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Common anti-inflammatory may increase risk of diabetes

A commonly prescribed anti-inflammatory may increase the risk of diabetes after just one week of treatment

A commonly prescribed anti-inflammatory may increase the risk of diabetes after just one week of treatment, according to new findings presented at The Society for Endocrinology Annual Conference. Healthy men given doses of the drug comparable to those used to treat inflammatory disorders had changes in markers of blood sugar metabolism associated with an increased risk of developing diabetes. The study findings highlight the potential long-term health implications for people regularly taking these drugs and that medical professionals may need to consider and monitor the potential side-effects, to avoid future debilitating conditions.

Glucocorticoids (GCs) are one of the most commonly prescribed anti-inflammatories for conditions such as arthritis, asthma, allergies and adrenal insufficiency. GC treatment at high doses for a long duration is known to be associated with metabolic side-effects that may increase risk of diabetes and obesity but there are currently no studies examining the short-term effects of GCs at the more regularly prescribed, lower doses. As 2-3% of the UK population take GCs for conditions of varying severity, it is important to investigate whether these metabolic side-effects occur in lower dose, short-term therapy.

Dr Riccardo Pofi, from Sapienza University of Rome and Prof Jeremy Tomlinson from the University of Oxford, measured markers of metabolism in healthy men given commonly prescribed doses (10 and 15mg) of GCs (prednisolone) after just one week of treatment. Although commonly checked clinical and biochemical parameters such as fasting blood sugar levels, weight and general health was unaffected, changes in metabolic markers indicated that their blood sugar regulation was impaired.

"This is the first study to examine the very short-term metabolic effects of commonly prescribed doses of glucocorticoids on healthy men and indicates, that even at these lower doses, glucose metabolism is impaired, suggesting an increased risk of diabetes with continued treatment," Dr Pofi comments.

These novel findings not only highlight the importance of determining the best GC dose that balances effectiveness with potentially negative metabolic effects, but also that medical professionals should be more aware of these risks and may need to monitor them in patients both on short and longer-term therapy.

Dr Pofi says, "This suggests that we need to more accurately assess GC use in patients to prevent and reduce the undesired effects, especially in patients for which steroid treatment is essential for life."

Future larger studies are required to confirm these findings and improve our understanding of how they are caused. Dr Pofi now plans to investigate the metabolic effects of taking GCs alongside diabetes drugs, to assess whether the unwanted side effects of GCs can be reduced or prevented with combined treatment.

Glucocorticoid treatment is associated with dose-dependent effects in healthy male volunteers

Riccardo Pofi, Ilaria Bonaventura, Nanthia Othonos, Thomas Marjot, Ahmed Moolla, Andrea M. Isidori, Leanne Hodson, Jeremy W. Tomlinson

OBJECTIVE: 2-3% of the population of the UK receive glucocorticoid(GC) therapy. Significant adverse effects are not confined to chronic use: recurrent short-course administration is associated with increased morbidity and mortality. Data about the cumulative dose responsible for drawbacks during GC treatment are still lacking. The aim of this study was to test the impact of 7 days of 10 or 15mg of Prednisolone on metabolism in healthy male volunteers.

METHODS: 16 healthy male volunteers were recruited from the Oxford Bio-Bank and divided into 2 age- and BMI-matched control groups as following: 6 volunteers received 10 mg of Prednisolone and 10 volunteers received 15 mg

of Prednisolone for 7 days. Anthropometric and metabolic parameters were recorded and all patients underwent low dose hyperinsulinaemic-euglycaemic clamp(HEC), before(pre) and after(post) treatment. The main outcome measure was the M-value gathered from the HEC.
RESULTS: Age, BMI and fasting blood glucose were not different between the groups at baseline. After one week of prednisolone 10 or 15 mg, no differences were found in delta(Δ=post-pre) fasting glucose(FG) (median ΔFG15mg 0.15±0.36 nmol/L vs ΔFG10mg 0.15±0.36 nmol/L, p=0.635). However, M-value was significantly reduced in patients taking 15 mg of prednisolone (median ΔM15mg -2.5±2.0 mg/Kg/min vs ΔM10mg -0.4±1.3 mg/Kg/min, p=0.016), as well as serum potassium (median ΔK15mg -0.3±0.2 mEq/L vs ΔK10mg 0.10±0.18 mEq/L, p=0.011). No differences were found in Δcholesterol (total, HDL and non-HDL), liver or kidney function.
CONCLUSIONS: In this small cohort of healthy male volunteers, we demonstrated that GC treatment is associated with a worsening of insulin sensitivity through a dose-dependent effect. In addition, the decrease of serum potassium underpin the dose-dependent mineralocorticoid activity of GC. Further studies are needed to confirm our findings in larger cohort of patients.

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Hurricanes have become bigger and more destructive for USA; new study from the Niels Bohr Institute

The worst of them are more than 3 times as frequent now than 100 years ago

A new study by researchers at the Niels Bohr Institute, University of Copenhagen, Aslak Grinsted, Peter Ditlevsen and Jens Hesselbjerg shows that hurricanes have become more destructive since 1900, and the worst of them are more than 3 times as frequent now than 100 years ago. A new way of calculating the destruction, compensating for the societal change in wealth, unequivocally shows a climatic increase in the frequency of the most destructive hurricanes that routinely raise havoc on the North American south- and east coast. The study is now published in PNAS.

Climate change used to be obscured by the statistical uncertainty

The traditional way of calculating hurricane damage, in order to be able to compare hurricanes and follow their development over time, was to survey the subsequent cost of the damage done by each hurricane. In other words, what would a hurricane from the 1950s cost, if it made landfall today? Using this method, a typical find is that the majority of the rising tendency in damage can be attributed to the fact that there are more of us and we are more wealthy, and there is quite simply more costly infrastructure to suffer damage. But evidence of a climatic change in destructive force by hurricanes has been obscured by statistical uncertainty.

Hurricanes are becoming bigger, stronger and more dangerous - an improved calculation method now shows a clear tendency

Aslak Grinsted has calculated the historical figures in a new way. Instead of comparing single hurricanes and the damage they would cause today, he and his colleagues have assessed how big an area could be viewed as an "area of total destruction". Meaning how large an area would you have to completely destroy in order to account for the financial loss. Simultaneously, this makes comparison between rural areas and more densely populated areas like cities easier, as the unit of calculation is now the same: The size of the "area of total destruction".

The climate signal in the new method has suddenly become apparent

In previous studies it proved difficult to isolate the "climate signal". The climate signal should be understood as the effect climate change has had on hurricane size, strength and destructive force. It lay hidden behind variations due to the uneven concentration of wealth and it was statistically uncertain whether there was any tendency in the destruction. But with the new method this doubt has been eradicated. The weather has indeed become more dangerous on the south- and east coast of the USA. Furthermore, the result obtained by the research team has turned out to be more congruent

with the climate models we use to predict and understand the development in extreme weather. It fits with the physics, quite simply, that global warming has the effect that there is an increase in the force released in the most extreme hurricanes.

http://bit.ly/33Mi1HM

New vaccine protects from widespread, costly infection, mice study shows

More than 80% effective in protecting mice from S. aureus *infection*

Washington, DC - A newly developed experimental vaccine was more than eighty percent effective in protecting mice from succumbing to *Staphylococcus aureus* infection. *S. aureus* causes more than 30,000 deaths from hospital-acquired infections annually in the US, costing the healthcare system $10 billion. The research is published this week in *Infection and Immunity*, a journal of the American Society for Microbiology.

S. aureus is associated with a wide range of acute and chronic diseases such as bacteremia, sepsis, skin and soft tissue infections, pneumonia endocarditis, and osteomyelitis (bone infection), and has a high rate of mortality, estimated at 20-30 percent in bacteremia (blood infection) patients.

In the study, the investigators tested the vaccine in mouse and rabbit models of *S. aureus* infection. More than 80 percent of immunized mice survived, and two thirds of them cleared the infection, versus less than 10 percent of controls. On the 21st day post infection, the surviving animals--both those immunized, and controls--showed no signs of ill health, such as ruffled fur, or other abnormalities of appearance, and all had regained pre-infection weight.

In the rabbit experiments, the researchers injected the pathogen into the tibial bone marrow. Twenty-four days post infection, nearly two thirds of the immunized rabbits had cleared the infection; none of the controls had done so. Additionally, while control rabbits had hole-like lesions within the bone, immunized rabbits had smaller lesions or no lesions at all. (Rabbits do not typically succumb to *S. aureus* infection.)

Effective vaccination "would have enormous therapeutic utility in patients undergoing surgery, especially orthopedic and cardiovascular procedures where medical structures or devices are implanted, and in cases of traumatic injury," said Janette M. Harro, PhD, Research Assistant Professor, University of Maryland, Baltimore. Surgical site infections represent 20 percent of hospital acquired infections, and *S. aureus* is the major causative agent.

The diversity of disease caused by *S. aureus* results from differential expression of more than 70 virulence factors. Virulence factors initiate colonization and growth, mediate damage to the host, and hinder immune response.

Biofilm formation is a powerful virulence factor. *S. aureus* is difficult to eradicate largely because it so readily forms biofilms.

Biofilms are communities of bacteria that adhere powerfully to surfaces, in the manner of dental plaque. They are notably resistant to host immune response, and to antibiotics, because they are hard to penetrate, and because microbes in biofilms have low metabolism, which further reduces the potential to gain entry into bacterial cells.

