared whenever a molecule called DNA polymerase — responsible for copying DNA -- was blocked in mammalian cells.

Since that discovery, research in the yeast *Saccharomyces cerevisiae* has shown that certain DNA sequences can make the polymerase slow down or pause as it makes copies. However, none of them have shown how those delays result in fragile sites.

In this study, Petes wanted to find the link between the copier malfunction and its genetic consequences on a genome-wide scale.

First, he knocked down the levels of DNA polymerase in yeast cells to ten-fold lower than normal. Then he used microarray or "gene chip" technology to map

where segments of DNA had been rearranged, indicating that a fragile site had once been there.

After finding those fragile sites, his laboratory spent more than a year combing through the literature for any recurring themes among the genomic regions they had uncovered.

Eventually they showed that the fragile sites were associated with sequences or structures that stalled DNA replication, esoteric entities such as inverted repeats, replication termination signals, and transfer RNA genes.

"We only published the tip of the iceberg -- there is a lot of work you don't see because the connections simply weren't significant enough. Even now, we didn't find any single sequence motif that would very clearly predict a fragile site," said Petes.

"I think there are just a lot of ways to slow down replication, so there is not just one signal to indicate that would occur."

In addition, Petes found that these fragile sites created a surprisingly unstable genome, resulting in a chaotic milieu of rearrangements, duplications and deletions of pieces of DNA or even the gain or loss of entire chromosomes.

"The ability to analyze these sites on a genome-wide basis is an important advance," said Gray Crouse, Ph.D., an expert unaffiliated with the new study who is a professor of biology at Emory University.

"It has been known for a long time that many cancer cells have an abnormal number of chromosomes, and many different chromosome rearrangements have been observed in various tumor cells. It is likely that there are many different causes of chromosome instability in cancer cells. The current work suggests that those chromosomal rearrangements observed at fragile sites and found in solid tumors may be due to breaks from perturbed replication."

*The research was supported by grants from the National Institutes of Health National Institutes of Health (GM24110, GM52319, and T32-AI52080).*

*CITATION: "Genome-wide high-resolution mapping of chromosome fragile sites in Saccharomyces cerevisiae," Wei Song, Margaret Dominska, Patricia W. Greenwell, Thomas D. Petes. PNAS, May. 5, 2014. DOI:*

<http://www.pnas.org/cgi/doi/10.1073/pnas.1406847111>

<http://bit.ly/1iHAObK>

## Having eczema may reduce your risk of skin cancer

*Eczema caused by defects in the skin could reduce the risk of developing skin cancer, according to new research by King's College London.*

The immune response triggered by eczema could help prevent tumour formation by shedding potentially cancerous cells from the skin.

There is ongoing debate surrounding allergic diseases and their impact on the likelihood of developing cancer, with some studies suggesting that eczema is associated with a reduced risk of skin cancer. However, it is difficult to draw firm conclusions based on studies of human populations because eczema symptoms vary in severity and drugs used to treat the condition might also influence cancer. Published today in eLife, the study by researchers at King's College London is the first to show that allergy caused by the skin defects could actually protect against skin cancer.

Skin cancer is a common and growing problem, accounting for one in every three cancers diagnosed worldwide, according to the World Health Organization. Recent findings suggest that malignant melanoma, the most dangerous form of skin cancer, is now five times more common in the UK than it was in the 1970s. Eczema can result from the loss of structural proteins in the outermost layers of the skin, leading to a defective skin barrier. Genetically engineered mice lacking three skin barrier proteins ('knock-out' mice) were used in the King's study to replicate some of the skin defects found in eczema sufferers.

The researchers, funded by the Medical Research Council (MRC), the Wellcome Trust and Cancer Research UK, compared the effects of two cancer-causing chemicals in normal mice and mice with the barrier defect (the knock-out mice). The number of benign tumours per mouse was six times lower in knock-out mice than in normal mice. The findings suggest that defects in the epidermal barrier protected the genetically engineered mice against benign tumour formation.

Researchers found that both types of mice were equally susceptible to acquiring cancer-causing mutations. However, an exaggerated inflammatory reaction in knock-out mice led to enhanced shedding of potentially cancerous cells from the skin. This cancer-protective mechanism bears similarities to that which protects skin from environmental assaults such as bacteria.

Professor Fiona Watt, Director of the Centre for Stem Cells and Regenerative Medicine, said: 'We are excited by our findings as they establish a clear link between cancer susceptibility and an allergic skin condition in our experimental model. They also support the view that modifying the body's immune system is an important strategy in treating cancer. I hope our study provides some small consolation to eczema sufferers – that this uncomfortable skin condition may actually be beneficial in some circumstances.'

Dr Mike Turner, Head of Infection and Immunobiology at the Wellcome Trust, said: 'Skin cancer is on the rise in many countries and any insight into the body's ability to prevent tumour formation is valuable in the fight against this form of

cancer. These findings that eczema can protect individuals from skin cancer support theories linking allergies to cancer prevention and open up new avenues for exploration whilst providing some (small) comfort for those suffering from eczema.'

<http://bit.ly/1jhOBkZ>

**Humans and squids evolved the same eyes using the same genes**  
*Despite belonging to completely different lineages, humans and squid evolved through tweaks to the same gene*

Eyes and wings are among the most stunning innovations evolution has created. Remarkably these features have evolved multiple times in different lineages of animals. For instance, the avian ancestors of birds and the mammalian ancestors of bats both evolved wings independently, in an example of convergent evolution. The same happened for the eyes of squid and humans. Exactly how such convergent evolution arises is not always clear. In a new study, published in Nature's Scientific Reports, researchers have found that, despite belonging to completely different lineages, humans and squid evolved through tweaks to the same gene.

**Eyes are the prize**

Like all organs, the eye is the product of many genes working together. The majority of those genes provide information about how to make part of the eye. For example, one gene provides information to construct a light-sensitive pigment. Another gene provides information to make a lens. Most of the genes involved in making the eye read like a parts list – this gene makes this, and that gene makes that. But some genes orchestrate the construction of the eye. Rather than providing instructions to make an eye part, these genes provide information about where and when parts need to be constructed and assembled. In keeping with their role in controlling the process of eye formation, these genes are called "master control genes".

The most important of master control genes implicated in making eyes is called Pax6. The ancestral Pax6 gene probably orchestrated the formation of a very simple eye – merely a collection of light-sensing cells working together to inform a primitive organism of when it was out in the open versus in the dark, or in the shade.

Today the legacy of that early Pax6 gene lives on in an incredible diversity of organisms, from birds and bees, to shellfish and whales, from squid to you and me. This means the Pax6 gene predates the evolutionary diversification of these lineages – during the Cambrian period, some 500m years ago.

The Pax6 gene now directs the formation of an amazing diversity of eye types. Beyond the simple eye, it is responsible for insects' compound eye, which uses a group of many light-sensing parts to construct a full image. It is also responsible for the type of eye we share with our vertebrate kin: camera eye, an enclosed structure with its iris and lens, liquid interior, and image-sensing retina. In order to create such an elaborate structure, the activities Pax6 controlled became more complex. To accommodate this, evolution increased the number of instructions that arose from a single Pax6 gene.

### **Making the cut**

Like all genes, the Pax6 gene is an instruction written in DNA code. In order for the code to work, the DNA needs to be read and then copied into a different kind of code. The other code is called RNA. RNA code is interesting in that it can be edited. One kind of editing, called splicing, removes a piece from the middle of the code, and stitches the two ends together. The marvel of splicing is that it can be used to produce two different kinds of instructions from the same piece of RNA code. RNA made from the Pax6 can be spliced in just such a manner. As a consequence, two different kinds of instructions can be generated from the same Pax6 RNA.

In the new study, Atsushi Ogura at the Nagahama Institute of Bio-Science and Technology and colleagues found that Pax6 RNA splicing has been used to create a camera eye in a surprising lineage. It occurs in the lineage that includes squid, cuttlefish, and octopus – the cephalopods.

Cephalopods have a camera eye with the same features as the vertebrate camera eye. Importantly, the cephalopod camera eye arose completely independently from ours. The last common ancestor of cephalopods and vertebrates existed more than 500m years ago. Pax6 RNA splicing in cephalopods is a wonderful demonstration of how evolution fashions equivalent solutions via entirely different routes. Using analogous structures, evolution can provide remarkable innovations.

<http://bit.ly/1jOByf4>

### **Novel antioxidant makes old arteries seem young again, CU-Boulder study finds**

#### *May be able to reverse some of the negative effects of aging on arteries*

An antioxidant that targets specific cell structures – mitochondria - may be able to reverse some of the negative effects of aging on arteries, reducing the risk of heart disease, according to a new study by the University of Colorado Boulder. When the research team gave old mice - the equivalent of 70- to 80-year-old

humans - water containing an antioxidant known as MitoQ for four weeks, their arteries functioned as well as the arteries of mice with an equivalent human age of just 25 to 35 years.

The researchers believe that MitoQ affects the endothelium, a thin layer of cells that lines our blood vessels. One of the many functions of the endothelium is to help arteries dilate when necessary. As people age, the endothelium is less able to trigger dilation and this leads to a greater susceptibility to cardiovascular disease.

"One of the hallmarks of primary aging is endothelial dysfunction," said Rachel Gioscia-Ryan, a doctoral student in CU-Boulder's Department of Integrative Physiology and lead author of the new study. "MitoQ completely restored endothelial function in the old mice. They looked like young mice."

The study, published in the Journal of Physiology, was funded by the National Institute on Aging, one of the 27 institutes and centers of the National Institutes of Health and a leader in the scientific effort to understand the nature of aging. To trigger blood vessel dilation, the endothelium makes nitric oxide. As we age, the nitric oxide meant to cause dilation is increasingly destroyed by reactive oxygen species such as superoxide, which are produced by many components of our body's own cells, including organelles called mitochondria.

In a double-whammy, superoxide also reacts directly with the enzyme that makes nitric oxide, reducing the amount of nitric oxide being produced to begin with. All of this means less blood vessel dilation.

Even in the young and healthy, mitochondria produce superoxide, which is necessary in low levels to maintain important cellular functions. Superoxide is kept in check by the body's own antioxidants, which combine with superoxide to make it less reactive and prevent oxidative damage to cells.

"You have this kind of balance, but with aging there is this shift," said Gioscia-Ryan, who works in Professor Douglas Seals' Integrative Physiology of Aging Laboratory at CU-Boulder. "There become way more reactive oxygen species than your antioxidant defenses can handle."

That phenomenon, known as oxidative stress, occurs when the cells of older adults begin to produce too much superoxide and other reactive oxygen species. Mitochondria are a major source of superoxide in aging cells. The increased superoxide not only interacts with nitric oxide and the endothelium, but can also attack the mitochondria themselves. The damaged mitochondria become increasingly dysfunctional, producing even more reactive oxygen species and creating an undesirable cycle.

Past studies have looked at whether taking antioxidant supplements long term could improve vascular function in patients with cardiovascular disease by restoring balance to the levels of superoxide, but they've largely shown that the strategy isn't effective.

This new study differs because it uses an antioxidant that specifically targets mitochondria. Biochemists manufactured MitoQ by adding a molecule to ubiquinone (also known as coenzyme Q10), a naturally occurring antioxidant. The additional molecule makes the ubiquinone become concentrated in mitochondria.

"The question is, 'Why aren't we all just taking a bunch of vitamin C?'" Gioscia-Ryan said. "Scientists think that, taken orally, antioxidants like vitamin C aren't getting to the places where the reactive oxygen species are being made. MitoQ basically tracks right to the mitochondria."

The findings of the study indicate that the strategy of specifically targeting the mitochondria may be effective for improving the function of arteries as we age. In addition to improving endothelial function, the MitoQ treatment increased levels of nitric oxide, reduced oxidative stress and improved the health of the mitochondria in the arteries of old mice.

*Other CU-Boulder co-authors of the study include postdoctoral researchers Thomas LaRocca and Amy Sindler, professional research assistant Melanie Zigler and Seals. Professor Michael Murphy of the Medical Research Council's Mitochondrial Biology Unit in Cambridge, England, who helped develop MitoQ, also is a co-author. MitoQ is manufactured by MitoQ Ltd., and is available as a dietary supplement and skin cream. The study was supported by award number T32AG000279 from the National Institute on Aging. The content of the published study is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.*

<http://bit.ly/1j9sQty>

### **Yawning to cool the brain**

#### ***Yawning frequencies of people vary with temperature of the season***

Common belief is that yawning helps to increase the oxygen supply. However, previous research has failed to show an association between yawning and blood oxygen levels. New research by a team of researchers led by Psychologist Andrew Gallup of SUNY College at Oneonta, USA now reveals that yawning cools the brain.

Sleep cycles, cortical arousal and stress are all associated with fluctuations in brain temperature, Yawning subsequently functions to keep the brain temperature balanced and in optimal homeostasis. According to this theory, yawning should also be easily manipulated by ambient temperature variation, since exchange with cool ambient air temperature may facilitate lowering brain

temperature. Specifically, the researchers hypothesized that yawning should only occur within an optimal range of temperatures, i.e., a thermal window.

To test this, Jorg Massen and Kim Dusch of the University of Vienna measured contagious yawning frequencies of pedestrians outdoors in Vienna, Austria, during both the winter and summer months, and then compared these results to an identical study conducted earlier in arid climate of Arizona, USA. Pedestrians were asked to view a series of images of people yawning, and then they self-reported on their own yawning behavior.