Biofilms frequently form on medical implants such as artificial knees, hips, and cardiac devices. They can form anywhere there's a surface, moisture, and a nutrient source.

The vaccine the investigators developed recognizes five different *S. aureus* proteins. Four of these proteins are specific to *S. aureus* biofilms, and one is specific to *S. aureus* in the planktonic state.

"We identified vaccine candidates by screening *S. aureus* proteins with antibodies elicited during chronic *S. aureus* infections in animal models," said Dr. Harro. "This method permitted us to select

protein targets for vaccination that were both expressed during an infection and were capable of being recognized by the immune response."

http://bit.ly/351ADUv

Anticoagulant benefits for atrial fibrillation decrease with age

Typical patient may not benefit after age 87, UCSF-led study finds

The net clinical benefit of anticoagulants for atrial fibrillation (AF) -- one of the most important causes of irregular heartbeats and a leading cause of stroke -- decreases with age, as the risk of death from other factors diminishes their benefit in older patients, according to a study led by researchers at UC San Francisco.

The multi-institutional study of nearly 15,000 AF patients found that the anticoagulant warfarin was not beneficial after age 87 and another, apixaban, after age 92. As a result, physicians should consider all mortality risks, such as cancer and end-stage kidney disease, when recommending anticoagulants to older adults with AF, the researchers said.

The study is online Nov. 11, 2019, in *Circulation: Cardiovascular Quality and Outcomes (CCQO)*, to coincide with a presentation at the annual American Heart Association Scientific Sessions 2019.

"Many prior studies looking at the benefit of blood thinners found older adults benefit more than younger adults, but they narrowly focus on atrial fibrillation and strokes and don't account for all other health conditions affecting older adults," said lead author Sachin Shah, MD, MPH, assistant professor of medicine at UCSF. "Our study is the first to find that when taking these factors into consideration, anticoagulant benefit actually decreases with age."

Atrial fibrillation affects an estimated 2.2 million Americans, according to the National Stroke Association, and about 15 percent of people who have strokes have AF. The stroke association estimates that up to 80 percent of strokes among people with AF could have been prevented.

While patients age 75 and older are at higher risk for stroke and advised to use anticoagulants, there is little evidence of their net benefit in this population. Advancing age also increases the likelihood of death from non-AF causes, thereby limiting the benefit or harm from AF and anticoagulant treatment.

Indeed, anticoagulant use in older patients with atrial fibrillation is similar to prostate specific antigen (PSA) testing. PSA testing is common in elderly men, despite evidence that those without aggressive prostate cancer are unlikely to benefit from diagnosis and treatment and may face significant risks for quality of life if they undergo prostate surgery.

"Competing risk of death is an important consideration when estimating the net clinical benefit of anticoagulation therapy," Shah said. "Failing to account for competing risks likely overestimates the net clinical benefit of anticoagulation, an effect that is more pronounced at older ages and with more effective anticoagulants."

In the CCQO study, Shah and his colleagues, including collaborators from Kaiser Permanente Northern and Southern California, reviewed the records of 14,946 adults from January 2006 to June 2009 in the Anticoagulation and Risk Factors in Atrial Fibrillation-Cardiovascular Research Network. They selected patients age 75 and older, with an average age of 81.

The researchers used a computerized decision analytic model called the Atrial Fibrillation Decision Support Tool (AFDST), developed by the University of Cincinnati, to determine the potential benefit of anticoagulants. The model uses patient characteristics and guidelines on AF treatment from the American College of Cardiology, American Heart Association and Heart Rhythm Society to offer a recommendation.

The research team estimated the lifetime net clinical benefit of warfarin and apixaban relative to no treatment in quality-adjusted life years (QALY). QALY is a measure of disease burden that includes both the length of life and its quality, with one QALY equaling one year in perfect health.

Using 0.10 lifetime QALYs as the minimal net clinical benefit, warfarin started at 0.45 QALYs at age 75, then fell below 0.10 at age 87, while apixaban started at 0.74 QALYs at age 75, then fell below 0.10 at age 92.

"For years, we have been telling our doctors and patients that we are not being aggressive enough in providing anticoagulant therapy to our patients with atrial fibrillation, and it's a national problem," said senior author Mark Eckman, MD, the Posey Professor of Clinical Medicine at the University of Cincinnati College of Medicine. "This study now adds a caution, acknowledging that while undertreatment is a major concern, at advanced years of age, maybe we should be a little more thoughtful and careful in our treatment decisions about anticoagulation."

Co-Authors: Margaret Fang, of UCSF; Kristi Reynolds, of Kaiser Permanente Southern California; Alan S. Go, of Kaiser Permanente Northern California and UCSF; and Daniel Singer, of Massachusetts General Hospital and Harvard Medical School.

Funding: Funded by the National Institutes of Health (NIH) National Center for Advancing Translational Sciences (NCATS UL1TR000077-05); National Heart, Lung, Blood Institute (U19HL91179, 1RC2HL101589); and National Institutes on Aging (R01 AG15478). Shah was partly supported by the UCSF Division of Hospital Medicine, and Singer was partly supported by the Eliot B. and Edith C. Shoolman Fund of Massachusetts General Hospital.

Disclosures: Eckman has current or recent investigator-initiated grant funding from the Heart Rhythm Society through a grant from Boehinger-Ingelheim Pharmaceuticals, Inc., NCATS (UL1TR000077-05), NIH National Institute of Child Health and Human Development (R01HD094213), Virus Action Coalition of the Centers for Disease Control, Merck, Pfizer Educational Group, Bristol-Myers Squibb/Pfizer Education Consortium, and the Cystic Fibrosis Foundation. Go has a research grant through Kaiser Permanente Northern California Division of Research from iRhythm Technologies. Reynolds has received research support through Kaiser Permanente Southern California Department of Research & Evaluation from iRhythm Technologies. Singer receives research support from Bristol-Myers Squibb and Boehringer-Ingelheim and serves as a consultant or an advisory board member for Bristol-Myers Squibb, Boehringer-Ingelheim, CVS Health, Johnson and Johnson, Merck, and Pfizer.

http://bit.ly/2NO0bys

Contraceptive drug shows promise for preventing and regressing cervical cancer

Findings reported in The American Journal of Pathology suggest medroxyprogesterone acetate as a new affordable non-invasive approach to combat cervical cancer, particularly for women with limited access to healthcare

Philadelphia - A new study in *The American Journal of Pathology*, published by Elsevier, reports that medroxyprogesterone acetate (MPA), the active ingredient in the common contraceptive injection Depo-Provera, was effective in preventing the development of cervical cancer in mice with precancerous lesions. The drug also decreased existing precancerous lesions. If proven effective clinically, MPA may be a boon to women who do not have access to human papillomavirus (HPV) vaccines.

"Although HPV vaccines have been available since 2006, the incidence of precancerous lesions (cervical intraepithelial neoplasia or CIN) and cervical cancer due to HPV has not decreased substantially. The high cost and lack of a global vaccination program have limited the use of these vaccines. A non-invasive, efficient means to treat CIN is urgently needed," explained lead investigator Sang-Hyuk Chung, PhD, of the Center for Nuclear Receptors and Cell Signaling, Department of Biology and Biochemistry at the University of Houston, Houston, TX, USA.

Similar to cervical cancer progression in women, mouse cervical neoplastic disease develops through multiple stages, starting from CIN and culminating in invasive cancer.

Previously, efforts to develop a non-invasive treatment for CIN have been limited. Dr. Chung and co-investigators at the University of Houston had previously shown that MPA regresses cervical

cancer in a mouse model expressing HPV genes responsible for cancer. In this study, they treated CIN-bearing mice with MPA.

Investigators found that cervical cancer did not develop in mice receiving MPA. Further, CIN was absent in most MPA-treated mice, indicating that MPA may be "chemoprotective," not only preventing CIN from progressing to invasive cancer, but also promoting its regression.

The study determined that MPA inhibited cell proliferation and promoted apoptosis (cell death) in CIN lesions. In addition, the preventive effect of MPA was absent in HPV transgenic mice in which the expression of progesterone receptor (PR) was genetically prevented. These results suggest that MPA is efficient for treating PR-positive CIN lesions. PR-positivity may be a useful biomarker for selecting patients who may benefit from MPA in future clinical trials.

"We are optimistic because the mouse model we used in this study has been validated and revealed important mechanisms of cervical cancer," stated Dr. Chung. "MPA injectable suspensions are cheap and stable at room temperature; therefore, there is no need for special storage, which facilitates easy distribution. It is already used as the self-injectable contraceptive Depo-Provera, and thus translation into clinical use would be faster. MPA would be an effective chemoprevention agent for cervical cancer, particularly in women who do not have access to HPV vaccines. However, we should note that several studies have shown that MPA increases the risk of breast cancer."

Cervical cancer is the third most common and third most deadly cancer in women worldwide. HPV is considered a major factor in the development of cervical precancerous lesions and cancers. Although HPV vaccines are effective at preventing HPV infections, they may not be readily available to women in under-developed countries and those of low socio-economic status in developed countries. Surgical removal of a CIN can be clinically beneficial but has adverse effects including shortening the cervix and increasing the risk of complications in future pregnancies - undesirable outcomes in women of child-bearing age. CIN has negative impact on psychological and psychosocial wellness of women at levels similar to those caused by cervical cancer, underscoring the significance of this finding.

http://bit.ly/33SIFyX

Humans' ability to read dog facial expressions is learned, not innate

Ability to recognize dogs' expressions is learned through age and experience and is not an evolutionary adaptation.

In a recent study published in *Scientific Reports*, a team of researchers from Germany and the United Kingdom assessed how experience with dogs affects humans' ability to recognize dog emotions. Participants who grew up in a cultural context with a dog-friendly attitude were more proficient at recognizing dog emotions. This suggests that the ability to recognize dogs' expressions is learned through age and experience and is not an evolutionary adaptation.

Dogs were the first domesticated animal, with humans and dogs sharing more than 40,000 years of social interactions and life together. According to the co-domestication hypothesis, this process allowed humans and dogs to evolve special emotional signals and cognitive skills that favor mutual understanding. We know, for example, that over the millennia, dogs have evolved the ability to understand human words, iconic signs, and other gestures, and research has shown that dogs can even use tone of voice and facial expressions to recognize human emotions. Beyond personal testimony from dog lovers, however, little attention has been paid to how well humans can understand their canine counterparts.

In the current study, led by Federica Amici of the Max Planck Institute for Evolutionary Anthropology and Juliane Bräuer of the Max Planck Institute for the Science of Human History, the researchers set out to understand how well humans can understand the emotional displays of dogs, and where that understanding comes from.

How well do we understand our species' best friend?

In order to test how well humans can understand the emotions behind dog facial expressions, researchers collected photographs of dogs, chimpanzees and humans displaying either happy, sad, angry, neutral, or fearful emotions as substantiated by the photographers. They then recruited 89 adult participants and 77 child participants and categorized them according to their age, the dog-positivity of their cultural context, and the participants' personal history of dog ownership.

Each participant was presented with photographs of dogs, chimps and humans, and asked to rate how much the individual in the picture displayed happiness, sadness, anger or fear. Adults were also asked to determine the context in which the picture had been taken (e.g., playing with a trusted conspecific partner; directly before attacking a conspecific).

The results of the study showed that, while some dog emotions can be recognized from early on, the ability to reliably recognize dog emotions is mainly acquired through age and experience. In adults, the probability of recognizing dog emotions was higher for participants who grew up in a cultural context with a positive attitude toward dogs, regardless of whether they owned a dog themselves.

Without a dog-positive context, we could be barking up the wrong tree

A dog-positive cultural background in which dogs are closely integrated into human life and considered highly important may result in a higher level of passive exposure and increased inclination and interest in dogs, making humans better at recognizing dogs' emotions even without a history of personal dog ownership.

"These results are noteworthy," says Amici, "because they suggest that it is not necessarily direct experience with dogs that affects humans' ability to recognize their emotions, but rather the cultural milieu in which humans develop."

The researchers also found that regardless of age or experience with dogs, all participants were able to identify anger and happiness reliably. While these results may suggest an innate ability favored by the co-domestication hypothesis, it is also possible that humans learn to recognize these emotions quickly, even with limited exposure.

Other than anger and happiness, the children in the study were not good at identifying dog emotions. They recognized anger and happiness more reliably in dogs than in chimps, but otherwise identified dog emotions as poorly as they did chimpanzee emotions, suggesting that the ability to understand how dogs are feeling is not innate.

"We think it would be valuable to conduct future studies that seek to determine exactly which cultural aspects affect one's ability to read dog emotions, and to include real-life stimuli and body expressions in addition to instructed stimuli and facial expressions," states Bräuer. "In this way, we could develop a better understanding of inter-cultural variation in emotion recognition. Hopefully this information could be used to reduce the occurrence of negative incidents between humans and dogs that are caused by humans' inability to read dog signals."

More information: *Federica Amici et al, The ability to recognize dog emotions depends on the cultural milieu in which we grow up, Scientific Reports (2019). DOI: 10.1038/s41598-019-52938-4*

http://bit.ly/33SJCY3

First evidence of feathered polar dinosaurs found in Australia

A cache of 118 million-year-old fossilized dinosaur and bird feathers has been recovered from an ancient lake deposit that once lay beyond the southern polar circle.

by Uppsala University

Feathered dinosaur fossils are famous, but known from a handful of localities worldwide. Examples from the Southern Hemisphere are especially rare, and mainly include only isolated feathers.

Credit: Uppsala universitet

An international team of scientists has analyzed a collection of 10 such fossil feathers found in Australia, which reveal an unexpected diversity of tufted hair-like 'proto-feathers' from meat-eating dinosaurs, together with downy body feathers, and wing feathers from primitive birds that would have been used for flight.

Uniquely, the fossil feathers from Australia were all entombed in fine muddy sediments that accumulated at the bottom of a shallow lake close to the South Pole during the Age of Dinosaurs.

"Dinosaur skeletons and even the fragile bones of early birds have been found at ancient high-latitudes before. Yet, to date, no directly attributable integumentary remains have been discovered to show that dinosaurs used feathers to survive in extreme polar habitats," said Dr. Benjamin Kear from Uppsala University in Sweden, a leading author on the study.

"These Australian fossil feathers are therefore highly significant because they came from dinosaurs and small birds that were living in a seasonally very cold environment with months of polar darkness every year."

The fossil feathers were discovered in the Koonwarra Fish Beds Geological Reserve, which is a heritage listed site 145 km southeast of Melbourne in Victoria, Australia.

"Fossil feathers have been known from Koonwarra since the early 1960s, and were recognized as evidence of ancient birds, but have otherwise received very little scientific attention. Our study is thus the first to comprehensively document these remains, which include new specimens that were examined using cutting-edge technologies," said Dr. Thomas Rich of the Melbourne Museum in Australia, who has led numerous expeditions to the Koonwarra locality.

A suite of advanced microscopic and spectroscopic techniques was employed to determine the anatomy and preservation of the Koonwarra fossil dinosaur and bird feathers.

"The Koonwarra feathers are preserved in incredible detail," said fossil bird expert Professor Patricia Vickers-Rich of Monash University and the Swinburne University of Technology in Melbourne. "There are even tiny filament-like structures that would have 'zipped' the feather vanes together, just as in the flight feathers of modern birds."

However, unlike the structurally complex feathers of birds today, which are characterized by interlocking branches called barbs and barbules, different kinds of small dinosaurs had coverings that comprised much more simpler hair-like 'proto-feathers."

"Dinosaur 'proto-feathers' would have been used for insulation," said Dr. Martin Kundrát, of Pavol Jozef Safarik University in Slovakia, a leading author on the study. "The discovery of 'proto-feathers' at Koonwarra therefore suggests that fluffy feather coats might have helped small dinosaurs keep warm in ancient polar habitats."

Microscopic remains of possible melanosomes—cellular structures that contain color pigments—were also detected on several of the

fossil feathers found at Koonwarra. These traces occurred across the uniformly dark feather surfaces, as well as in distinct bands that might represent original patterning from the polar dinosaurs and birds. Melanic residues have been reported on fossil feathers from elsewhere around the world, and are widely acknowledged as indicators of dinosaur coloration.

The densely packed fossil melanosomes occurring on the Koonwarra feathers could suggest dark colors that perhaps assisted in camouflage, visual communication, and/or heat absorbance in cold polar climates. Possible preservation of biomolecules was also assessed, but proved to be too degraded, and were apparently lost during weathering of the rock.

The Koonwarra fossil feathers provide the first record of dinosaur integument from the ancient polar regions, and hint what was once a global distribution of feathered dinosaurs and early birds.

Some of the fossil feathers found at Koonwarra are on display in the '600 Million Years' exhibition at the Melbourne Museum in Australia.

More information: *Martin Kundrát et al. A polar dinosaur feather assemblage from Australia, Gondwana Research (2019). DOI: 10.1016/j.gr.2019.10.004*

http://bit.ly/2Xj5jha

New Martian Mystery: Oxygen

On Mars, oxygen behaves in a way that so far planetary scientists cannot explain through any known atmospheric or surface process.

The **Sample Analysis at Mars** (SAM) instrument onboard NASA's Curiosity rover has measured the seasonal changes in the gases that fill the air directly above the surface of Gale Crater, and noticed something baffling: oxygen behaves in a way that so far planetary scientists cannot explain through any known atmospheric or surface process.

Over the course of nearly five years (three Mars years) the SAM instrument inhaled the air of Gale Crater and analyzed its composition.

The results confirmed the makeup of the Martian atmosphere at the surface: 95% by volume of carbon dioxide, 2.6% molecular nitrogen, 1.9% argon, 0.16% molecular oxygen, and 0.06% carbon monoxide.

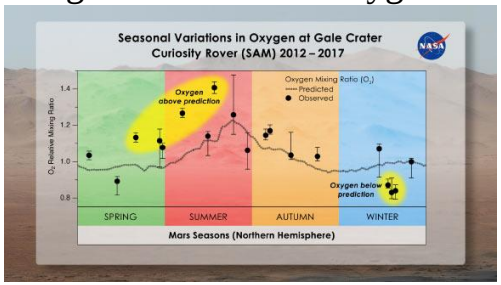
They also revealed how the molecules in the Martian air mix and circulate with the changes in air pressure throughout the year.

These changes are caused when carbon dioxide freezes over the poles in the winter, thereby lowering the air pressure across the planet following redistribution of air to maintain pressure equilibrium. When carbon dioxide evaporates in the spring and summer and mixes across Mars, it raises the air pressure.

Within this environment, the researchers found that nitrogen and argon follow a predictable seasonal pattern, waxing and waning in concentration in Gale Crater throughout the year relative to how much carbon dioxide is in the air.

They expected oxygen to do the same. But it didn't. Instead, the amount of the gas in the air rose throughout spring and summer by as much as 30%, and then dropped back to levels predicted by known chemistry in fall.