Results showed that in Vienna people yawned more in summer than in winter, whereas in Arizona people yawned more in winter than in summer. It turned out that it was not the seasons themselves, nor the amount of daylight hours experienced, but that contagious yawning was constrained to an optimal thermal zone or range of ambient temperatures around 20o C. In contrast, contagious yawning diminished when temperatures were relatively high at around 37o C in the summer of Arizona or low and around freezing in the winter of Vienna. Lead author Jorg Massen explains that where yawning functions to cool the brain, yawning is not functional when ambient temperatures are as hot as the body, and may not be necessary or may even have harmful consequences when it is freezing outside.

While most research on contagious yawning emphasizes the influence of interpersonal and emotional-cognitive variables on its expression, this report adds to accumulating research suggesting that the underlying mechanism for yawning, both spontaneous and contagious forms, is involved in regulating brain temperature. In turn, the cooling of the brain functions to improve arousal and mental efficiency. The authors of this study suggest that the spreading of this behavior via contagious yawning could therefore function to enhance overall group vigilance.

*Massen, J.J.M., Dusch, K., Eldakar, O.T. & Gallup, A.C. (2014) A thermal window for yawning in humans: Yawning as a Brain Cooling Mechanism. Physiology & Behavior. Published online on April 12th. doi: 10.1016/j.physbeh.2014.03.032.*

<http://bit.ly/1mQbO9H>

### **Are we ready for contact with extraterrestrial intelligence? *The SETI project scientists are known for tracking possible extraterrestrial signals, but now they are also considering sending messages from Earth telling of our position.***

A researcher from the University of Cádiz (Spain) questions this idea in view of the results from a survey taken by students, revealing the general level of ignorance about the cosmos and the influence of religion when tackling these

matters. The Search for ExtraTerrestrial Intelligence (SETI) project is an initiative that began in the 70s with funding from NASA, but that has evolved towards the collaboration of millions of Internet users for the processing of data from the Arecibo Observatory (Puerto Rico), where space tracking is carried out. Now the members of this controversial project are trying to go further and not only search for extraterrestrial signs, but also actively send messages from Earth (Active SETI) to detect possible extraterrestrial civilisations. Astrophysicists, such as Stephen Hawking, have already warned of the risk that this implies for humanity, since it could favour the arrival of beings with more advanced technology and dubious intentions.

The ethical and sociological implications of this proposal have been analysed by the neuro-psychologist Gabriel G. de la Torre, professor at the University of Cádiz and participant in previous projects such as Mars 500 or space psychology topical team project financed by the European Space Agency, who wonders: "Can such a decision be taken on behalf of the whole planet? What would happen if it was successful and 'someone' received our signal? Are we prepared for this type of contact?"

To answer these questions, the professor sent a questionnaire to 116 American, Italian and Spanish university students. The survey assessed their knowledge of astronomy, their level of perception of the physical environment, their opinion on the place that things occupy in the cosmos, as well as religious questions – for example, "do you believe that God created the universe?" – or on the likelihood of contact with extraterrestrials.

The results, published in the journal 'Acta Astronautica', indicate that, as a species, humanity is still not ready for trying to actively contact a supposed extraterrestrial civilisation, since people lack knowledge and preparation. For this reason, SETI researchers are recommended in this study to look for alternative strategies.

"This pilot study demonstrates that the knowledge of the general public of a certain education level about the cosmos and our place within it is still poor. Therefore, a cosmic awareness must be further promoted – where our mind is increasingly conscious of the global reality that surrounds us – using the best tool available to us: education," De la Torre emphasised. "In this respect, we need a new Galileo to lead this journey".

It was deduced from the questionnaires, which will soon be available to everyone on line, that university students and the rest of society lack awareness on many astronomical aspects, despite the enormous progress of science and technology. It also revealed that the majority of people consider these subjects

according to their religious belief and that they would rely on politicians in the event of a huge global-scale crisis having to be resolved.

"Regarding our relation with a possible intelligent extraterrestrial life, we should not rely on moral reference points of thought, since they are heavily influenced by religion. Why should some more intelligent beings be 'good'?", added the researcher, who believes that this matter should not be monopolized by a handful of scientists: "In fact, it is a global matter with a strong ethical component in which we must all participate".

*Gabriel G. De la Torre. "Toward a new cosmic consciousness: Psychoeducational aspects of contact with extraterrestrial civilizations". Acta Astronautica 94 (2): 577–583, 2014.*

<http://www.medscape.com/viewarticle/824673?src=rss#1>

## **Diabetes Rates Rocket in US Tweens and Teens**

***Type 2 diabetes in American tweens and teens skyrockets by 35% over 8-year period***  
Yael Waknine

The prevalence of type 2 diabetes in American tweens and teens has skyrocketed by 35% over an 8-year period, according to new data from the SEARCH for Diabetes in Youth study, published online May 3 in the Journal of the American Medical Association on May 3.

"Our study is really the first in the US to quantify the burden of type 2 diabetes at the population level - and not just in a clinic or group of clinics, [but] in all major racial/ethnic groups in the US - and documents increasing trends in several racial-ethnic groups," emphasized lead author Dana Dabelea, MD, PhD, from the Colorado School of Public Health, Aurora, in an interview with Medscape Medical News.

The results also show that the prevalence of pediatric type 1 diabetes increased by 30% from 2001 to 2009, which was "consistent with worldwide estimates," say the researchers.

The work involved data collected in 2001 and 2009 from 38 counties in 5 states as well as from American Indian reservations in Arizona and New Mexico and involved youths of white, black, Hispanic, Asian Pacific Islander, and American Indian descent.

Although the researchers acknowledge that the study doesn't include information from the past 5 years, they state that these increases "are serious and draw attention to the public-health impact of pediatric diabetes," noting that each new case at a young age means a lifetime burden of difficult and costly treatment, as well as an increased risk for early serious complications.

**Greatest Change in Type 2 Diabetes in Hispanic Kids**

In 2001, only 588 cases of type 2 diabetes were diagnosed among 1.7 million youth aged 10 to 19 years, for a prevalence rate of 0.34 per 1000. By 2009 the prevalence rate had risen to 0.46 per 1000, reflecting 810 cases in a population of 1.8 million. The magnitude of the increase was greatest among Hispanics (prevalence, 0.45 per 1000 in 2001 to 0.79 in 2009;  $P < .001$ ), followed by blacks (0.95 to 1.06 per 1000;  $P = .02$ ), and whites (0.14 to 0.17 per 1000;  $P < .001$ ); no significant changes were observed among Asian Pacific Islanders ( $P = .73$ ) or American Indians ( $P = .83$ ).

According to Dr. Dabelea, increasing rates of type 2 diabetes likely reflect the current obesity epidemic and also the long-term impact of higher gestational diabetes rates.

"It is likely that prevention of type 2 diabetes in youth will require sustained lifestyle changes that need to be implemented and maintained throughout the life course but starting very early in life, during (or even before) pregnancy," she said. This will include optimizing maternal weight gain and nutrition patterns during pregnancy, promotion of breastfeeding, and promotion of healthy diets and physically active behaviors at the earliest possible times, she added.

Alarm bells have been rung about type 2 diabetes in children; just last year, the TODAY series of studies indicated that some youths with type 2 diabetes have a more aggressive form of the disease than is seen adults, with a high risk for complications such as early renal and cardiovascular disease.

### **Type 1 Diabetes Also Growing Among Minorities**

With respect to type 1 diabetes, 4958 cases were diagnosed among 3.3 million youths aged 0 to 19 years in 2001, for a prevalence rate of 1.48 per 1000. By 2009, the prevalence had risen to 1.93 per 1000, reflecting 6666 new cases among 3.5 million children under the age of 19 years.

As might be expected, the greatest increase in prevalence occurred among white youth (from 1.86 per 1000 in 2001 to 2.55 in 2009;  $P < .001$ ). However, significant increases were also observed among minorities, including blacks (1.29 to 1.62 per 1000;  $P < .001$ ), Hispanics (0.96 to 1.29 per 1000;  $P < .001$ ), and Asian Pacific Islanders (0.50 to 0.60 per 1000;  $P = .006$ ); no significant changes occurred among American Indians ( $P = .19$ ).

"Historically, type 1 diabetes has been considered a disease that affects primarily white youth; however, our findings highlight the increasing burden of type 1 diabetes experienced by youth of minority racial/ethnic groups as well," the authors write.

Asked about potential causes for this increase, Dr. Dabelea said the causes of type 1 diabetes in general remain unclear. "Still, it is likely that something has

changed in the environment in which our children are born and raised - both in the US and elsewhere in the world - causing more youth to develop the disease, maybe at increasingly younger ages," she pointed out.

Speculating on potential causes, she mentioned a lack of certain viral or bacterial triggers at an early age ("hygiene hypothesis"), changes in early diet that might negatively affect the developing gut microenvironment, and increased rates of obesity in the general population. In conclusion, she and her colleagues say that further studies are required to determine the causes of these increases in both type 1 and 2 diabetes in children.

*The study was funded by the Centers for Disease Control and Prevention and the National Institute of Diabetes and Digestive and Kidney Diseases. Dr. Dabelea reports no relevant financial relationships. Disclosures for the coauthors are listed in the article. JAMA.*

2014;311:1778-1786. [Abstract](#)

<http://bit.ly/1g3tyrT>

### **Surgeons Try Cold Cutting for Critically Injured**

*A clinical trial tests whether induced hypothermia can allow surgeons to save critically wounded patients who would not survive surgery at normal temperatures. Erika Beras reports*

May 6, 2014 | By Erika Beras

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On rare occasions, a swimmer can survive a near-drowning because cold water has protected their brains—even if they were submerged for up to an hour. Now a clinical trial is testing whether extreme cold can save critically injured gunshot and knife wound patients.

It's called the Emergency Preservation and Resuscitation for Cardiac Arrest from Trauma Study. Lead Surgeon Sam Tisherman at the University of Pittsburgh Medical Center explains that patients are not declared dead, but: "They'd be close. They're in cardiac arrest and certainly if we can't get a pulse back they'd be dead. But we're not waiting to the point where the surgeons would declare them dead. We're trying to do this right before this, and find the best window of opportunity where our standard of care hasn't worked but it's not too late to try something new."

Surgeons will cool the patients' bodies to 50 degrees by pumping dozens of liters of cold saline into the heart. This induced hypothermia nearly halts all activity in the body and brain. By freezing patients, the surgeons will also freeze time—giving them the opportunity to repair wounds a warm patient wouldn't typically survive. Should the procedure prove effective, it could give a new meaning to the phrase "cold comfort."



<http://bit.ly/LiESP6l>

## Psilocybin inhibits the processing of negative emotions in the brain

### *Psilocybin influences the amygdala to weaken the processing of negative stimuli*

Emotions like fear, anger, sadness, and joy enable people to adjust to their environment and react flexibly to stress and strain and are vital for cognitive processes, physiological reactions, and social behaviour. The processing of emotions is closely linked to structures in the brain, i.e. to what is known as the limbic system. Within this system the amygdala plays a central role – above all it processes negative emotions like anxiety and fear. If the activity of the amygdala becomes unbalanced, depression and anxiety disorders may develop.

Researchers at the Psychiatric University Hospital of Zurich have now shown that psilocybin, the bioactive component in the Mexican magic mushroom, influences the amygdala, thereby weakening the processing of negative stimuli. These findings could "point the way to novel approaches to treatment" comments the lead author Rainer Krähenmann on the results which have now been published in the renowned medical journal "Biological Psychiatry".

### **Psilocybin inhibits the processing of negative emotions in the amygdala**

The processing of emotions can be impaired by various causes and elicit mental disorders. Elevated activity of the amygdala in response to stimuli leads to the neurons strengthening negative signals and weakening the processing of positive ones. This mechanism plays an important role in the development of depression and anxiety disorders. Psilocybin intervenes specifically in this mechanism as shown by Dr. Rainer Krähenmann's research team of the Neuropsychopharmacology and Brain Imaging Unit led by Prof. Dr. Franz Vollenweider.

Psilocybin positively influences mood in healthy individuals. In the brain, this substance stimulates specific docking sites for the messenger serotonin. The scientists therefore assumed that psilocybin exerts its mood-brightening effect via a change in the serotonin system in the limbic brain regions. This could, in fact, be demonstrated using functional magnetic resonance imaging (fMRI). "Even a moderate dose of psilocybin weakens the processing of negative stimuli by modifying amygdala activity in the limbic system as well as in other associated brain regions", continues Krähenmann. The study clearly shows that the modulation of amygdala activity is directly linked to the experience of heightened mood.

### Next study with depressive patients

According to Krähenmann, this observation is of major clinical importance. Depressive patients in particular react more to negative stimuli and their thoughts often revolve around negative contents. Hence, the neuropharmacologists now wish to elucidate in further studies whether psilocybin normalises the exaggerated processing of negative stimuli as seen in neuroimaging studies of depressed patients - and may consequently lead to improved mood in these patients. .

Rainer Krähenmann considers research into novel approaches to treatment very important, because current available drugs for the treatment of depression and anxiety disorders are not effective in all patients and are often associated with unwanted side effects.