This pattern repeated each spring, though the amount of oxygen added to the atmosphere varied, implying that something was producing it and then taking it away. "The first time we saw that, it was just mind boggling," said University of Michigan's Professor Sushil Atreya.



Seasonal variations in oxygen at Gale Crater in 2012-2017. **Melissa Trainer / Dan Gallagher / NASA's Goddard Space Flight Center.**

As soon as the scientists discovered the oxygen enigma, Mars experts set to work trying to explain it.

They first double- and triple-checked the accuracy of the SAM instrument they used to measure the gases: the Quadrupole Mass Spectrometer. The instrument was fine.

They considered the possibility that carbon dioxide or water molecules could have released oxygen when they broke apart in the atmosphere, leading to the short-lived rise. But it would take five times more water above Mars to produce the extra oxygen, and carbon dioxide breaks up too slowly to generate it over such a short time.

What about the oxygen decrease? Could solar radiation have broken up oxygen molecules into two atoms that blew away into space? No, the researchers concluded, since it would take at least 10 years for the oxygen to disappear through this process.

"We're struggling to explain this. The fact that the oxygen behavior isn't perfectly repeatable every season makes us think that it's not an issue that has to do with atmospheric dynamics. It has to be some chemical source and sink that we can't yet account for," said Dr. Melissa Trainer, a planetary scientist at NASA's Goddard Space Flight Center.

To planetary scientists, the oxygen story is curiously similar to that of methane. Methane is constantly in the air inside Gale Crater in such small quantities (0.00000004% on average) that it's barely discernable even by the most sensitive instruments on Mars. Still, it's been measured by SAM's Tunable Laser Spectrometer.

The instrument revealed that while methane rises and falls seasonally, it increases in abundance by about 60% in summer months for inexplicable reasons.

With the new oxygen findings in hand, the team is wondering if chemistry similar to what's driving methane's natural seasonal variations may also drive oxygen's. At least occasionally, the two gases appear to fluctuate in tandem. "We're beginning to see this tantalizing correlation between methane and oxygen for a good part of the Mars year. I think there's something to it. I just don't have the answers yet. Nobody does," Professor Atreya said.

The team's paper was published in the *Journal of Geophysical Research: Planets*.

Melissa G. Trainer *et al*. Seasonal variations in atmospheric composition as measured in Gale Crater, Mars. *Journal of Geophysical Research: Planets*, published online November 12, 2019; doi: 10.1029/2019JE006175

This article is based on text provided by the National Aeronautics and Space Administration.

http://bit.ly/2Ogozb6

The world finally has an approved vaccine against Ebola

The WHO wasted no time to "prequalifying" the newly approved vaccine.

Beth Mole

Regulators in Europe have granted the world's first approval of a vaccine against Ebola—and health officials are wasting no time in rolling it out.

The European Commission announced at the start of the week that it had granted a landmark marketing authorization of Merck's Ebola vaccine Ervebo. The vaccine has been in the works since the 2014 West African Ebola outbreak. It is now being used in the ongoing outbreak in the Democratic Republic of Congo based on a "compassionate use" protocol.

The current outbreak in the DRC has killed nearly 2,200 since August 2018, causing nearly 3,300 cases. The outbreak is the second-largest recorded, surpassed only by the 2014 West African outbreak that caused more than 11,000 deaths and 28,000 cases.

Preliminary vaccine data from the current DRC outbreak suggested that Ervebo is 97.5% effective at preventing the devastating viral disease. It protected well over 90,000 people in the outbreak.

The vaccine protects against one of four species of Ebola known to infect humans—the Zaire ebolavirus species. Zaire is responsible for the current outbreak in the DRC, as well as the 2014 West African outbreak, and nearly all other outbreaks recorded since Ebola was first discovered in 1976.

"Finding a vaccine as soon as possible against this terrible virus has been a priority for the international community ever since Ebola hit West Africa five years ago," European Commissioner Vytenis Andriukaitis said in a statement Sunday, November 10. "Today's decision is therefore a major step forward in saving lives in Africa and beyond."

The vaccine was initially developed by researchers at the Public Health Agency of Canada's National Microbiology Laboratory, which subsequently licensed it to NewLink Genetics Corporation. Merck obtained the license in 2014 amid the West African outbreak and developed it further.

The company celebrated the vaccine's approval Monday.

"It is a historic milestone and a testament to the power of science, innovation and public-private partnership," Merck CEO Kenneth Frazier said in a statement. "After recognizing the need and urgency for an Ebola Zaire vaccine, many came together across sectors to answer the global call for outbreak preparedness. We at Merck are honored to play a part in Ebola outbreak response efforts and we remain committed to our partners and the people we serve."

The US Food and Drug Administration is expected to make an approval decision on the vaccine in March 2020.

In the meantime, the World Health Organization announced Tuesday, November 12 that it has "prequalified" Ervebo, signaling to member countries that the vaccine meets WHO standards for safety, quality, and effectiveness. In an announcement, the WHO noted that it was "the fastest vaccine prequalification process ever conducted by WHO" and came less than 48 hours after the European Commission approved the vaccine. The organization said it is also working to facilitate licensing in countries at risk of Ebola outbreaks.

"[Prequalification] is a historic step towards ensuring the people who most need it are able to access this life-saving vaccine," WHO Director-General Dr. Tedros Adhanom Ghebreyesus said in the announcement. "Five years ago, we had no vaccine and no therapeutics for Ebola. With a prequalified vaccine and experimental therapeutics, Ebola is now preventable and treatable."

http://bit.ly/33SXlOh

The smart move: People learn more by trusting than by not trusting

Experiences lead us to believe that people are too trusting, often verging on gullibility. In fact, we don't trust enough.

Hugo Mercier

We all know people who have suffered by trusting too much: scammed customers, jilted lovers, shunned friends. Indeed, most of us have been burned by misplaced trust. These personal and vicarious experiences lead us to believe that people are too trusting, often verging on gullibility.

In fact, we don't trust enough.

Take data about trust in the United States (the same would be true in most wealthy democratic countries at least). Interpersonal trust, a measure of whether people think others are in general trustworthy, is at its lowest in nearly 50 years. Yet it is unlikely that people are any less trustworthy than before: the massive drop in crime over the past decades suggests the opposite. Trust in the media is also at bottom levels, even though mainstream media outlets have an impressive (if not unblemished) record of accuracy.

Meanwhile, trust in science has held up comparatively well, with most people trusting scientists most of the time; still, in some areas at least, from climate change to vaccination, a share of the

population doesn't trust science enough—with devastating consequences.

Social scientists have a variety of tools to study how trusting, and how trustworthy, people are. The most popular is the trust game, in which two participants play, usually anonymously. The first participant is given a small amount of money, $10 say, and asked to decide how much to transfer to the other participant. The amount transferred is then tripled, and the second participant chooses how much to give back to the first. In Western countries at least, trust is rewarded: the more money the first participant transfers, the more money the second participant sends back, and thus the more money the first participant ends up with. In spite of this, first participants on average transfer only half the money they have received. In some studies, a variant was introduced whereby participants knew each other's ethnicity. Prejudice led participants to mistrust certain groups—Israeli men of Eastern origin (Asian and African immigrants and their Israeli-born offspring), or black students in South Africa—transferring them less money, even though these groups proved just as trustworthy as more esteemed groups.

If people and institutions are more trustworthy than we give them credit for, why don't we get it right? Why don't we trust more?

In 2017, the social scientist Toshio Yamagishi was kind enough to invite me to his flat in Machida, a city in the Tokyo metropolitan area. The cancer that would take his life a few months later had weakened him, yet he retained a youthful enthusiasm for research, and a sharp mind. On this occasion, we discussed an idea of his with deep consequences for the question at hand: the informational asymmetry between trusting and not trusting.

When you trust someone, you end up figuring out whether your trust was justified or not. An acquaintance asks if he can crash at your place for a few days. If you accept, you will find out whether or not he's a good guest. A colleague advises you to adopt a new software application. If you follow her advice, you will find out whether the new software works better than the one you were used to.

By contrast, when you don't trust someone, more often than not you never find out whether you should have trusted them. If you don't invite your acquaintance over, you won't know whether he would have made a good guest or not. If you don't follow your colleague's advice, you won't know if the new software application is in fact superior, and thus whether your colleague gives good advice in this domain.

This informational asymmetry means that we learn more by trusting than by not trusting. Moreover, when we trust, we learn not only about specific individuals, we learn more generally about the type of situations in which we should or shouldn't trust. We get better at trusting.

Yamagishi and his colleagues demonstrated the learning advantages of being trusting. Their experiments were similar to trust games, but the participants could interact with each other before making the decision to transfer money (or not) to the other. The most trusting participants were better at figuring out who would be trustworthy, or to whom they should transfer money.

We find the same pattern in other domains. People who trust the media more are more knowledgeable about politics and the news. The more people trust science, the more scientifically literate they are. Even if this evidence remains correlational, it makes sense that people who trust more should get better at figuring out whom to trust. In trust as in everything else, practice makes perfect.

Yamagishi's insight provides us with a reason to be trusting. But then, the puzzle only deepens: if trusting provides such learning opportunities, we should trust too much, rather than not enough. Ironically, the very reason why we should trust more—the fact that

we gain more information from trusting than from not trusting—might make us inclined to trust less.

When our trust is disappointed—when we trust someone we shouldn't have—the costs are salient, and our reaction ranges from annoyance all the way to fury and despair. The benefit—what we've learned from our mistake—is easy to overlook. By contrast, the costs of not trusting someone we could have trusted are, as a rule, all but invisible. We don't know about the friendship we could have struck (if we'd let that acquaintance crash at our place). We don't realize how useful some advice would have been (had we used our colleague's tip about the new software application).

We don't trust enough because the costs of mistaken trust are all too obvious, while the (learning) benefits of mistaken trust, as well as the costs of mistaken mistrust, are largely hidden. We should consider these hidden costs and benefits: think of what we learn by trusting, the people whom we can befriend, the knowledge that we can gain.

Giving people a chance isn't only the moral thing to do. It's also the smart thing to do.

Hugo Mercier is a research scientist at the CNRS (Institut Jean Nicod) in Paris where he works with the Evolution and Social Cognition team. He is the author of The Enigma of Reason *(2017), co-authored with Dan Sperber, and* Not Born Yesterday *(forthcoming, 2020). He lives in Nantes, France.*

http://bit.ly/2qWOKM2

Scientists Find a Spot Where No Life Can Survive. That's Bad News for Alien Hunters.

Some hydrothermal pools seem to be completely devoid of life, according to a new study

By Yasemin Saplakoglu - Staff Writer

Unearthly greens and yellows color the scorching-hot landscape surrounding the Dallol volcano in northern Ethiopia. This alien-like world is filled with hydrothermal pools that are some of the most extreme environments on the planet — and some of them seem to be completely devoid of life, according to a new study.

***The Dallol hydrothermal pools are harsh environments.* © Shutterstock**

Different life-forms on our planet have adapted to survive under some pretty harsh conditions, places that are superhot, superacidic or supersalty, to name a few, said study senior author Purificación López-García, the research director at the French National Centre for Scientific Research.

But can life survive in a single environment that combines all three conditions, such as in the colorful waters of the Dallol hydrothermal region?

To figure out if this extreme environment oversteps the limits for life on our planet, the researchers sampled a number of brines— or pools of water with high concentrations of salt — in the area. Some were extremely hot, salty and acidic, while others were still very hot and salty but weren't too acidic or basic. The scientists analyzed all the genetic material found in the samples to identify any organisms living there.

Some of the milder pools were chock-full of sodium chloride, a condition that some tiny organisms can withstand; the more extreme environments had high concentrations of magnesium-based salt, which is "deleterious for life," because magnesium breaks down the cell membrane, López-García said.

In these most extreme environments, that were really acidic, hot and contained magnesium salts, the researchers found no DNA and thus no trace of a living organism, the study said. The scientists did detect a small hint of DNA from single-celled organisms called archaea if they "forced the conditions" in those samples, López-García said. That means they took the sample and kept amplifying

the DNA — imagine zooming into a picture — to see if there was a very small quantity that they'd missed. But the researchers hypothesized that this small amount of DNA is likely the result of contamination from a neighboring salt plain, brought from people who visit the area or wind.

On the other hand, in the less extreme ponds, the researchers found a large diversity of microbes, again mostly archaea. "The diversity of archaea is really very, very large and very surprising," López-García said. Researchers found some archaea that are well known to live in areas of high salt concentration and some that the scientists had no idea could survive in even the relatively less-salty ponds.

Their findings suggest that there is a gradient of extreme environments, some of which harbor life and others that don't and might serve as a bit of a caution in the search for life elsewhere in the cosmos, she added. "There is this idea … that says any planet with liquid water on the surface is habitable," she said. But as the lifeless pools of Ethiopia may suggest, water "might be a necessary condition, but it is far from sufficient."

What's more, using electron microscopes, the researchers also detected the presence of biomorphs or "mineral precipitates that can mimic tiny cells" in samples taken from both the lifeless pools and those found to harbor life, López-García said. "If you go to Mars or to fossil environments and you see little, rounded things, you might be tempted to say that these are microfossils, but they might not be."

Proving that life doesn't exist

There were some weaknesses in this study, John Hallsworth, a lecturer at The Institute for Global Food Security at Queen's University Belfast in Northern Ireland wrote in an accompanying commentary published in the journal Nature Ecology & Evolution.

For example, the researchers' DNA analysis couldn't determine if the detected organisms were alive or active, and it's unclear if their measurements of the water factors such as pH were done accurately, he wrote.

Even so, the team "managed to characterize the geochemistry and microbial diversity of a large number of brines that span a wide range of physicochemical conditions, revealing the extensive diversity of the archaeal communities present," Hallsworth wrote.

What's more, a couple of months ago, another group of researchers came up with the opposite conclusion after they, too, sampled the waters in the Dallol area. In the most extreme ponds of the area, those researchers found that archaea were "thriving," and various types of analysis suggested that these microorganisms didn't originate from any type of contamination, said Felipe Gómez, a biochemist at Spain's Center of Astrobiology and the lead author of that study, which was published in May in the journal Scientific Reports.

"Given the risk of detecting any type of contamination, microbiologists that work in extreme environments take many precautions to avoid it," he said. "In our work, we sampled in completely aseptic conditions," or those free from contamination.

It's unclear why there is a discrepancy between the studies, and though "they claim that they do not see what we report," that doesn't mean the older findings are incorrect, he said. "More work needs to be done." But this older paper is "weak" because the researchers only found traces of one type of archaea that's similar to archaea living in the neighboring salt plain, and didn't do enough to prevent contamination, López-García said.

"Dispersal is active in the area," so this trace of archaea could have been carried in by the wind or tourists, similar to how her team also discovered traces of archaea but hypothesized that they were contaminants from the neighboring salt plain, she said.

The new findings were published on Oct. 28 in the journal Nature Ecology & Evolution.

http://bit.ly/2CSkyVc

Extinct giant ape directly linked to the living orangutan

Protein sequencing retrieves ancient genetic information from primates living in subtropics almost two million years ago

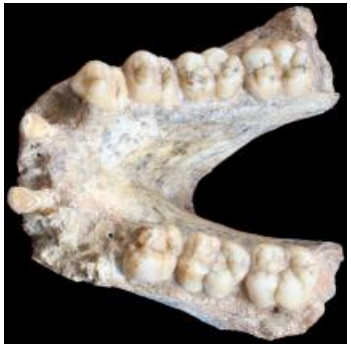
By using ancient protein sequencing, researchers have retrieved genetic information from a 1.9 million year old extinct, giant primate that used to live in a subtropical area in southern China. The genetic information allows the researchers to uncover the evolutionary position of Gigantopithecus blacki, a three-meter tall and possibly 600 kg primate, revealing the orangutan as its closest living relative.

Artistic representation of Gigantopithecus blacki. **Credit: Ikumi Kayama (Studio Kayama LLC)**

It is the first time that such old genetic material has been retrieved from a warm, humid environment. The study is published in the scientific journal *Nature*, and the results are groundbreaking within the field of evolutionary biology, according to Frido Welker, Postdoc at the Globe Institute at the Faculty of Health and Medical Sciences and first author of the study.

"Primates are relatively close to humans, evolutionary speaking. With this study, we show that we can use protein sequencing to retrieve ancient genetic information from primates living in subtropical areas even when the fossil is two million years old. Until now, it has only been possible to retrieve genetic information from up to 10.000-year-old fossils in warm, humid areas. This is interesting, because ancient remains of the supposed ancestors of our species, Homo sapiens, are also mainly found in subtropical areas, particularly for the early part of human evolution. This means that we can potentially retrieve similar information on the evolutionary line leading to humans," says Frido Welker.

Today, scientists know that the human and the chimpanzee lineages split around seven or eight million years ago. With the previous methodologies though, they could only retrieve human genetic information not older than 400.000 years. The new results show the possibility to extend the genetic reconstruction of the evolutionary relationships between our species and extinct ones further back in time, at least up to two million years—covering a much larger portion of the entire human evolution.



A Gigantopithecus blacki mandible (P1-M2=74mm). **Credit: Prof. Wei Wang; Photo retouching: Theis Jensen.**

Analyzing ancient dental enamel proteins using mass spectrometry-based proteomics

In a recent study, also published on *Nature*, Enrico Cappellini, Associate Professor at the Globe Institute and senior author on this study, initially demonstrated, together with an international team of colleagues, the massive potential of ancient protein sequencing.

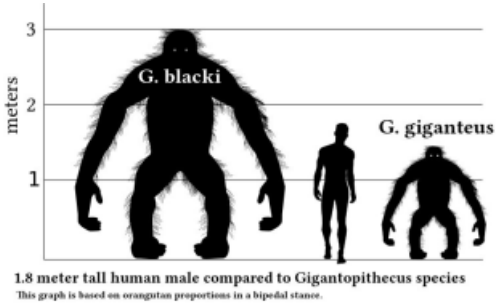
"By sequencing proteins retrieved from dental enamel about two million years old, we showed it is possible to confidently reconstruct the evolutionary relationships of animal species that went extinct too far away in time for their DNA to survive till now. In this study, we can even conclude that the lineages of orangutan and Gigantopithecus split up about 12 million years ago," says Enrico Cappellini.

Sequencing protein remains two million years old was made possible by stretching to its limits the technology at the base of proteomic discovery: mass spectrometry. State of the art mass spectrometers and the top palaeoproteomics expertise needed to get

the best out of such sophisticated instrumentation are key resources deriving from the decade-long strategic collaboration with Jesper Velgaard Olsen, Professor at Novo Nordisk Foundation Center for Protein Research and co-author on this study.