Rainer Kraehenmann, Katrin H. Preller, Milan Scheidegger, Thomas Pokorny, Oliver G. Bosch, Erich Seifritz, Franz X. Vollenweider, (in press). *Psilocybin-Induced Decrease in Amygdala Reactivity Correlates with Enhanced Positive Mood in Healthy Volunteers*. *Biological Psychiatry*. <http://dx.doi.org/10.1016/j.biopsych.2014.04.010>

<http://bit.ly/SHTUFF>

### Pinocchio Rex: T's Tough Cousin (No Lie)

*Bloodthirsty T. rex had an unusual relative, nicknamed Pinocchio rex, which a new study describes as having an extremely long snout.*

May 7, 2014 05:00 AM ET // by Jennifer Viegas

According to the study, published in the journal Nature Communications, the new tyrannosaur, Qianzhousaurus sinensis, lived in southern China approximately 66 million years ago. "Although it's not definite, it's quite likely that Qianzhousaurus was around when that fireball from space so suddenly ended the age of dinosaurs," co-author Steve Brusatte of the University of Edinburgh's School of GeoSciences told Discovery News.



*Pinocchio rex, aka Qianzhousaurus sinensis, a newly discovered tyrannosaur.*  
Chuang Zhao

But before that happened, Pinocchio rex was an active predator living in a lush environment that included many different kinds of dinosaurs, lizards and other animals. With its long snout adorned with a row of horns, Pinocchio rex likely preyed on the lizards and bird-like dinosaurs known as oviraptorosaurs.

"We suspect it wasn't the top predator in its ecosystem, but maybe a second-tier predator," Brusatte said. "But just because it was second-tier doesn't mean that it wasn't a fearsome, formidable, blood-curdling predator!"

Before this discovery, paleontologists had found a few other dino specimens with unusually long heads. At first, it seemed like the stuff of fairytales that such dinosaurs actually existed. The scientists initially suspected that the fossils represented an early growth stage in dinosaurs, or could have been deformities. Pinocchio rex - found largely intact and remarkably well preserved - clarifies the matter. It's no lie: tyrannosaurs with long snouts really did exist. There appear to have been a lot of them, too.

As lead author Junchang Lü of the Institute of Geology, Chinese Academy of Geological Sciences, said: "The new discovery is very important. Along with Alioramus from Mongolia, it shows that the long-snouted tyrannosaurids were widely distributed in Asia. "Although we are only starting to learn about them, the long-snouted tyrannosaurs were apparently one of the main groups of predatory dinosaurs in Asia." These were lean, mean dinosaurs, so the researchers do not think that tyrannosaurs were more vulnerable to extinction than most other species of their time. Nevertheless, the fact remains that these big predators all went extinct around 66 million years ago.

"I suspect that the sudden environmental devastation caused by the impact was so severe that it killed off many, many species on land, and perhaps birds were the one dinosaur group able to make it through because they were smaller, could fly, and could grow fast," Brusatte explained, adding that it's still a mystery as to why so many bird-like feathered dinosaurs, like Velociraptor, did not survive. Pinocchio rex, at least, lives on today in the dinosaur record books, via its well-preserved remains.

Both Thomas Carr, an associate professor of biology at Carthage College, and Thomas Williamson, from the New Mexico Museum of Natural History and Science, are leading experts on tyrannosaurs. They told Discovery News that they believe that Pinocchio rex is an important find.

Carr said he was curious about "how the long snout evolved in the first place," and that he looked forward to more discoveries that could answer this and other questions concerning the dinosaur.

Williamson mentioned that "tyrannosaur evolution was even more complex and interesting than previously thought," especially given that tyrannosaurs with long and shorter snouts co-existed. This suggests that they must have lived and hunted differently -- somehow not eating each other to death before the asteroid hit.

<http://www.bbc.com/news/uk-england-beds-bucks-herts-27299259###rssowlmlink>

### **Placenta smoothies firm in 'health risk' case**

*A company that processes raw placentas for new mothers to eat could be shut down over health fears.*

The Independent Placenta Encapsulation Network (IPEN), based in Herts, makes smoothies and capsules that it claims have health benefits. Dacorum Borough Council prevented IPEN from trading in October last year over concerns about bacterial contamination. The case, believed to be the first of its kind, was heard before Watford magistrates and judgement reserved.

#### **'Hazard to health'**

District Judge Annabel Pilling heard that IPEN founder Lynnea Shrief, of Berkhamsted, set up the company after her own struggle to provide enough breast milk for her baby. She claims eating the raw placenta can increase breast milk supply, help reduce the impact of post-natal depression and improve general mood and a sense of well-being. IPEN had been trading for two and a half years when it was given an Emergency Prohibition Notice by the council in October.

The council's barrister Nicholas George said the bacteria staphylococcus aureus, present in the vagina of 10% of women, could be passed on to the placenta. This presented a "hazard" and a risk to health in what he described as a "significant proportion" of women. He added that the production process was out of the control of IPEN, which relied on the mother or partner to properly look after the placenta before it came to IPEN to be processed.

#### **'Trust the consumer'**

IPEN argued that the placenta should be cooled to 8 degrees Celsius within four hours of birth, is cool when they receive it and therefore presented no threat to health.

Bradley Say, for IPEN - which is challenging the prohibition notice - said the company always asked for a history of each placenta and would refuse any that had been left at room temperature for 24 hours. He said all IPEN could do was issue good advice and trust the consumer to look after their own health. "The law does not need to step in and protect people when what they are eating is a product of themselves," he told the court.

The court heard that women asked to complete feedback forms after consuming placenta products from IPEN had not reported any ill-effects.

District Judge Pilling said she was unlikely to hand down her judgement before the end of next week.

<http://bit.ly/1g0nIr3>

## **New order of marine creatures discovered among sea anemones** *Researchers build first tree of life for sea anemones, identify giant species as imposter*

A deep-water creature once thought to be one of the world's largest sea anemones, with tentacles reaching more than 6.5 feet long, actually belongs to a new order of animals. The finding is part of a new DNA-based study led by the American Museum of Natural History that presents the first tree of life for sea anemones, a group that includes more than 1,200 species. The report, which is published today in the journal PLOS ONE, reshapes scientists' understanding of the relationships among these poorly understood animals.

"The discovery of this new order of Cnidaria—a phylum that includes jellyfish, corals, sea anemones, and their relatives—is the equivalent to finding the first member of a group like primates or rodents," said Estefanía Rodríguez, an assistant curator in the Museum's Division of Invertebrate Zoology and the lead author of the new publication. "The difference is that most people are far more familiar with animals like chimpanzees and rats than they are with life on the ocean floor. But this amazing finding tells us that we have so much more to learn and discover in the ocean."

Rodríguez, along with an international team of researchers, conducted a four-year study to organize sea anemones in a "natural," or phylogenetic, way, based on their evolutionary relationships. Sea anemones are stinging polyps that spend most of their time attached to rocks on the sea floor or on coral reefs. Although they vary greatly in size and color, anemones have very few defining structures. As a result, classifying these animals based on morphology alone can be difficult. "Anemones are very simple animals," Rodríguez said. "Because of this, they are grouped together by their lack of characters—for example, the absence of a skeleton or the lack of colony-building, like you see in corals. So it wasn't a huge surprise when we began to look at their molecular data and found that the traditional classifications of anemones were wrong."

The researchers compared particular sections of DNA of more than 112 species of anemones collected from oceans around the world. Based on genetic data and the organization of their internal structures, the scientists reduced the sub-orders of anemones from four to two.

They also discovered that one of species that they analyzed is not a sea anemone at all. This animal, previously called *Bolocerooides daphneae*, was discovered in 2006 in the deep east Pacific Ocean and labeled as one of the largest sea anemones in existence. But the new study shifts it outside of the tree of life for

anemones. Instead, the researchers placed it in a newly created order—a classification equal to carnivoria in mammals or crocodilia in reptiles—under the sub-class Hexacorallia, which includes stony corals, anemones, and black corals. The new name of the animal, which lives next to hydrothermal vents, is *Relicanthus daphneae*.

*Relicanthus daphneae* is a classic example of convergent evolution, the independent evolution of similar features in species of different lineages. "Even though this animal looks very much like a sea anemone, it is not one," Rodríguez said. "Both groups of animals lack the same characters, but our research shows that while the anemones lost those characters over millions of years of evolution, *R. daphneae* never had them. Putting these animals in the same group would be like classifying worms and snakes together because neither have legs."

For now, *Relicanthus daphneae* is the only species in the new order, but researchers hope to change that. "Although we've long known about the existence of this giant animal, it's only in recent years that we're really starting to understand where it fits into the tree of life," Rodríguez said. "So imagine what else is still out there to discover."

*Other authors on the paper include Marcos Barbeitos, Universidade Federal do Paraná; Mercer Brugler, Louise Crowley, and Alejandro Grajales, American Museum of Natural History; Luciana Gusmão, Universidade de São Paulo; Verena Häussermann, Pontifícia Universidad Católica de Valparaíso; Abigail Reft, University of Heidelberg; and Meg Daly, Ohio State University.*

*This work was partially supported by the U.S. National Science Foundation under Grant No. EF-0531763 to Daly, the Lerner Gray Fund for Marine Research to Rodríguez, the Gerstner Family Foundation to Brugler, and the Chilean FONDECYT project under number 1131039 to Häussermann.*

[http://www.eurekalert.org/pub\\_releases/2014-05/p-ssi050114.php#rssowlmlink](http://www.eurekalert.org/pub_releases/2014-05/p-ssi050114.php#rssowlmlink)

## **Study suggests improved survivorship in the aftermath of the medieval Black Death**

*Skeletal analysis may support increased survival and mortality risk after Black Death*

Human mortality and survival may have improved in the generations following the Black Death, according to results published May 7, 2014, in the open access journal PLOS ONE by Sharon DeWitte from University of South Carolina.

As one of the most devastating epidemics in human history, the medieval Black Death (c. 1347-1351) killed tens of millions of Europeans.

Previous studies have shown that the disease targeted elderly adults and sick or stressed people; however, not much is known about any substantial changes in

the population, like overall health and mortality, before and after its occurrence. Following the epidemic, standards of living—particularly diet—improved, and in this study, Dr. DeWitte examined whether the deaths of frail people during the Black Death, combined with consequent rising standards of living, may have resulted in a healthier post-epidemic population in London.

Unfortunately, most available data is in historical documentation (e.g., tax records and postmortem analysis), but in this study, Dr. DeWitte sampled nearly 600 skeletons from several pre- and post-Black Death London cemeteries and then analyzed their age and modeled age estimates, mortality hazards, and birth rate data for these samples.

Post-Black Death samples had a higher proportion of older adults, suggesting that survival may have improved following the epidemic.

Additionally, results of hazards analysis indicate that overall, mortality risks were lower in the post-Black Death population than before the epidemic.

Together, these results may indicate enhanced survival and decreased mortality after the Black Death, and by inference, improved health in some age groups in the post-epidemic population.

Although other factors could have influenced these differences, like the migration of people to London after the plague, Dr. DeWitte suggests that this study highlights the power that infectious diseases may have in shaping population-wide patterns of health and demography over both the short- and long-term.

Sharon DeWitte added, "This study suggests that even in the face of major threats to health, such as repeated plague outbreaks, several generations of people who lived after the Black Death were healthier in general than people who lived before the epidemic."

*Citation: DeWitte SN (2014) Mortality Risk and Survival in the Aftermath of the Medieval Black Death. PLoS ONE 9(5): e96513. doi:10.1371/journal.pone.0096513*

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*Anthropologists (<http://www.physanth.org>). Preliminary analyses were conducted during a summer fellowship at the School for Advanced Research, sponsored by the Ethel-Jane*

*Westfeldt Bunting Foundation (<http://www.sarweb.org>). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.*

*Competing Interest Statement: The authors have declared that no competing interests exist.*

<http://bit.ly/1hGIs2j>

## AM Radio Can Disorient Birds

*Certain radio stations are bad for birds, according to a new study in Nature that finds AM radio signals disorient songbirds migrating at night.*

May 7, 2014 01:00 PM ET // by Jennifer Viegas

The study shows how even weak electromagnetic fields produced by AM radio signals can throw off a small animal — begging the question what are other electromagnetic fields doing to them, not to mention to the rest of us? Electronic pollution, or as the researchers call it, “electrosmog,” is a growing problem that is only expected to get worse as more of us plug or tune into radio stations, TVs, computers and countless other electrical devices. It’s a reminder that not all pollution is visible to us.

Biologist Henrik Mouritsen, a professor at the University of Oldenburg, and his colleagues determined that AM affects birds after studying European robins.

These birds have an internal magnetic compass that helps them to find their way even when other cues, such as visual ones, are poor. At night, the internal magnetic compass is key to the birds finding their way. Mouritsen and his team housed the birds in windowless huts. The researchers figured out a way to easily turn on and off the birds’ exposure to background urban electromagnetic signals. With the electrosmog, the birds could not orient themselves properly. Without it, they were good to go.

The researchers further determined that only certain electromagnetic noise bothered the birds. It was in the frequency range of two to five kilohertz megahertz, putting it right in the range of AM radio signals.

“These perturbations do not originate from power lines or mobile networks,” Mouritsen said.

He added that the identified magnetic component is much weaker than the lower exposure limits currently recommended by guidelines adopted by the World Health Organization.

What then could the implications be for humans?

In an accompanying “News & Views” piece in the same journal, Joseph Kirschvink mulled over that very question. Kirschvink is in the Division of Geological and Planetary Sciences at the California Institute of Technology.

He’s also a researcher at the Tokyo Institute of Technology.

Kirschvink notes that human tissues contain biological magnetite, which has geomagnetic sensitivity.