The mystery of Gigantopithecus

The fossil evidence attributed to Gigantopithecus was initially discovered in southern China in 1935, and it is currently limited to just a few lower jaws and lots of teeth. No complete skull and no other bone from the rest of the skeleton has been found so far. As a result, there has been a lot of speculation about the physical appearance of this mysterious animal.



This is a comparison graph comparing the height of a 1.8 meter tall human male with Gigantopithecus species. This graph is based on orangutan proportions in a bipedal stance. It is most likely that Gigantopithecus would have spent most of its time in a quadrupedal stance on all fours. **Credit: Discott**

"Previous attempts to understand which could be the living organism most similar to Gigantopithecus could only be based on the comparison of the shape of the fossils with skeletal reference material from living great apes. Ancient DNA analysis was not an option, because Gigantopithecus went extinct approximately 300.000 years ago, and in the geographic area Gigantopithecus occupied no DNA older than approximately 10.000 years has been retrieved so far. Accordingly, we decided to sequence dental enamel proteins to reconstruct its evolutionary relation with living great apes, and we found that orangutan is Gigantopithecus' closest living relative," says Enrico Cappellini.

The study of human evolution by palaeoproteomics will continue in the next years through the recently established "Palaeoproteomics to Unleash Studies on Human History (PUSHH)" Marie Skcodowska Curie European Training Network (ETN) Programme. The article, "Enamel proteome shows that Gigantopithecus was an early diverging pongine," has just been published in *Nature*.

More information: *Enamel proteome shows that Gigantopithecus was an early diverging pongine, Nature (2019). DOI: 10.1038/s41586-019-1728-8 , https://www.nature.com/articles/s41586-019-1728-8*

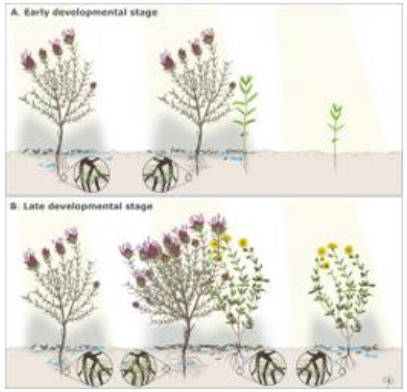
Journal information: Nature *Provided by University of Copenhagen*

http://bit.ly/37cVB4F

Plants might be helping each other more than thought

In harsh environments mature plants help smaller ones - and thrive as a result

Contrary to the long-held belief that plants in the natural world are always in competition, new research has found that in harsh environments mature plants help smaller ones - and thrive as a result.



The first study to examine plant interactions in a hostile environment over their lifespan found that plants sheltering seedlings help the smaller plant survive and are more successful themselves, a processed in ecology called facilitation.

Scientific illustration created by Dr I M Berenjeno (DhramaBeren Studio, www.dharmaberen.com)

The study, led by Dr Rocio Pérez-Barrales at the University of Portsmouth and Dr Alicia Montesinos-Navarro at Desertification Research Center in Valencia, Spain, studied adult and seedling plants in the 'ecological desert' of gypsum soil in the south-east of Spain. The findings could have significance for those managing harsh environments including coastal management.

Dr Pérez-Barrales said: "If you're a seedling in a barren landscape - the top of a mountain or a sand dune, for example - and you're

lucky enough to end up underneath a big plant, your chances of survival are certainly better than if you landed somewhere on your own.

"What we have found which was surprising is an established large plant, called a 'nurse', shields a seedling, it also produces more flowers than the same plants of similar large size growing on their own." This win-win for adult and seedling plants in harsh environments has not previously been reported.

"Scientists have often looked at such plant relationships and found an adult or a seedling at one stage of its life, and made conclusions," Dr Pérez-Barrales said. "But by studying these plants' entire lifespan, from seed germination and establishment, growth of young plants, and flowering in adult plants, we have evidence that the benefits for both stack up over time."

Dr Pérez-Barrales and her all-female team of scientists studied plant growth in southern Spain over three months during summer. The plants were growing in gypsum, a very poor soil, with little nutrients or water. They found clear evidence the seedling and nurse were more likely to thrive when grown together, compared to either plant growing alone.

The seedling benefited from shade, more moisture and more nutrients, from the leaf litter of the 'nurse' plant, and probably higher bacteria and fungi in the soil, among other things. As it matured, the 'nurse' plant grew more flowers than similar plants nearby growing alone, greatly increasing her chances of producing seeds and propagating.

Other benefits of nurse-seedling partnerships include that more variety of plants growing together can trigger a positive cascade effects in the environment. For example, vegetation patches with nurse and facilitated plants with more flower density might be able to attract higher numbers and diversity of pollinators in an area, in turn supporting insect and soil life, and even provide a greater range of different fruit types for birds and mammals.

"The biggest winner for this system of nursing a plant is biodiversity," Dr Pérez-Barrales said. "The more biodiverse an area, the more we have a greater number of species of plants, insect life, bacteria, fungi, mammals and birds, the better the chances are of long-term healthy functioning of the environment and ecosystems."

The research is likely to be of value to those who manage and protect plants in hostile and harsh environments, such as shingle and sand dunes ecosystems, both of which encircle the UK and are considered at high risk due to human intervention and climate change.

Most home gardeners and arable farmers plan to ensure their soil and conditions are the best they can be for optimum plant growth, but the findings might be of value to those who garden in inhospitable places.

Dr Pérez-Barrales suggested gardeners experiment with planting different species of different ages together to test which partnerships help plants thrive in any particular location.

http://bit.ly/2NVPq5O

Hair-raising truth behind pigeons' lost toes

Next time you visit your hairdresser spare a thought for the pigeons.

For a long time scientists thought the fact that pigeons in urban environments often lost their toes was due to some form of infection, or was a reaction to chemical pollutants.

But now researchers in France believe they've stumbled upon the real culprit: human hair.

The team from the National Museum of Natural History and the University of Lyon recorded the occurrence and extent of toe mutilations from pigeons eking out their time in 46 sites across Paris.

The found that human pollution likely played a part in nearly all cases of missing toes—pigeons living in areas with higher rates of air and noise pollution tended to have fewer digits than those that lived in leafier environs.

Perhaps most strikingly, the team noticed that toe mutilation "tended to increase with the density of hairdressers"—meaning the poor birds often lose their extremities by getting them entangled in human hair.

The team suggested that more green spaces might benefit the population of birds seen by many city-dwellers as pests.

But they do in fact serve a worthy purpose for science.

"Measuring the impacts of urban pollution on biodiversity is important to identify potential adaptations and mitigations needed for preserving wildlife even in city centres," the team wrote.

The study was published in the journal *Biological Conservation*.

http://bit.ly/2QqZJrS

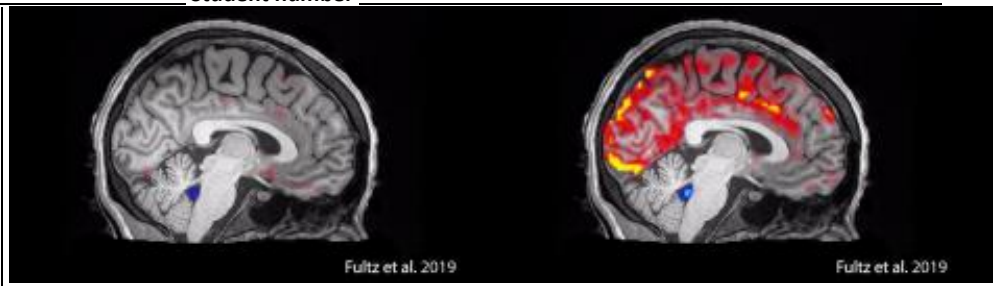
Watch Spinal Fluid 'Wash' the Sleeping Brain in Rhythmic, Pulsing Waves

What happens to your brain as you sleep? A new video holds the answer: A juicy mix of blood and cerebrospinal fluid slosh through your smushy noggin in a rhythmic pulsating dance.

By Stephanie Pappas - Live Science Contributor

The movement appears almost tidal in a video released Oct. 31 along with an article in the journal Science. Though researchers knew that brain activity takes on a rhythmic pattern during sleep, this video and study mark the first time anyone has observed a similarly rhythmic flow of cerebrospinal fluid.

"We've known for a while that there are these electrical waves of activity in the neurons," study co-author Laura Lewis, a professor of biomedical engineering at Boston University, said in a statement. "But before now, we didn't realize that there are actually waves in the CSF, too."



During sleep, waves of oxygenated blood (red) and then cerebrospinal fluid (blue) wash over the brain. **(Image: © Laura Lewis])**

Cerebrospinal fluid is a clear fluid that surrounds and cushions the brain and spinal cord. It circulates in the meninges, or the casing, that surrounds the brain. Research has suggested that one of the jobs of cerebrospinal fluid is to cleanse the brain of toxic proteins during sleep. The new video might show the CSF doing just that.

Lewis and her colleagues captured images of sleeping brains by having 13 research participants, all in their 20s and 30s, fall asleep in a magnetic resonance imaging (MRI) machine. The participants, who were paid for this uncomfortable arrangement, also had to wear a net of electrodes on their scalps to measure the electrical activity of their brains.

The results showed a pulsing, predictable flow. First, neural activity quiets. Then, blood flows out of the brain. Next, cerebrospinal fluid flows in. Lather, rinse, repeat. The pattern is so consistent that it's possible to just look at cerebrospinal fluid in a given brain region and tell whether a person is awake or asleep, Lewis said.

The findings may be a new insight into brain-related problems of aging. Toxic proteins are implicated in Alzheimer's disease and other dementias. These dementias are often associated with disrupted sleep, and atypical sleep patterns might be linked to a greater risk of developing dementia. Both slow-wave sleep — a phase of deep sleep during which dreaming occurs — and cerebrospinal fluid have been associated with the cleansing of the

brain. The new research suggests that the brain activity and the fluid flow are linked.

It's not yet clear exactly how or why the neural activity, blood flow and cerebrospinal fluid remain so in sync. Lewis and her colleagues speculate that when brain activity drops, the neurons need less oxygenation, so blood flow declines. The cerebrospinal fluid might then rush in to maintain pressure in the brain and prevent brain damage. That's only speculation, though, Lewis said. More studies of the brain's nocturnal cadences are needed to understand the cleansing flow.

http://bit.ly/33WZyZ1

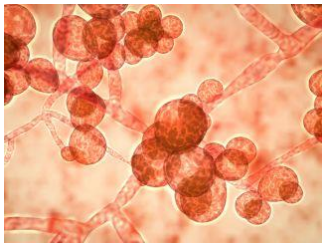
These Two Drug-Resistant Microbes Are New 'Urgent Threats' to Americans' Health

A new report reveals that drug-resistant germs infect and kill more people than previous estimates suggested.

By Nicoletta Lanese - Staff Writer 4 days ago Health

The U.S. faces two new urgent threats to public health: a couple of drug-resistant germs called *Candida auris* and *Acinetobacter*, health officials announced today.

These microbes have built up resilience against the drugs designed to kill them, meaning they can be incredibly dangerous and difficult to treat. In fact, drug-resistant bacteria and fungi may pose a greater threat to American health than previous estimates suggested, according to a report released today (Nov. 13) by the Centers for Disease Control and Prevention (CDC).



***The Candida auris fungus has been called an "urgent threat" to public health.* (Image: © Shutterstock)**

A previous report, published in 2013, estimated that at least 2 million people in the U.S. get an antibiotic-resistant infection each year and that at least 23,000 people die from these infections. For the new report, CDC officials recrunched these numbers.

They found that closer to 2.6 million drug-resistant infections likely occurred at the time of the last report, resulting in nearly 44,000 deaths — nearly double the previous estimate.

"We knew and said [in 2013] that our estimate was conservative … and we were right," Michael Craig, a senior adviser for the CDC Antibiotic Resistance Coordination and Strategy Unit, said during a news conference today. Drawn from "millions and millions" of electronic records from 700 hospitals, along with other new data sources, the 2019 report provides a clearer picture of the danger that drug-resistant bugs pose to the nation's health and global security, Craig said.

Today, drug-resistant bacteria and fungi cause more than 2.8 million infections and 35,000 deaths across the country annually. This represents a roughly 18% decrease in deaths from these infections overall since 2013 and a 30% decrease in deaths that occur in hospitals.

But to put those numbers into perspective, someone in the U.S. catches a drug-resistant infection every 11 seconds, and someone dies as a result of these infections about every 15 minutes, according to a CDC statement.

"Despite significant progress, this threat remains our enemy," Dr. Robert Redfield, CDC director, said during the news conference.

A fungus called *Candida auris* is among the most dangerous of these microbes, the report noted. "It is a pathogen that we didn't even know about when we wrote our last report in 2013, and since then, it's circumvented the globe," Craig said. The fungus appeared to crop up on five continents, simultaneously, and kills 1 in 5 people who become infected with it, Redfield said. Some infections appear to be resistant to all three classes of medications designed to treat it.

The bacterium *Acinetobacter*, also new to the "urgent threat" list, caused an estimated 8,500 infections in hospitalized patients and 700 estimated deaths in the U.S. in 2017, according to the CDC. Infections with this bacterium often arise in health care settings and appear resistant to multiple antibiotics, the report noted.

In addition, 223,900 cases of infection with *Clostridioides difficile* bacteria occurred in 2017 and claimed at least 12,800 lives. Though not typically antibiotic-resistant, these bacteria often infect people who are currently taking or have recently stopped taking antibiotics.

Drug-resistant gonorrhea infections are also on the rise, with most bacteria showing resistance to all but one class of antibiotics. Another resistant group of bugs, known as ESBL-producing Enterobacteriaceae (which includes *E. coli*), represents one of the leading causes of death among resistant infections and seriously complicate urinary tract infections in women. In addition, antibiotic-resistant infections with group A strep bacteria quadrupled since the 2013 report, and the death count will rise if serious measures aren't taken now, officials said.

"The good news is, we know how we can protect ourselves from this threat," Redfield said.

The noted decline in deaths from drug-resistant infections likely resulted from a number of nationwide initiatives to prevent the spread of germs, to contain emerging pathogenic threats, and to improve how doctors, patients and food producers use antibiotics. The CDC and the Food and Drug Administration also supply microbe samples to drug developers so they may design alternative treatments and better diagnostics for these pathogens.

Everyone can help prevent the spread of antibiotic resistance by taking antibiotics only as prescribed, Craig said. People can also help by maintaining good hygiene and washing their hands regularly, making sure to cook meat adequately and practicing safe sex (given that some antibiotic-resistant infections, such as gonorrhea, can be transmitted sexually).

In addition, Redfield encouraged people to make sure they're up to date on vaccinations, which reduces the rates of infection in general and thus can decrease antibiotic use in sick people. (Most antibiotic-resistant bacteria, with the exception of *Streptococcus pneumoniae*, do not yet have a specific vaccine to prevent the infection.)

"Bacteria and fungi will continue to develop resistance to drugs designed to kill them," Redfield said. "The report further underlines that this threat isn't going away."

http://bit.ly/2KtFtC5

If Dr Google's making you sick with worry, there's help

It's a busy day at the office and your left eye has been twitching uncontrollably. So, out of curiosity and irritation you Google it.

Jill Newby * **Eoin McElroy** **

Various benign causes — stress, exhaustion, too much caffeine — put your mind at ease initially. But you don't stop there. Soon, you find out eye twitches could be a symptom of something more sinister, causing you to panic.

You ruin the rest of the day trawling through web pages and forums, reading frightening stories convincing you you're seriously ill.

For many of us, this cycle has become common. It can cause anxiety, unnecessary contact with health services, and at the extreme, impact our day-to-day functioning.

But our recently published research, the first to evaluate online therapy for this type of excessive and distressing health-related Googling, shows what can help.

I've heard of 'cyberchondria'. Do I have it?

The term "cyberchondria" describes the anxiety we experience as a result of excessive web searches about symptoms or diseases. It's not an official diagnosis, but is an obvious play on the word

"hypochondria", now known as health anxiety. It's obsessional worrying about health, online.

Some argue cyberchondria is simply a modern form of health anxiety. But studies show even people who don't normally worry about their health can see their concerns spiral after conducting an initial web search.

Cyberchondria is when searching is:

- **excessive:** searching for too long, or too often
- **difficult to control:** you have difficulty controlling, stopping or preventing searching
- **distressing:** it causes a lot of distress, anxiety or fear
- **impairing:** it has an impact on your day-to-day life.

If this sounds like you, there's help.

We tested an online therapy and here's what we found

We tested whether an online treatment program helped reduce cyberchondria in 41 people with severe health anxiety. We compared how well it worked compared with a control group of 41 people who learned about general (not health-related) anxiety and stress management online. The online treatment is based on cognitive behaviour therapy (CBT), which involves learning more helpful ways of thinking and behaving.

Participants completed six online CBT modules over 12 weeks, and had phone support from a psychologist.

The treatment explained how excessive web searching can become a problem, how to search about health effectively, and practical tools to prevent and stop it (see a summary of those tips below).

We found the online treatment was more effective at reducing cyberchondria than the control group. It helped reduce the frequency of online searches, how upsetting the searching was, and improved participants' ability to control their searching. Importantly, these behavioural changes were linked to improvements in health anxiety.

Although we don't know whether the program simply reduced or completely eliminated cyberchondria, these findings show if you're feeling anxious about your health, you can use our practical strategies to reduce anxiety-provoking and excessive online searching about health.

So, what can I do?

Here are our top tips from the treatment program:

- **be aware of your searching**: don't just search on auto-pilot. Take note of when, where, how often, and what you are searching about. Keep track of this for several days so you can spot the warning signs and high-risk times for when you're more likely to get stuck in excessive searching. Then you can make a plan to do other things at those times
- **understand how web searches work**: web search algorithms are mysterious beasts. But top search results are not necessarily the most likely explanation for your symptoms. Top search results are often click-bait – the rare, but fascinating and horrific stories about illness we can't help clicking on (not the boring stuff)
- **be smart about how you search:** limit yourself to websites with reliable, high quality, balanced information such as government-run websites and/or those written by medical professionals. Stay away from blogs, forums, testimonials or social media
- **challenge your thoughts by thinking of alternative explanations for your symptoms:** for example, even though you think your eye twitch might be motor neuron disease, what about a much more likely explanation, such as staring at the computer screen too much
- **use other strategies to cut down, and prevent you from searching:** focus on scheduling these activities at your high-risk times. These can be absorbing activities that take your focus and

can distract you; or you can use relaxation strategies to calm your mind and body

- **surf the urge:** rather than searching straight away when you feel the urge to search about your symptoms, put it off for a bit, and see how the urge to search reduces over time.

And if those don't help, consult a doctor or psychologist.