“Many people claim to be bothered by radio transmissions, and some have even moved to live in radio frequency ‘quiet zones’ around radio telescopes,” he

wrote. "Modern-day charlatans will undoubtedly seize on this study as an argument for banning the use of mobile phones, despite the different frequency bands involved."

"However," he continued, "if the effect reported by the authors stands the acid test of reproducibility, we might consider gradually abandoning our use of this portion of the electromagnetic spectrum and implementing engineering approaches to minimize incidental low-frequency noise, to help migratory birds find their way."

In other words, don't buy stock in AM radio anytime soon. Like the non-avian dinosaur relatives of birds, it'll probably go extinct before long.

<http://phys.org/news/2014-05-kelp-ocean-borne-fukushima.html#rssowlmlink>

### **Kelp study finds no ocean-borne Fukushima radiation**

*West Coast shoreline shows no signs of ocean-borne radiation from Japan's Fukushima nuclear power plant disaster*

Phys.org - Scientists working together on Kelp Watch 2014 announced today that the West Coast shoreline shows no signs of ocean-borne radiation from Japan's Fukushima nuclear power plant disaster, following their analysis of the first collection of kelp samples along the western U.S. coastline.

Kelp Watch 2014 is a project that uses coastal kelp beds as detectors of radioactive seawater arriving from Fukushima via the North Pacific Current. It is a collaborative effort led by Steven Manley, marine biology professor at California State University, Long Beach (CSULB), and Kai Vetter, head of applied nuclear physics at the Lawrence Berkeley National Laboratory (Berkeley Lab) and a nuclear engineering professor at the University of California, Berkeley.

The new results are from samples primarily collected from Feb. 24 through March 14.

During the first phase of the project, samples were taken from 38 of the 44 sites originally identified, and the data being presented comes from an analysis of 28 of the 38 sample sites represented.

"Our data does not show the presence of Fukushima radioisotopes in West Coast Giant Kelp or Bull Kelp," Manley said. "These results should reassure the public that our coastline is safe, and that we are monitoring it for these materials. At the same time, these results provide us with a baseline for which we can compare samples gathered later in the year."

### **Kelp study finds no ocean-borne Fukushima radiation**

The samples analyzed to date were gathered from as far north as Kodiak Island, Alaska, to as far south as Baja California. Two sites in the tropics—Hawaii and

Guam, where non-kelp brown algae were sampled (kelps are not found in the tropics)—were also negative for Fukushima radiation.

"The samples of greatest concern were those from the north, Alaska to Washington State, where it is thought the radioactive water will first make contact with North America," Manley continued. "The tell-tale isotopic signature of Fukushima, Cs-134, was not seen, even at the incredibly low detection limits provided by Dr. Vetter's group at the Berkeley Lab and UC Berkeley."

Vetter added, "We will also publish results of naturally occurring radiation sources, such as those associated with the decay of uranium and thorium, to help provide context to our findings on radioisotopes Cs-134 and Cs-137 from Fukushima."

Although initiated as a California-centric endeavor with 30 sites, Kelp Watch 2014 has steadily grown to include many sites along the west coast of North America and beyond. Manley noted that the project also has Giant Kelp from Chile in South America that will serve as a reference site, far removed from any potential influence from Fukushima.

Information about the procedures and results, including the results of the first samples' analyses, are available to the public at [kelpwatch.berkeley.edu](http://kelpwatch.berkeley.edu). The researchers will continually update the website for public viewing as more samples arrive and are analyzed, including samples from Canada.

"Because the Pacific Northwest may be ground zero for its arrival, we will be receiving monthly samples from the west and southern coastline of Vancouver Island (Canada)," Manley explained. "One of the goals of Kelp Watch 2014 is to keep the public informed, to let them know we are on top of this event, and to document the amount of Fukushima radiation that enters our kelp forest ecosystem." The second of the three 2014 sampling periods is scheduled to begin in early July.

<http://bit.ly/1gcZ1wx>

### **Study confirms mitochondrial deficits in children with autism** *Children with autism experience deficits in a type of immune cell that protects the body from infection.*

Sacramento, Calif. - Called granulocytes, the cells exhibit one-third the capacity to fight infection and protect the body from invasion compared with the same cells in children who are developing normally. The cells, which circulate in the bloodstream, are less able to deliver crucial infection-fighting oxidative responses to combat invading pathogens because of dysfunction in their tiny energy-generating organelles, the mitochondria. The study is published online in the journal *Pediatrics*.

"Granulocytes fight cellular invaders like bacteria and viruses by producing highly reactive oxidants, toxic chemicals that kill microorganisms. Our findings show that in children with severe autism the level of that response was both lower and slower," said Eleonora Napoli, lead study author and project scientist in the Department of Molecular Biosciences in the UC Davis School of Veterinary Medicine. "The granulocytes generated less highly reactive oxidants and took longer to produce them."

The researchers also found that the mitochondria in the granulocytes of children with autism consumed far less oxygen than those of the typically developing children — another sign of decreased mitochondrial function.

Mitochondria are the main intracellular source of oxygen free radicals, which are very reactive and can harm cellular structures and DNA. Cells can repair typical levels of oxidative damage. However, in the children with autism the cells produced more free radicals and were less able to repair the damage, and as a result experienced more oxidative stress. The free radical levels in the blood cells of children with autism were 1 ½ times greater than those without the disorder.

The study was conducted using blood samples of children enrolled in the Childhood Risk of Autism and the Environment (CHARGE) Study and included 10 children with severe autism age 2 to 5 and 10 age-, race- and sex-matched children who were developing typically.

In an earlier study the research team found decreased mitochondrial fortitude in another type of immune cell, the lymphocytes. Together, the findings suggest that deficiencies in the cells' ability to fuel brain neurons might lead to some of the cognitive impairments associated with autism. Higher levels of free radicals also might contribute to autism severity.

"The response found among granulocytes mirrors earlier results obtained with lymphocytes from children with severe autism, underscoring the cross-talk between energy metabolism and response to oxidative damage," said Cecilia Giulivi, professor in the Department of Molecular Biosciences in the UC Davis School of Veterinary Medicine and the study's senior author. "It also suggests that the immune response seems to be modulated by a nuclear factor named NRF2," that controls antioxidant response to environmental factors and may hold clues to the gene-environment interaction in autism, Giulivi said.

*Other study authors include Sarah Wong and Irva Hertz-Picciotto of UC Davis.*

*The study was funded by grants from the Simons Foundation Autism Research Initiative (SFARI), and National Institutes of Environmental Health Sciences grants ES011269, ES015359 and ES020392.*

<http://bit.ly/119MOjm>

## **Recycling a patient's lost blood during surgery better than using banked blood**

***Patients whose own red blood cells are recycled during heart surgery have blood cells better able to carry oxygen compared to those who get transfusions***

Patients whose own red blood cells are recycled and given back to them during heart surgery have healthier blood cells better able to carry oxygen where it is most needed compared to those who get transfusions of blood stored in a blood bank, according to results of a small study at Johns Hopkins.

In a report for the June issue of the journal *Anesthesia & Analgesia*, the researchers say they found that the more units of banked blood a patient received, the more red cell damage they observed. The damage renders the cells less flexible and less able to squeeze through a body's smallest capillaries and deliver oxygen to tissues. Among patients who received five or more units of red blood cells from a hospital blood bank during the study, the damage persisted for at least three days after surgery. In the past, studies have linked transfusions to increased risk of hospital-acquired infections, longer hospital stays and increased risk of death.

"We now have more evidence that fresh blood cells are of a higher quality than what comes from a blood bank," says study leader Steven Frank, M.D., an associate professor of anesthesiology and critical care medicine at the Johns Hopkins University School of Medicine. "If banked blood, which is stored for up to six weeks, is now shown to be of a lower quality, it makes more sense to use recycled blood that has only been outside the body for one or two hours. It's always been the case that patients feel better about getting their own blood, and recycling is also more cost effective."

To recycle the blood, a machine known as a cell saver is used to collect what a patient loses during surgery, rinse away unneeded fat and tissue, and then centrifuge and separate the red cells, which are then returned to the patient should he or she need it. Disposable parts of the cell saver, which can be used to process multiple units of blood, cost around \$120, compared to \$240 for each unit of banked blood.

Such recycling first became popular during the early years of the HIV/AIDS crisis, so patients could avoid the risk of getting the virus in transfused blood. Today, Frank says, the blood supply is much safer, with the incidence of contracting HIV from a transfusion down from one in 100 in the early 1980s to one in 2 million now — but focus should be on recycling, because fresher blood

is better. The practice also reduces the risk of contracting hepatitis B or C infections, or of bad transfusion-related reactions, Frank adds.

For the study, the Johns Hopkins researchers categorized 32 cardiac surgery patients by their transfusion status: those who received only their own recycled red blood cells (12 patients), those who received their own blood plus fewer than five units of banked blood (10) and those who received their own blood plus five or more units of stored blood (10). All had blood samples drawn before, during and for three days after surgery. The samples were examined for blood cell membrane stiffness and flexibility, a measure of how well oxygen is likely to get to where it is needed.

In patients who received only their own recycled blood, their cells behaved normally right away, as if they had never been outside the body. The more blood a patient got from the bank, the less flexible their entire population of red blood cells. Three days after surgery, the red blood cells in the group that got the largest number of transfused units still had not recovered their full function. "If something is bad for you, a little bit might be OK, but a lot of it is much worse," Frank says. "It turns out that blood is more like milk, which has a relatively short shelf life, than a fine wine, which gets better with age."

Frank cautions that cell saver machines are not appropriate for all operations, and not all hospitals have access to round-the-clock perfusionists to run them. For heart surgeries, however, a perfusionist is already in the operating room to run the heart-lung bypass machine. And, he adds, many operations are considered to be low risk for blood loss, in which case the cell saver is unnecessary. But he advocates wider use of recycled blood.

"In any patient where you expect to give one unit of red blood cells or more, it's cost-effective and beneficial to recycle," he says. Patients who lose blood may also need platelets and plasma, which they receive regardless of whether they receive their own blood or blood from a bank.

Frank is medical director of the Johns Hopkins Center for Bloodless Medicine and Surgery, which primarily serves Jehovah's Witnesses, who do not accept blood transfusions but will accept recycled cell saver blood.

By using the cell saver as a primary method of blood conservation, their efforts have allowed these patients to do just as well or better than patients who get transfusions of donated blood, he says. Preliminary findings suggest patients who avoid banked blood develop fewer hospital-acquired infections.

*Other Johns Hopkins researchers who contributed to this study include Osman N. Salaria, M.D.; Viachaslau M. Barodka, M.D.; Charles W. Hogue Jr., M.D.; Dan E. Berkowitz, M.D.; Paul M. Ness, M.D.; and Jack O. Wasey, M.D.*

*The research was supported by grants from the National Institutes of Health's National Heart, Lung and Blood Institute (R01 HL105296-03 and R01 HL1092259-01) and the New York Community Trust.*

*Frank has received funding from Haemonetics Corporation to do future research and is also a paid consultant to Haemonetics Corporation. This arrangement has been reviewed and approved by the Johns Hopkins University in accordance with its conflict of interest policies.*

<http://bit.ly/1sCJPG9>

### **Anti-aging factor offers brain boost too**

***A variant of the gene KLOTHO is known for its anti-aging effects in people fortunate enough to carry one copy.***

Now researchers find that it also has benefits when it comes to brain function. The variant appears to lend beneficial cognitive effects by increasing overall levels of klotho in the bloodstream and brain.

What's more, the improvements in learning and memory associated with klotho elevation aren't strictly tied to aging. They do occur in aging mice, but also in young animals, according to a report published in the Cell Press journal Cell Reports on May 8th. That means klotho works to enhance brain power, but in an unexpected way.

"Based on what was known about klotho, we expected it to affect the brain by changing the aging process," said Lennart Mucke of the Gladstone Institute and the University of California, San Francisco, who directed the study. "But this is not what we found, which suggests to us that we are on to something new and different."

Aging is a primary risk factor for cognitive decline, lead author Dena Dubal explained. The question was: Would a factor known to play a role in long life have benefits for cognition too?

Together with a large group of collaborators, Mucke and Dubal examined the question in three separate cohorts of people participating in aging studies of various kinds, adding up to more than 700 people. Their analysis showed that people with one of the life-extending variants of the KLOTHO gene scored better on cognitive tests. Because those effects were associated with higher circulating levels of klotho, the researchers turned to genetically engineered mice that express higher-than-normal levels of the life-extending substance. Indeed, klotho worked there too. "Mice with elevated klotho performed twice as well as controls in some cognitive tests – such as remembering where a hidden platform was located in a water maze," Dubal said. In other tests, the mice did better too, but in some cases only slightly.

Elevating *klotho* in mice also enhanced the formation and flexibility of neural connections, the cellular basis for learning and memory. Surprisingly, the effects of *klotho* were evident in mice young and old. They didn't correlate with age in humans, either.

In other words, *klotho* appears to work in a manner independent of aging and may increase cognitive reserve at different life stages. The researchers say that in healthy, aging humans the positive cognitive effects of carrying one copy of the *KLOTHO* variant may even exceed the harmful effect of carrying the notorious  $\epsilon 4$  variant of the *APOE* gene, best known for its contributions to Alzheimer's disease.

Mucke says that means the findings could have broad therapeutic implications. "Because cognition is a highly valued aspect of brain function that diminishes with aging and disease, the potential to enhance it even slightly is of great potential relevance to the human condition," Dubal said.

*Cell Reports, Dubal et al.: "Life extension factor klotho enhances cognition."*

<http://bit.ly/1oH4jv4>

### What doesn't kill you may make you live longer

#### *McGill research finds unexpected link between cell suicide and longevity*

What is the secret to aging more slowly and living longer? Not antioxidants, apparently.

Many people believe that free radicals, the sometimes-toxic molecules produced by our bodies as we process oxygen, are the culprit behind aging. Yet a number of studies in recent years have produced evidence that the opposite may be true. Now, researchers at McGill University have taken this finding a step further by showing how free radicals promote longevity in an experimental model organism, the roundworm *C. elegans*. Surprisingly, the team discovered that free radicals – also known as oxidants – act on a molecular mechanism that, in other circumstances, tells a cell to kill itself.

Programmed cell death, or apoptosis, is a process by which damaged cells commit suicide in a variety of situations: to avoid becoming cancerous, to avoid inducing auto-immune disease, or to kill off viruses that have invaded the cell. The main molecular mechanism by which this happens is well conserved in all animals, but was first discovered in *C. elegans* – a discovery that resulted in a Nobel Prize.

The McGill researchers found that this same mechanism, when stimulated in the right way by free radicals, actually reinforces the cell's defenses and increases its lifespan. Their findings are reported in a study published online May 8 in the journal *Cell*.

"People believe that free radicals are damaging and cause aging, but the so-called 'free radical theory of aging' is incorrect," says Siegfried Hekimi, a professor in McGill's Department of Biology and senior author of the study. "We have turned this theory on its head by proving that free radical production increases during aging because free radicals actually combat – not cause – aging. In fact, in our model organism we can elevate free radical generation and thus induce a substantially longer life."

The findings have important implications. "Showing the actual molecular mechanisms by which free radicals can have a pro-longevity effect provides strong new evidence of their beneficial effects as signaling molecules", Hekimi says. "It also means that apoptosis signaling can be used to stimulate mechanisms that slow down aging".

"Since the mechanism of apoptosis has been extensively studied in people, because of its medical importance in immunity and in cancer, a lot of pharmacological tools already exist to manipulate apoptotic signaling. But that doesn't mean it will be easy."

Stimulating pro-longevity apoptotic signaling could be particularly important in neurodegenerative diseases, says Hekimi. In the brain the apoptotic signaling might be particularly tilted toward increasing the stress resistance of damaged cells rather than killing them, explains Hekimi. That's because it is harder to replace dead neurons than other kinds of cells, partly because of the complexity of the connections between neurons.

*"The Intrinsic Apoptosis Pathway Mediates the Pro-Longevity Response to Mitochondrial ROS in C. elegans", Callista Yee, Wen Yang, and Siegfried Hekimi, Cell, May 8, 2014*  
<http://dx.doi.org/10.1016/j.cell.2014.02.055>

<http://bit.ly/SLx1RF>

### Handshake Strength Reveals Education, Age

*A handshake can tell a lot about a person. Researchers say the strength of someone's grip may reveal how fast they're aging, their education level - and even their future health.*

The International Institute for Applied Systems Analysis researchers reviewed more than 50 published studies from around the world for their article in the journal [PLOS ONE](#). They found that people with more education at age 69 tended to grip just as strongly as less educated people at age 65, suggesting the latter were aging about four years faster. "According to hand grip strength, people with high education... feel several years younger compared to people with lower education," says study co-author Sergei Scherbov.



Another study the authors reviewed was done on more than a million Swedish adolescent males, whose handgrip strength was measured as part of an exam for military service. Those with lower handgrip strength were significantly more likely to die earlier, have heart disease, be at higher risk of suicide and experience psychological problems.

### [A Formula for the Perfect Handshake?](#)

Differences in health that correlated with handgrip strength could be seen across many of the studies, the researchers write. "Low handgrip strength has been shown definitively to predict poor outcomes in a wide variety of mortality, morbidity, and other health outcomes," according to the study. However, any educational and racial differences that could be parsed by handgrip tended to disappear as people reached their 90s.

The study was funded by the European Research Council as part of a grant to define new measures of aging based on people's characteristics, such as their longevity, health, disability status and other demographic factors.

<http://www.bbc.com/news/health-27239241###rssowlmlink>

### **The drive for 'natural motherhood'**

*The very personal issue of how a woman gives birth and feeds her baby is subject to much public debate.*

**By Alison Phipps Director of gender studies, Sussex University**

In this week's Scrubbing Up, Alison Phipps, director of gender studies at Sussex University, suggests women are coming under undue pressure to conform to "natural motherhood", deemed the "ultimate feminine achievement".

This April, Brazilian authorities ordered 29-year-old Adelir Carmen Lemos de Goés to have a Caesarean section because her baby was breech. She was taken to hospital - under a court order - by police officers and had the operation against her wishes. But when the case reached Western media, it seemed that the C-section itself was the horror, not the fact that it was forced. Headlines such as 'Inside a War on Natural Birth', preoccupations with the health of the foetus (as if delivering a healthy baby surgically was the real crime), and analyses of global Caesarean rates all betrayed the status of 'natural birth' as paragon of motherhood.

In the 1980s, the World Health Organization (WHO) defined a "normal birth" as one low in intervention, stating that women with "low risk" pregnancies should not be unnecessarily interfered with.

By the 2000s, this term had become common in the UK and elsewhere. However, during that time protecting women from medicalisation had become pressuring

them to give birth "naturally" whether they wanted to or not. Outcomes were being measured and targets were being set.

### **Empowered?**

Today, the idea of "normal birth" unites a coalition of governmental and professional bodies, non-governmental organisations, community groups and activists. It's the apex of "natural motherhood", a package which also includes a glowing, uncomplicated pregnancy and exclusive, extended breastfeeding.

This is the ultimate feminine achievement - women are empowered, we are told, by experiencing labour pains and feeding at the breast.

This needs to be set in context. In the 1970s, feminists fought against the medicalisation of motherhood, arguing that women had been disempowered by the establishment. They were right.

However, the pendulum has now swung back again, with women who don't achieve the new "natural" ideals experiencing depression and feelings of failure.

### **Caricatures**

There are good reasons why "natural motherhood" is hard. A lot of them are structural - maternity services are stretched and extra support costs money, whether it's a doula or hypno-birthing classes.

Women who don't get much maternity leave often can't breastfeed for long.

There are also a host of medical, psychological and practical reasons why birth interventions or infant formula might be needed.

However, these are often ignored - instead, women who eschew the "natural" are caricatured as immature, ignorant and selfish.

Last year, socially excluded mothers in South Yorkshire and Derbyshire were offered £200 in shop vouchers to breastfeed for six weeks. Nobody explained how this was going to help babies with tongue-tie to latch on, or persuade bosses to offer pumping facilities.

### **A lack of support**

In our culture - and health service - we revere personal choice. Except, it seems, when it comes to women's bodies.

In 2011, an outcry followed guidance issued by the National Institute for health and Care Excellence (NICE) which stated that the final decision about whether to have an elective Caesarean section should be the mother's.

Research shows that birth trauma is more often about lack of support than the mode of delivery.

In 2011, a UK Birth Trauma Association survey revealed that many labouring women had been denied the pain relief they requested. In 2012, Amanda

Braithwaite lost her overdue baby girl, something the family believe was due to Amanda being refused a C-section by doctors who had assessed her as 'low risk'. Is her story any less harrowing than that of Adelir Carmen Lemos de Goés? The experiences of Adelir Carmen Lemos de Goés and Amanda Braithwaite are horrendous, but even away from these extremes, surely delivering "naturally" or surgically under duress are as bad as each other. Nowadays, a "natural" birth is the only "right" one. But right for whom? If it's not a choice, then "natural motherhood" isn't about women's empowerment. And if it makes a woman miserable, then it's probably not best for her baby either.

<http://nyti.ms/1uN8FoJ>

### **Deadly Illness in Nicaragua Baffles Experts**

*A painful disease that affects the kidneys has killed at least 20,000 people over the past decade and has become the leading cause of deaths in hospitals among men in El Salvador*

By HEATHER MURPHY MAY 8, 2014

Chichigalpa, Nicaragua - During the harvest season, when exhausted workers spend seven days a week cutting sugar cane, the signs of illness were hard to spot at first.

It was in the off-season, out on the baseball field, that some residents noticed a change. Base-stealers were lethargic. Pitchers were losing their aim. In the evening, outfielders were burning up as if standing under the scorching sun of the day.

"That's Mosquito, now dead," said Arnulfo Téllez Aguilera, 49, pointing to a photograph of his smiling teammates before their muscles withered, like his.

"That's my brother, Danilo, dead too."

Across Central America, a painful disease that affects the kidneys has killed at least 20,000 people over the past decade and has become the leading cause of deaths in hospitals among men in El Salvador. But the illness, often called Chronic Kidney Disease of unknown causes, or CKDu, is so poorly understood that it still does not have a universally agreed upon name.

Theories vary drastically, citing a combination of possible factors, including heat stress, chronic dehydration, toxic chemicals, painkillers, sugar consumption and even volcanic ash.

But there is a rare point of consensus, many researchers say: Nicaragua's sugar cane heartland — in particular Chichigalpa, the town that is home to the country's largest sugar mill — has been one of the hardest hit places in the world.

Cane-cutting fathers and sons in the same family have died, and seemingly healthy young men are quickly wasting away.

The Nicaraguan government, the country's sugar mills, even the World Bank, which has poured tens of millions of dollars into the sugar industry here, all say that until the mystery of the disease is solved, there is little they can do to prevent it. Now, after years of inconclusive research, the Centers for Disease Control and Prevention is stepping in to help with some of the most ambitious studies of the illness yet.

But the sick former sugar cane workers here have little faith that more studies will bring improvements anytime soon. The fact that the research will be funded entirely by the sugar industry is only fueling the distrust.

"I don't think anybody has clean hands," said Kristen Genovese, a lawyer who helped sick former workers file a complaint against the World Bank's lending arm in 2008 for lending \$55 million to the sugar mill here, called San Antonio, without looking into the disease or the possibility that it might be connected to the industry.

"The government of Nicaragua has done nothing to help these people," said Ms. Genovese, who was with the Center for International Environmental Law in Washington when she filed the complaint. And the World Bank, she argued, "should have spotted this problem, and didn't do anything about it, and continued to invest in sugar in the region."

Along the Island of the Widows, as one area next to the big sugar mill is called, the disease is such a dominant part of life that health updates sometimes replace "hello."

Before each harvest, workers must take blood or urine tests that measure kidney function to determine whether they will be allowed to return to the fields. In preparation, some ingest concoctions of fresh tamarind juice and linseed oil, avoid the sun and force themselves to rest. Others simply pray.

"When you're sick they tell you, 'You're done here,'" said Mr. Aguilera, who failed the test in 2001. "They washed us away, without any kind of helping hand." The complete dependence on the industry, whatever the risks may be, is stark. Glassy-eyed men are convinced that something in the water or the fields made them sick. Yet they are desperate to return to work, borrowing their wives' and sisters' identity numbers in a furtive attempt to stay employed. In one neighborhood, even conservative studies show that one in three men have the illness.

"The sugar mill said that all of this is part of nature. But this isn't nature," said Gilma Urbina, 37, a mother of five who recently lost her husband to the disease.

“The day that my husband died, he died at 6 in the morning,” Ms. Urbina said. “Down the block, another one died at 2. The next day, three more died.” She added, “I think we’re going to end up without any people here.”

Radically different perceptions of the cause have stirred debate over who should cover the enormous costs of treating patients with the disease.

Mario Amador, general manager of Nicaragua’s National Committee of Sugar Producers, one of the groups financing the C.D.C. studies, said the sugar cane business in Nicaragua had quadrupled over the past 10 years into a \$500-million-a-year industry, supplying everything from Coca-Cola to rum makers. But he said the annual kidney exams, which he called necessary to avoid putting sick workers at additional risk, had created the false impression that the mills were linked to the disease.

“I don’t think there’s any relationship between CKD and the sugar cane industry,” Mr. Amador said.

Executives at the San Antonio mill in Chichigalpa say they also take the annual test, but it is rare that they fail.

“We drink the water here, too,” said Álvaro Bermúdez Castillo, the mill’s administrative director, who has been working at the mill since the 1970s.

Lawmakers in El Salvador and Sri Lanka, where similar kidney problems have emerged, have moved to ban certain herbicides. But few of the researchers who are focusing on Nicaragua — the country with the highest death rate from the disease, according to the Pan American Health Organization — are willing to say the answer is that simple.

Agricultural chemicals alone, they say, do not explain why the disease has been detected in some Nicaraguan miners at similar rates, why women who grew up on the sugar mill’s grounds have generally been unscathed, or why workers here are affected at much higher rates than people exposed to the same chemicals elsewhere. Perhaps extreme heat, dehydration, the intensity of the labor or other factors play a role, the researchers say.

“The problem is, this is a silent killer,” said Aurora Aragón, a Nicaraguan researcher.

Residents say they began noticing the sickness shortly after the Nicaraguan government, which had nationalized the sugar industry, returned the mills to private owners in 1992. As the operations at San Antonio rapidly expanded, driven partly by American and European appetites for sugar and a move into ethanol production, families say the cane cutters — many of whom had been born in a hospital on the mill grounds and went to a school there — grew sick in larger numbers.

The mill says it pays the government every year so that workers can receive pensions and health insurance. Without a scientifically decisive link to the disease, industry officials question why they should be responsible for more. Throughout the early 2000s, lawsuits seeking compensation from the mill mostly went nowhere. Then in 2008, a workers’ group known as the Association of Chichigalpinos for Life filed the complaint against the World Bank’s lending arm, known as the International Finance Corporation, which had lent the mill \$55 million. The complaint alleged that by failing to acknowledge the disease in documentation about the project, it had violated its own lending standards. When deciding whether to invest in the industry, no one had brought up or looked into the disease, a spokesman for the finance corporation said.

“CKD is not a common disease in the sugar sector worldwide, so I.F.C. did not look into this issue at appraisal,” said the spokesman, Aaron Rosenberg, based in Washington. The institution has lent more than \$100 million to Nicaraguan sugar mills over the past decade.

After the complaint, the mill agreed to provide food and other assistance to widows and sick workers. It also agreed to open its door to a team of outside researchers.

Yet “nature can be very reluctant to give up its secrets,” said Daniel Brooks, a researcher from Boston University, which was selected to investigate the disease by a committee that included mill executives and sick workers.

Five years later, Dr. Brooks has as many questions about the disease as answers. His report published in 2012 neither entirely ruled out nor formally endorsed any theory.

Now, Dr. Brooks and his team from Boston University will be leading the charge in three C.D.C. Foundation studies, which he believes could have implications far beyond Nicaragua.

“We don’t know if this is the tip of the iceberg yet,” he said. “If this is heat stress and the climate is changing in the direction of getting hotter over time, are we seeing something that will happen much more in the future and maybe extend its geographic range?”

“Similarly, if it’s agrochemicals, the agrochemicals they use are very widespread across the whole world,” he added.

For now his team is more focused on two other areas: whether the disease has a genetic component and its presence in children. Research into work-related risk factors is also planned, but sugar industry funding so far covers only preliminary work. The C.D.C. says that numerous measures are in place to protect the research from undue influence. Some others are suspicious.

“If your main concern is limiting liability, you are never going to get to the bottom of what is causing this,” said Jason Glaser, president of La Isla Foundation, an organization focused on the disease in Nicaragua.

On a recent afternoon, Mr. Aguilera was feeling well enough to take careful steps toward the graves of his two brothers and father, all victims of the disease. He was thrown off by all the new mounds of earth that had appeared — at least 30 over the past few months.

“I will be buried over there,” he said calmly, pointing to a plot nearby. He died three weeks later.

<http://www.bbc.com/news/health-27323472###rsslmlink>

### **Multiple sclerosis discovery may explain gender gap**

*A key difference in the brains of male and female MS patients may explain why more women than men get the disease, a study suggests.*

By Pippa Stephens Health reporter, BBC News

Scientists at Washington University School of Medicine in the US found higher levels of protein S1PR2 in tests on the brains of female mice and dead women with MS than in male equivalents. Four times more women than men are currently diagnosed with MS. Experts said the finding was "really interesting". MS affects the nerves in the brain and spinal cord, which causes problems with muscle movement, balance and vision. It is a major cause of disability, and affects about 100,000 people in the UK.

#### **Blood-brain barrier**

Abnormal immune cells attack nerve cells in the central nervous system in MS patients. There is currently no cure, although there are treatments that can help in the early stages of the disease.

Researchers in Missouri looked at relapsing remitting MS, where people have distinct attacks of symptoms that then fade away either partially or completely. About 85% of people with MS are diagnosed with this type.

Scientists studied the blood vessels and brains of healthy mice, mice with MS, and mice without the gene for S1PR2, a blood vessel receptor protein, to see how it affected MS severity. They also looked at the brain tissue samples of 20 people after they had died. They found high levels of S1PR2 in the areas of the brain typically damaged by MS in both mice and people.

The activity of the gene coding for S1PR2 was positively correlated with the severity of the disease in mice, the study said.

Scientists said S1PR2 could work by helping to make the blood-brain barrier, in charge of stopping potentially harmful substances from entering the brain and

spinal fluid, more permeable. A more permeable barrier could let attacking cells, which cause MS, into the central nervous system, the study said.

#### **Understanding 'crucial'**

Prof Robyn Klein, of the Washington University School of Medicine, said: "We were very excited to find the molecule, as we wanted to find a target for treatment that didn't involve targeting the immune cells. "This link [between MS and S1PR2] is completely new - it has never been found before."

Prof Klein said she did not know why the levels of S1PR2 were higher in women with MS, adding she had found oestrogen had "no acute role".

She would be looking at taking her findings to clinical trials in the "next few years", she added.

Dr Emma Gray, of the MS Society, said: "We don't yet fully understand why MS affects more women than men, and it's an area that's intrigued scientists, and people with MS, for many years. "A number of theories have been suggested in the past, including the influence of hormones or possible genetic factors - and this study explores one such genetic factor in further detail, which is really interesting." She said understanding the causes of MS was a "priority" for the MS Society in the UK, and could be "crucial" in finding new treatments.

The research was published in the Journal of Clinical Investigation.

<http://bit.ly/1ld3mKl>

### **Did an impact knock the Moon on its side?**

*Anomalies suggest the far side and pole weren't always where they are today.*

by John Timmer - May 9 2014, 8:00pm TST

We tend to think of the Moon as a static, dead world, with no atmosphere and no plate tectonics. But there are various signs the Moon has been active—volcanoes and indications of a magnetic field frozen in rocks. Impact craters that flooded with molten rock are also indications of more active periods in the Moon's history. Now, some researchers are suggesting that the residual magnetic fields contain hints that the Moon was once flipped on its side by a violent event.

All evidence indicates that the Moon was formed when a Mars-sized body collided with the early Earth, leaving both in a molten state. This would have left the Moon with a sufficiently molten core that it should have generated a magnetic field for hundreds of millions of years. Remnants of that field should remain trapped in rocks that solidified while it was still in place and remain trapped there to this day.

A team of Japanese researchers has now analyzed magnetic data from two lunar orbiters, the Lunar Prospector and Kaguya. Both orbited the Moon at low altitudes (under 40km) and tracked the local magnetic fields. After eliminating a

variety of areas with complex magnetic anomalies, the team looked at data from 57 different sites on the Moon and used the readings to calculate the orientation of the Moon's magnetic field at various points in its past.

Many of the data points clustered at the current pole. But a second set clustered well away from there, somewhere between 45 and 60 degrees from the existing pole. Although the Earth has experienced some degree of polar wander, the pole has always made a gradual track as the Earth's angular momentum shifted. Here, it appears that the Moon made a sudden jump, as there are no indications of gradual track between these two locations.

As the authors note, "A change in the apparent pole position corresponds to a reorientation of the lunar surface with respect to the rotation axis." And this reorientation appeared to occur relatively suddenly. The authors suggest a number of events could have been the cause, including giant impacts, internal instabilities, and the gravitational disturbances caused by migrations of the Solar System's gas giants.

This isn't the first indication that the Moon may have shifted its orientation. An earlier work examined the distribution of craters on its surface, which should be biased toward a greater number on the far side. Instead, some researchers have suggested the near and far side of the Moon swapped places at some point in the distant past.

Neither of the methods of tracking this shift have been precise enough to indicate when this event took place, which might allow us to associate it with some of the Moon's larger impact basins. But there certainly seems to be enough evidence of this sort of shift to make the idea worth exploring further.

*Nature Geoscience*, 2014. DOI: 10.1038/NGEO2150 (About DOIs).

<http://bit.ly/1sCK3wX>

## Astronomers find Sun's 'long-lost brother,' pave way for family reunion

*A team of researchers led by astronomer Ivan Ramirez of The University of Texas at Austin has identified the first "sibling" of the sun—a star almost certainly born from the same cloud of gas and dust as our star.*

Phys.org - Ramirez's methods will help astronomers find other solar siblings, which could lead to an understanding of how and where our sun formed, and how our solar system became hospitable for life. The work appears in the June 1 issue of *The Astrophysical Journal*.

"We want to know where we were born," Ramirez said. "If we can figure out in what part of the galaxy the sun formed, we can constrain conditions on the early solar system. That could help us understand why we are here."

Additionally, there is a chance, "small, but not zero," Ramirez said, that these solar sibling stars could host planets that harbor life.

In their earliest days within their birth cluster, he explains, collisions could have knocked chunks off of planets, and these fragments could have traveled between solar systems, and perhaps

even may have been responsible for bringing primitive life to Earth. "So it could be argued that solar siblings are key candidates in the search for extraterrestrial life,"

Ramirez said.

The solar sibling his team identified is called HD 162826, a star 15 percent more massive than the sun, located 110 light-years away in the constellation Hercules.

The star is not visible to the unaided eye but easily can be seen with low-power

binoculars, not far from the bright star Vega.

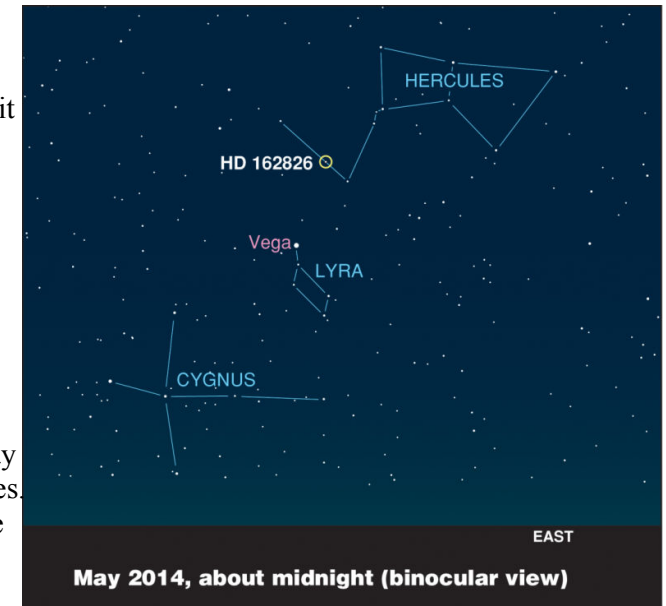
***Solar sibling HD 162826 is not visible to the unaided eye, but can be seen with low-power binoculars near the bright star Vega in the night sky. Credit: Ivan Ramirez/Tim Jones/McDonald Observatory***

The team identified HD 162826 as our sun's sibling by following up on 30 possible candidates found by several groups around the world looking for solar siblings.

Ramirez's team studied 23 of these stars in depth with the Harlan J. Smith Telescope at McDonald Observatory, and the remaining stars (visible only from the southern hemisphere) with the Clay Magellan Telescope at Las Campanas Observatory in Chile.

All of these observations used high-resolution spectroscopy to get a deep understanding of the stars' chemical make-up.

But several factors are needed to really pin down a solar sibling, Ramirez said. In addition to chemical analysis, his team also included information about the stars' orbits—where they had been and where they are going in their paths



around the center of the Milky Way galaxy. Considering both chemistry and orbits narrowed the field of candidates down to one: HD 162826.

No one knows whether this star hosts any life-bearing planets. But by "lucky coincidence," Ramirez said, the McDonald Observatory Planet Search team has been observing HD 162826 for more than 15 years.

Studies by The University of Texas' Michael Endl and William Cochran, together with calculations by Rob Wittenmyer of the University of New South Wales, have ruled out any massive planets orbiting close to the star (so-called hot Jupiters), and indicate that it's unlikely that a Jupiter analog orbits the star. The studies do not rule out the presence of smaller terrestrial planets.

The finding of a single solar sibling is intriguing, but Ramirez points out the project has a larger purpose: to create a road map for how to identify solar siblings, in preparation for the flood of data expected soon from surveys such as Gaia, the European Space Agency mission to create the largest and most precise 3-D map of the Milky Way.

The data coming soon from Gaia is "not going to be limited to the solar neighborhood," Ramirez said, noting that Gaia will provide accurate distances and proper motions for a billion stars, allowing astronomers to search for solar siblings all the way to the center of our galaxy.

"The number of stars that we can study will increase by a factor of 10,000," Ramirez said.

He says his team's road map will speed up the process of winnowing down the field of potential solar siblings.

"Don't invest a lot of time in analyzing every detail in every star," he said. "You can concentrate on certain key chemical elements that are going to be very useful."

These elements are ones that vary greatly among stars, which otherwise have very similar chemical compositions. These highly variable chemical elements are largely dependent on where in the galaxy the star formed. Ramirez's team has identified the elements barium and yttrium as particularly useful.

Once many more solar siblings have been identified, astronomers will be one step closer to knowing where and how the sun formed. To reach that goal, the dynamics specialists will make models that run the orbits of all known solar siblings backward in time to find where they intersect: their birthplace.

*More information: The research article is available on Ramirez' website at*

[www.as.utexas.edu/~ivan/sun\\_siblings.pdf](http://www.as.utexas.edu/~ivan/sun_siblings.pdf)

<http://bit.ly/1hGIOWI>

## **Interesting prospects for Comet A1 Siding Spring versus the Martian atmosphere**

***It may be the chance of a lifetime for planetary science. This October, a comet will brush past a planet, giving scientists a chance to study how it possibly interacts with a planetary atmosphere.***

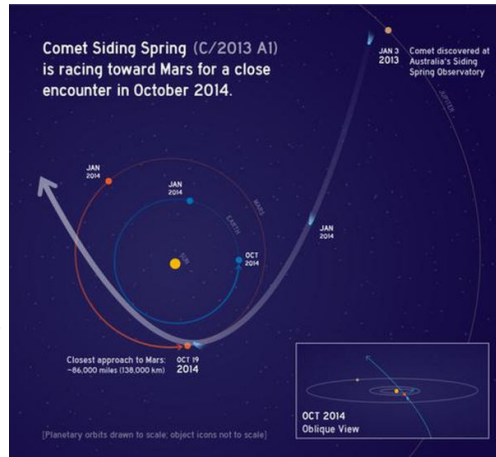
The comet is C/2013 A1 Siding Spring, and the planet in question Mars. And although an impact of the comet on the surface of the Red Planet has long been ruled out, a paper in the May 2014 issue of *Icarus* raises the interesting possibility of possible interactions of the coma of A1 Siding Spring and the tenuous atmosphere of Mars. The study comes out of the Department of Planetary Sciences at the University of Arizona, the Belgian Institute for Space Aeronomy, the Institut de Planétologie et d'Astrophysique de Grenoble at the Université J. Fourier in France, and the Cooperative Institute for Research in Environmental Sciences at the University of Colorado in Boulder. For the study, researchers considered how active Comet A1 Siding Spring may be at the time of closest approach on October 19th, 2014.

Discovered early last year by Robert McNaught from the Siding Spring Observatory in Australia, Comet A1 Siding Spring created a stir in the astronomical community when it was found that it will pass extremely close to Mars later this year. Further measurements of its orbit have since ruled this possibility out, but its passage will still be a close one, with a nominal passage of 138,000 kilometres from Mars. That's about one third the distance from Earth to the Moon, and 17 times closer than the nearest recorded passage of a comet to the Earth, Comet D/1770 L1 Lexell in 1780. Mars' outer moon Deimos has an orbital distance of about 23,500 kilometres.

And although the nucleus will safely pass Mars, the brush with its extended atmosphere might just be detectable by the fleet of spacecraft and rovers in service around Mars. At a distance of 1.4 Astronomical Units (A.U.) from the Sun during the encounter, the vast coma is expected to be comprised primarily of H<sub>2</sub>O. At an input angle of about 60 degrees, penetration was calculated in the study to impinge down and altitude of 154 kilometres to the topside of the Martian ionosphere, in the middle of the thermosphere.

Such an effect should linger for just over 4 hours, well over the interaction period of Mars' atmosphere with the coma of just over an hour, centered on 18:30 UT on October 19th, 2014.

What kind of views might missions like HiRISE and MSL get of the comet remains to be seen, although NEOWISE and Hubble are already monitoring the comet for enhanced activity. The Opportunity rover is also still functioning, and Mars Odyssey and ESA's Mars Express are still in orbit around the Red Planet and sending back data. But perhaps the most interesting possibilities for observations of the event are still en route: India's Mars Orbiter Mission and NASA's MAVEN orbiter arrive just before the comet. MAVEN was designed to study the upper atmosphere of Mars, and carries an ion-neutral mass spectrometer (NGIMS) which could yield information on the interaction of the coma with the Martian upper atmosphere and ionosphere. The NGIMS cover is slated for release just two days before the comet encounter. All spacecraft orbiting Mars may feel the increased drag effects of the encounter.



***The passage of Comet 2013 A1 Siding Spring through the inner solar system.*** Credit: NASA.

Proposals for using Earth-based assets for further observations of the comet prior to the event in October are still pending. Amateur observers will be able to follow the approach telescopically, as Comet A1 Siding Spring is expected to reach +8th magnitude in October and pass 7' from Mars in the constellation Ophiuchus as seen from the Earth. Mars just passed opposition last month, but both will be low to the south west at dusk for northern hemisphere observers in October.

It's also interesting to consider the potential for interactions of the coma with the surfaces of the moons of Mars as well, though the net amount of water vapor expected to be deposited will not be large.

The H<sub>2</sub>O coma of A1 Siding Spring is expected to have a radius of 150,000 kilometres when it passes Mars, just a shade over the nominal flyby distance. "There is a more extended coma made up of H<sub>2</sub>O dissociation products (such as hydrogen and hydroxide) that extends for ~1,000,000 kilometres," researcher at the Department of Planetary Sciences at the University of Arizona and lead author on the paper Roger Yelle told Universe Today.

"Essentially, Mars is in the outer reaches of the coma. The main ion tail misses Mars but there will be some ions from the comet that do reach Mars. The dust tail just misses Mars, which is fortunate."

The paper also notes that significant perturbations of the upper atmosphere of Mars will occur if the cometary production rate is  $10^{28}$  s<sup>-1</sup> or larger, which corresponds to about 300 kilograms per second.

"The MAVEN spacecraft will make very interesting observations," Roger Yelle also told Universe Today. "The comet will perturb primarily the upper atmosphere of Mars and MAVEN was designed to study the upper atmosphere of Mars. Also, it's just such an incredible coincidence that the comet arrives at Mars less than one month after MAVEN does. MAVEN is nominally in its checkout phase then, and the main science phase of the mission was not scheduled to start until November 1st. However, we are reassessing our plans to see what observations we can make. It's all quite exciting, and we have to balance safety and the desire to make the best science measurements."

<http://bit.ly/1uNjV4J>

### **A brief history of space flight – in numbers** *Thirty-one astronauts have made a return-trip to Mars.*

16:43 09 May 2014 by [Jacob Aron](#)

Well, not quite – but they have put in the requisite hours in space. That's just one of the surprising insights to come out of a recent attempt to chart humanity's 52-year history in space.

Gilles Clément and Angelia Bukley of the International Space University in Illkirch-Graffenstaden, France, [used publicly available information from the US, Russian and Chinese space programmes](#). Between 12 April 1961, when [Yuri Gagarin took a single orbit around the Earth on board the Soviet Vostok-1 craft](#) and December 2013, they counted the humans who have flown to space, how long they collectively spent there and who they were.

We picked out our favourites six insights, then put them in context with data from elsewhere.

#### **1. Astronauts are as common as Nobel prizewinners**

As of 31 December 2013, 539 individuals had been to space, defined as reaching an altitude of 100

539

People have been into space

566

Nobel prizewinners in science subjects

kilometres or more. That's a rate of about 10 per year, and roughly equivalent to the 566 people who have ever won a Nobel prize in a science subject (physics, chemistry, or physiology/medicine).

(Note: Clément and Bukley's analysis does not include the two commercial astronauts who piloted the [SpaceShipOne test flights in 2004](#), who were in space for just a few minutes each.)

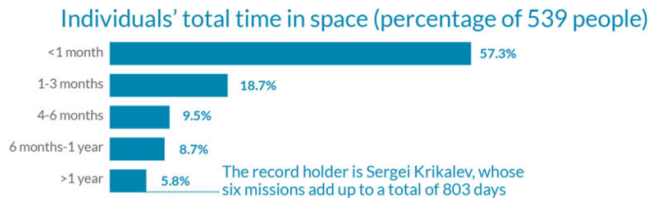
## 2. Space trips last days, months... but rarely years

Gagarin's single orbit of the Earth [lasted just 108 minutes](#). Clément and Bukley found that of a total of 1211 person-flights, defined as a single crew member flying one mission, most last less than a month. Presumably these short hops were [trips to the moon](#) and missions spent inside NASA's now-retired space shuttle, to build and repair the International Space Station. But a significant minority spent five or six months, representing stays on board the ISS.



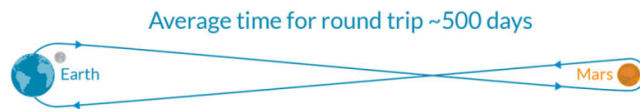
## 3. Many astronauts spend more than a year of their life in space

Though no single trip has been longer than Valeri Polyakov's 437 days aboard the Soviet space station Mir, if you count total time in space over a lifetime, the figures are quite different.



## 4. Thirty-one astronauts have been to Mars and back, sort of

One of Clément and Bukley's most surprising observations is that 31 travellers have spent over a year in total in space, [enough to make a trip to Mars and back](#) – though the exact travel time depends on relative positions of Earth and the Red Planet.



## 5. Like many adventures, space is sexist

Last year, a private foundation announced plans to send a man and woman on a [501-day round-trip to Mars](#), to "represent all of humanity". Spaceflight so far has been far from representative when it comes to gender, though it is not the only extreme pursuit with a skew.



## 6. Space travel is not as dangerous as you might expect

Clément and Bukley also examined the risks of space travel. Counting two lost shuttles and two lost Soyuz capsules, the pair calculated that the chance of dying on a space mission is 1.5 per cent, markedly less than the [percentage of people who die attempting to reach the summit of Mount Everest](#).

Journal reference: [Acta Astronautica, DOI: 10.1016/j.actaastro.2014.04.002](#)

<http://bit.ly/1mdFqrm>

## Polar bear evolved to survive being a heart attack waiting to happen

### Genes involved in cardiovascular health, cholesterol have changed rapidly.

If you ask what separates two species, a lot of people would tell you that an inability to produce fertile offspring is the key test. But the polar bear provides a great example of how biology refuses to be pinned down by these simple binary distinctions. Despite a number of very obvious features that distinguish the polar bear from other bears, it readily produces fertile offspring with brown bears. And fossil evidence suggests that the two species separated about half a million years ago.

Researchers have now sequenced 80 separate polar bear genomes as well as a number of brown bears for comparison, and the results show that appearances only tell a very small part of the story. Polar bears have taken up a blubber-rich diet that may leave them with up to half their body weight made up by fat. And as it turns out, most of the polar bear's genes that have undergone rapid evolution seem to be involved in keeping its cholesterol under control and its heart from exploding under the strain.

Although fossil evidence has suggested that polar and brown bears shared a common ancestor relatively recently, there have been a few indications that the two may have been separated for over a million years. The new genome pretty much rules the older date out. The DNA indicates that the split occurred between 300,000 and 470,000 years ago. For a variety of reasons, the authors think the actual date is in the lower end of this range.

That, as it turns out, roughly lines up with the longest interglacial period of the last million years, a 50,000-year period in which forests established themselves in southern Greenland. The authors suggest that this will have allowed brown bears to settle many areas within the Arctic and left isolated populations when the glaciers began to grow again. These remnant populations would have been under intense pressure to adapt to the icy environment they found themselves in. What ended up being a polar bear obviously has a lot of physical adaptations: white fur, broad paws for swimming, a distinctive head shape, etc. The authors



came up with a couple of genes that are likely involved in the pale coat color, but they didn't look into anything else in any detail.

In part, that's because when they looked for genes that have changed rapidly, they came up with so many other things. One of the most obvious is a gene involved in cholesterol metabolism. Despite all the time since their split from a common ancestor, panda bears and brown bears have no differences in this gene. In contrast, polar bears have picked up nine different changes in this gene in the 400,000 years or so that they've been breeding separately.

Whatever it's doing, the modified protein isn't keeping the bear's cholesterol down; the authors note that "Cholesterol levels in blood plasma of polar bears are extreme." Instead, the bear's evolution seems to have reworked the heart to survive these extreme cholesterol levels. Nine of the 16 genes that are changing the most in response to selective pressure are involved in cardiovascular development or maintenance. A few of the rest are involved in forming adipose tissue—presumably to get the bear up to the 50 percent fat figure noted above. Based on a typical generation time of a bit over 11 years, all of these changes have taken place in less than 20,000 generations—the blink of an eye in evolutionary terms, especially for a large mammal. So, although we tend to think of species as distinctive and evolution as a slow process, the polar bear clearly tells us that generalizations like these only count for so much. *CELL*, 2012. DOI: 10.1016/j.cell.2014.03.054 (About DOIs).

<http://nyti.ms/1hFXdCy>

### A Simple Theory, and a Proposal, on H.I.V. in Africa

*While around the world a vast majority of AIDS victims are men, Africa has long been the glaring exception: Nearly 60 percent are women. And while there are many theories, no one has been able to prove one.*

By [DONALD G. McNEIL Jr.](#) MAY 10, 2014

Otimati, South Africa - In a modest public health clinic behind a gas station here in South Africa's rural KwaZulu/Natal Province, a team of Norwegian infectious disease specialists think they may have found a new explanation.

It is far too soon to say whether they are right. But even skeptics say the explanation is biologically plausible. And if it is proved correct, a low-cost solution has the potential to prevent thousands of infections every year.

The Norwegian team believes that African women are more vulnerable to H.I.V. because of a chronic, undiagnosed parasitic disease: genital schistosomiasis (pronounced shis-to-so-MY-a-sis), often nicknamed "schisto."

The disease, also known as bilharzia and snail fever, is caused by parasitic worms picked up in infested river water. It is marked by fragile sores in the far

reaches of the vaginal canal that may serve as entry points for H.I.V., the virus that causes AIDS. Dr. Eyrun F. Kjetland, who leads the Otimati team, says that it is more common than syphilis or herpes, which can also open the way for H.I.V. Also, the foreign bodies in the sores — the worms and eggs — attract CD4 cells, the immune system's sentinels, and those are the very cells that H.I.V. attacks. The worms can be killed by a drug that costs as little as 8 cents a pill. Dr. Kjetland's team is trying to determine whether that will heal the sores in young women.

Some prominent AIDS experts doubt the schistosomiasis theory, pointing out, for example, that urban women raised far from infested water also die of AIDS. But proponents of the theory say that two decades ago, many experts were just as skeptical of the idea that circumcision protected men against H.I.V. It was not until 2006 that three clinical trials proved it correct.

Schistosomiasis "is arguably the most important cofactor in Africa's AIDS epidemic," said Dr. Peter J. Hotez, dean of the National School of Tropical Medicine at Baylor College of Medicine. "And it's a huge women's health issue: Everyone has heard of genital mutilation and obstetric fistulas. But mention this, and the headlights just go dim."

The idea is slowly gaining ground. The Bill & Melinda Gates Foundation, the United Nations, the National Institutes of Health, and the Danish and Norwegian governments have all given some grant support. But leaders of the two agencies that pay for the fight against global AIDS want more evidence before diverting funds from their campaigns for condoms, drugs and circumcision.

"We need to track all these things down and see what's a cause and what's just another disease you have at the same time, like cervical cancer," said Dr. Mark R. Dybul, executive director of one of the agencies, the Global Fund to Fight AIDS, Tuberculosis and Malaria.

Dr. Eric Goosby, who recently finished a five-year stint as coordinator of the other agency, the President's Emergency Plan for AIDS Relief, or PEPFAR, agreed that vaginal sores could help the virus



enter. “But it’s complicated,” he added. “A lot of women who have H.I.V. don’t have schisto, and vice versa.”

From her small clinic just off the highway here, Dr. Kjetland makes visits to high schools where she has government permission to work because their communities have the highest rates of schistosomiasis. On the dirt roads around these hills, it can take her hours to reach each one.

Through school nurses, she gives deworming drugs to all students, male and female. (To her frustration, although the drug is sold by generic makers for as little as eight cents a pill, South African patent laws permit only the Bayer version, which costs \$4.)

Then she meets with groups of girls ages 16 and up to ask the sexually active ones to come to Otimati for gynecological exams and blood tests.

“I am as gentle as I can be, much more gentle than sex is for them,” she said, “but even the slightest touch and they bleed.”

Gentleness is part of Dr. Kjetland’s nature. A 49-year-old stepmother of five, she watches like a mother over the girls in her study. She ordered that extra rooms be built where they can cry if they test positive for H.I.V. She tries to make sure the boys in their schools never realize she chooses only sexually active girls. And she has KFC delivered, since it is a treat for girls who often have only cornmeal mush to eat for days on end.

Though trained in Norway, she has spent most of her life in Africa, growing up in Tanzania as the eldest of a missionary couple’s six children, attending prep school in South Africa, and, after college and medical school in Norway, doing graduate work in Malawi and Zimbabwe.

An estimated 200 million Africans have had schistosomiasis. Although it is rarely fatal, the bleeding it causes in children can lead to [anemia](#), stunted growth and learning problems. It is caused by tiny worms that live in freshwater snails and [emerge](#) with pointed heads that can penetrate the skin of people collecting water or washing clothes.

Once inside, the worms mate, with the female living in a cleft in the male’s body “like a hot dog in a bun,” Dr. Kjetland said. Most nest in the urinary tract — [bloody urine](#) is the classic symptom — but a portion end up in the vagina, creating “sandy patches” of damaged tissue and calcified eggs.

Studies by Dr. Kjetland in Zimbabwe and South Africa and by Dr. Jennifer A. Downs of Weill Cornell Medical College in Tanzania have shown that women with the patches are about [three times as likely](#) as their neighbors [to be infected](#) with H.I.V.

A gold standard study to prove the connection would be both impractical and unethical: Researchers would have to divide hundreds of infant girls into two groups, give half deworming drugs and half placebos, wait until they were perhaps 20 years old, and see how many had H.I.V. No ethics board would approve placebos under those conditions.

So Dr. Kjetland studies teenagers, hoping to heal their sores and see if their H.I.V. infection rates are lower than the norm. (In grown women, the sores persist even after the worms die.)

For years, theories have abounded as to why African women become infected with H.I.V.: for example, that they are more likely to have overlapping sexual partners — not always by choice — while women elsewhere have boyfriends or husbands in series. That rape, incest and domestic violence are rife in southern Africa, where the AIDS epidemic is worst. That syphilis and herpes are rampant. That impoverished, fatherless young women are forced to pay with sex for food, clothes, grades and even car rides.

The schisto hypothesis can now be added to that list, but to some prominent experts it remains unlikely.

One is Daniel Halperin, an epidemiologist now at the Ponce School of Medicine and Health Sciences in Puerto Rico. He knows how it feels to be doubted: In the 1990s, he was the chief proponent of the theory that circumcision protected men against H.I.V.

He argues that tropical West Africa, where schisto is common, has little H.I.V., while countries with little schisto, like arid Botswana and mountainous Swaziland, have sky-high H.I.V. rates.

Dr. Salim Abdool Karim, a renowned South African AIDS researcher who admires Dr. Kjetland’s work, is also skeptical. His team follows more than 1,000 women in an area only 40 miles from Otimati with equally high H.I.V. rates.

“We’ve studied genital tracts in detail for 20 years, photographing them sequentially,” he said, “and we see no sandy patches.”

Upon hearing that, Dr. Kjetland reached for the mounted magnifying scope she uses to examine girls.

“They’re not looking in the right places,” she said.

Most gynecologists, she explained, are trained to look for [cancer](#), which usually starts near the center of the cervix, while sandy patches are tucked away in crevices that can be seen only by swinging the scope to extreme angles. It takes her weeks to train doctors to find them consistently, she said.

Fighting schisto across Africa would take an extensive pill-distribution effort, but Dr. Hotez, the Baylor dean, argues that it is worth it.

Seventy million African children could be dewormed twice a year for 10 years at a cost of \$112 million, he said in [an essay](#) titled "Africa's 32 Cents Solution for H.I.V./AIDS" (32 cents being the cost of two generic deworming pills twice a year). That is cheap compared with the \$38 billion Pefpar is expected to spend on AIDS in that period, he said.

A vaccine would be even better, and [several](#) are in development, including one at the [Sabin Vaccine Institute](#), which Dr. Hotez also heads.

But even if one works, "it will be at least five to 10 years before the testing is finished," he said. "We shouldn't wait for that."

<http://bit.ly/110ObSW>

### Ice-loss moves the Earth 250 miles down

*At the surface, Antarctica is a motionless and frozen landscape. Yet hundreds of miles down the Earth is moving at a rapid rate, new research has shown.*

The study, led by Newcastle University, UK, and published this week in Earth and Planetary Science Letters, explains for the first time why the upward motion of the Earth's crust in the Northern Antarctic Peninsula is currently taking place so quickly.

Previous studies have shown the earth is 'rebounding' due to the overlying ice sheet shrinking in response to climate change. This movement of the land was understood to be due to an instantaneous, elastic response followed by a very slow uplift over thousands of years.

But GPS data collected by the international research team, involving experts from Newcastle University, UK; Durham University; DTU, Denmark; University of Tasmania, Australia; Hamilton College, New York; the University of Colorado and the University of Toulouse, France, has revealed that the land in this region is actually rising at a phenomenal rate of 15mm a year – much greater than can be accounted for by the present-day elastic response alone.

And they have shown for the first time how the mantle below the Earth's crust in the Antarctic Peninsula is flowing much faster than expected, probably due to subtle changes in temperature or chemical composition. This means it can flow more easily and so responds much more quickly to the lightening load hundreds of miles above it, changing the shape of the land.

Lead researcher, PhD student Grace Nield, based in the School of Civil Engineering and Geosciences at Newcastle University, explains: "You would expect this rebound to happen over thousands of years and instead we have been able to measure it in just over a decade. You can almost see it happening which is just incredible.

"Because the mantle is 'runnier' below the Northern Antarctic Peninsula it responds much more quickly to what's happening on the surface. So as the glaciers thin and the load in that localised area reduces, the mantle pushes up the crust.

"At the moment we have only studied the vertical deformation so the next step is to look at horizontal motion caused by the ice unloading to get more of a 3-D picture of how the Earth is deforming, and to use other geophysical data to understand the mechanism of the flow."

Since 1995 several ice shelves in the Northern Antarctic Peninsula have collapsed and triggered ice-mass unloading, causing the solid Earth to 'bounce back'.

"Think of it a bit like a stretched piece of elastic," says Nield, whose project is funded by the Natural Environment Research Council (NERC). "The ice is pressing down on the Earth and as this weight reduces the crust bounces back. But what we found when we compared the ice loss to the uplift was that they didn't tally – something else had to be happening to be pushing the solid Earth up at such a phenomenal rate."

Collating data from seven GPS stations situated across the Northern Peninsula, the team found the rebound was so fast that the upper mantle viscosity - or resistance to flow - had to be at least ten times lower than previously thought for the region and much lower than the rest of Antarctica.

Professor Peter Clarke, Professor of Geophysical Geodesy at Newcastle University and one of the authors of the paper, adds: "Seeing this sort of deformation of the earth at such a rate is unprecedented in Antarctica. What is particularly interesting here is that we can actually see the impact that glacier thinning is having on the rocks 250 miles down."

<http://www.bbc.com/news/uk-england-27307476###rsslwmlink>

### Why do people eat placentas?

*It is a waste product from childbirth, so why would anyone choose to eat a raw human placenta?*

By Laura Devlin BBC News, East

It sustains life in the womb and has fulfilled its primary purpose once it leaves the mother during childbirth. The human placenta then becomes something rather messy to be discarded - but not everyone sees it that way.

The nutrients that have passed from mother to foetus over months of pregnancy are, some believe, still packed inside the bloody organ and should not be wasted. Instead, the raw placenta could provide just what the mother needs as she recovers from childbirth and begins breastfeeding. And that means eating it.

Some women are opting to drink the placenta in a fruit smoothie within hours of giving birth, keeping it cool and sending it off to be dried and made into capsules, or even ripping of a chunk and placing it by their gums.

They are convinced that the magic bullet gives them an energy boost, can encourage breast milk production and even prevent post-natal depression.

The Independent Placenta Encapsulation Network (IPEN) is tapping into the recent trend for eating placenta and charges £150 for capsules, £25 for a smoothie. But the company is currently awaiting the outcome of a court case that could see it shut down. In what is thought to be the first case of its kind, Dacorum Borough Council prevented IPEN from trading in October last year over concerns about bacterial contamination. Watford magistrates heard the case and have reserved judgement on the matter.

Charlie Poulter, from Reading, is convinced that ingesting a palm-sized piece of placenta within a cocktail of red berries and banana gave her energy after her labour. "I did drink it rather quickly because I didn't want to think about it," she said. "But I'd just pushed a baby out, I'd had lots of people look at me. It felt insignificant compared to what I had just gone through. "I thought 'if this is going to stop post-natal depression and give me some energy, then I can drink three-quarters of a pint of liquid. Man up and drink it'.

The 30-year-old's motivation was pretty clear. She had been receiving therapy for depression for 18 months when she became pregnant and was concerned about developing post-natal depression. "I had never heard of placenta encapsulation before but found out that it could help with the baby blues. "I was willing to try anything and my husband said that even if it had a placebo effect, it doesn't matter, it's not going to harm you."

IPEN advised her to put her wishes in in her birthing plan and inform her midwife. An IPEN specialist was to make a placenta smoothie at her hospital bedside, so Charlie requested a private room for her labour in June 2011. A cool box was also an essential item in her hospital bag, so IPEN could store more of her placenta to dry and make into capsules, which Charlie received within a few days.

Another piece was soaked in alcohol for a tincture, the latter of which she still uses "like Rescue Remedy".

"My daughter Lillian was my first child, so I have nothing to compare this to, but I had a lot of energy - I didn't feel completely dead. "My husband was more exhausted than I was." She also says she did not develop post-natal depression and "swears it was the placenta". She has since become an IPEN specialist herself.

Humans are in the minority over placentophagy, or eating the placenta. With the exception of marine mammals and some domesticated ones, all other mammals consume the afterbirth - possibly to help with the bonding process.

Dried placenta is used in some traditional Chinese medicine and is thought to be a restorative, but the practice of placentophagy is a more recent trend in western culture and is not without controversy

In 1998, Channel 4 was reprimanded for showing a woman's afterbirth being served up as pate by TV chef Hugh Fearnley-Whittingstall. The placenta was fried with shallots and garlic, flambeed, pureed and served to a new mother's 20 relatives and friends as a pate on focaccia bread. The Broadcasting Standards Commission said the episode of TV Dinners, shown in February, breached a taboo and "would have been disagreeable to many".

Labour's Kevin McNamara, who was then MP for Hull North, said the programme was "offensive to the public". More recently, US actress and vegan Alicia Silverstone published placenta recipes after eating her afterbirth, and Mad Men actress January Jones reported consumed dried placenta capsules after the birth of her son Xander.

Writer Nick Baines gave a mixed review after he whizzed up a smoothie and tucked into a taco - both containing his wife's raw placenta following the birth of their son. But what of the science?

### **Mother's choice**

To date, there is not one double-blind placebo controlled study on human placentophagy.

Last year, the University of Nevada surveyed women who had eaten their placenta. Many reported health benefits, but, the researchers said, very little work had been carried out to assess this anecdotal evidence.

The Royal College of Midwives (RCM) said there was not enough evidence for the organisation to "either support or not support" placentophagy as there had not been enough research on the health benefits.

But spokeswoman Jacque Gerrard said: "Our view is that if a mother wants to keep her placenta, it's her choice and it should be facilitated." She added that while "anecdotally" they were hearing about more women asking to keep the placenta, they could not say if there had been an "actual rise" because it was not something they monitored.

Whatever the health benefits, there is no denying the strong reaction the subject raises whenever placentophagy makes the news.