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Disclosure statement

Jill Newby receives funding from the Australian Medical Research Future Fund (MRFF). She is affiliated with UNSW Sydney, and the Clinical Research Unit for Anxiety and Depression (CRUfAD), which operates THIS WAY UP online treatment service. She is a member of the Australian Association for Cognitive Behavioural Therapy.

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

http://bit.ly/2CNewoC

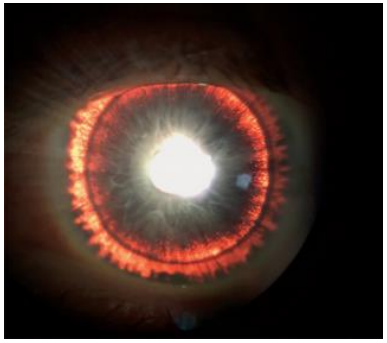
Man's 'Glowing' Iris Was a Sign of Rare Eye Syndrome

The man's eye exam showed something odd.

By Rachael Rettner - Senior Writer 3 days ago Health

A man's eye exam showed something unusual: His iris appeared to be "glowing."

This eerie appearance turned out to be a sign of a rare disorder that caused his eye pigment to flake off, according to a new report of the case.



A man's eye exam revealed an eerie "glow" in his iris, which was a sign of a rare disorder that caused his eye pigment to flake off. **(Image: © The New England Journal of Medicine ©2019)**

The 44-year-old man went to an eye clinic after he moved to a new area and wanted to get set up with an eye doctor there, according to the report, published today (Nov. 13) in The New England Journal of Medicine.

He said that he had a family history of glaucoma, an eye disease that can damage the optic nerve, the bundle of nerve fibers that connects the back of the eye to the brain. This damage is typically caused by increased pressure in the eye, and indeed, the man had already been diagnosed with high eye pressure and was taking medication to reduce it, according to the report authors, from the University of Texas Medical Branch at Galveston.

Still, tests found that the pressure in his eye was very slightly above the normal range.

What's more, when the doctor performed an eye exam, using a microscope and a bright light to view the eye, the evaluation revealed "iris transillumination" in both of the patient's eyes, the report said. In other words, light was shining through the iris, or the colored part of the eye. This happens when sections of pigment are missing from the iris, which allows light to pass through, according to the National Institutes of Health.

The man was diagnosed with pigment dispersion syndrome. In this eye condition, pigment rubs off the back of the iris, according to the American Academy of Ophthalmology (AAO). These flakes of pigment can clog the eye's drainage system, resulting in increased eye pressure, which in turn can lead to glaucoma.

Pigment dispersion syndrome is rare, although it is more commonly diagnosed in men in their 20s and 30s and may be inherited, the AAO says.

In this case, the man underwent laser therapy to open the eyes's clogged drainage channels. This therapy helps fluid flow out of the eye and reduces eye pressure, the AAO says. However, patients often need to continue taking pressure-reducing eye medications after the surgery, as was the case for this patient, the case report said.

http://bit.ly/35q0HeN

Link between hearing and cognition begins earlier than once thought

A new study found lower levels of cognitive function even in people whose hearing was slightly impaired from age but was still considered "normal."

NEW YORK, NY - Research has shown that adults with age-related hearing loss have higher rates of cognitive decline. Now, a study from researchers at Columbia University Vagelos College of Physicians and Surgeons has found that even the earliest stage of hearing loss--when hearing is still considered normal--is linked to cognitive decline.

The study was published online today in *JAMA Otolaryngology-Head and Neck Surgery*.

Link Between Hearing Loss and Cognitive Impairment

Age-related hearing loss is one of the most common health disorders of aging, affecting two-thirds of those over age 70. However, few adults are tested for hearing loss, and even fewer are treated. Only 14% of adults with hearing loss in the United States wear hearing aids, the standard treatment.

Because studies show people with age-related hearing loss are more likely to have impaired cognition, it is thought that hearing loss may trigger cognitive decline.

But these studies have only examined people diagnosed with hearing loss, which is defined as the inability to hear sounds under 25 decibels (dB).

"Physicians in this field have used 25 dB--about the loudness of a whisper--to define the border between normal hearing and mild hearing loss in adults, but this level is arbitrary," says Justin S. Golub, MD, MS, assistant professor of otolaryngology-head and neck surgery at Columbia University Vagelos College of Physicians and Surgeons and a hearing specialist at Columbia University Irving Medical Center and NewYork-Presbyterian Hospital. "It has been assumed that cognitive impairment wouldn't begin until people passed this threshold. But no one actually looked at whether this was true."

Any Hearing Loss May Be Cause for Concern

The researchers looked at data from 6,451 adults (average age 59) who were enrolled in two ethnically diverse epidemiologic studies. Participants underwent hearing and cognitive testing as part of the studies.

Golub and his colleagues found that for every 10 dB decrease in hearing, there was a significant decrease in cognitive ability, a pattern seen across the entire spectrum of hearing.

Surprisingly, the largest decrease in cognitive ability occurred in those whose hearing was just starting to become impaired, just 10 dB off the perfect mark.

"Most people with hearing loss believe they can go about their lives just fine without treatment, and maybe some can," says Golub. "But hearing loss is not benign. It has been linked to social isolation, depression, cognitive decline, and dementia. Hearing loss should be treated. This study suggests the earlier, the better."

Can Hearing Aids Prevent Cognitive Loss?

The current study did not address whether hearing loss causes cognitive impairment. It is possible that early declines in both hearing and cognitive performance are related to common aging-related processes, the researchers noted.

"But it's also possible that people who don't hear well tend to socialize less and, as a result, they have fewer stimulating conversations. Over many years, this could have a negative impact on cognition," says Golub. "If that's the case, preventing or treating hearing loss could reduce dementia incidence--by more than 9%, according to a recent analysis published in The Lancet."

A new study, funded by the National Institutes of Health, is now testing the possibility that hearing aids can slow cognitive decline in older people with age-related hearing loss.

More studies are needed before recommending changes in hearing loss categories. "One possibility is to formally introduce a new category, such as borderline hearing loss, ranging from 16 to 25 dB of hearing ability," says Golub.

The study is titled, "Association of Subclinical Hearing Loss With Cognitive Performance." The other contributors include: Adam M. Brickman (Columbia University Irving Medical Center, New York, NY), Adam J. Ciarleglio (George Washington University, Washington, DC), Nicole Schupf (CUIMC), and Jose A. Luchsinger (CUIMC).

The research was supported by grants from the National Institutes of Health (K23AG057832, L30AG060513, and K24AG045334).

Dr. Golub reports receiving travel expenses for industry-sponsored meetings from Cochlear, Advanced Bionics, and Oticon Medical; consulting fees from Oticon Medical, and Auditory Insight, Optinose, and Decibel Therapeutics; honoraria from Abbott; and departmental educational grants from 3NT, Storz, Stryker, Acclarent, and Decibel Therapeutics.

http://bit.ly/341AG2E

New study casts doubt on China's organ donation data

***The Chinese government may have been systematically misreporting the number of organs it claims it has voluntarily collected since 2010, according to new research published in* BMC Medical Ethics.**

In 2015 China promised the world they would no longer source organs from prisoners - their almost sole source previously.

The study, led by PhD scholar Matthew Robertson from The Australian National University (ANU), used statistical forensics on official Chinese datasets.

"Our research shows Beijing's reported organ donation numbers don't stack up and there is highly compelling evidence that they are being falsified," Mr Robertson said. "The figures appear to have been based on a simple mathematical formula, a quadratic function, which would be familiar to many high school students.

"When you take a close look at the numbers of organs apparently collected they almost match this artificial equation point for point, year in, year out. They're too neat to be true. "These figures don't appear to be real data from real donations. They're numbers generated using an equation. "It is difficult to imagine how this model could have been arrived at by mere chance, raising the distinct possibility that it was intended to deceive."

The study looked at data on voluntary hospital-based donated organs between 2010 and 2018 published by the China Organ Transplant Response System and the Red Cross Society of China*.

"We found major anomalies with the datasets, with implausibly high ratios of transplants per donor and mismatches when the two sets of data were meant to be identical," Mr Robertson said.

"Provincial and hospital-level data we examined also showed anomalies that are extremely difficult to explain.

"The implication is that it is highly likely the numbers the Chinese government have put out were not actually real figures created by actual organ donations, but instead generated by a simple mathematical formula."

The researchers also found the misclassification of non-voluntary organ donors as voluntary. "This is all highly suggestive evidence of data manufacturing and manipulation that could only have been done by human intervention," Mr Robertson said.

"The patterns we observed in the data can only be plausibly explained by the falsification of official organ transplant figures."

The China Organ Transplant Response System (COTRS) forms the basis of China's current voluntary organ donation reforms, with every organ transplant allocated solely through it. The Red Cross Society of China is mandated to verify and witness every such organ donation. While data from COTRS is not usually publicly available, data from it has been published twice - in 2014 and in 2017.

Data from the Red Cross Society of China was previously available on four websites, with three of the websites recently taken offline. The dataset is currently available at http://www.codac.org.cn.

The researchers' findings have been reviewed by one of the world's leading statisticians - Sir David Spiegelhalter, former president of the Royal Statistical Society in the UK.

"The anomalies in the data examined...follow a systematic and surprising pattern," Spiegelhalter wrote.

"The close agreement of the numbers of donors and transplants with a quadratic function is remarkable and is in sharp contrast to other countries who have increased their activity over this period... I cannot think of any good reason for such a quadratic trend arising naturally."

Mr Robertson and his team's research comes just months after the findings of the China Tribunal, led by the former UN war crimes prosecutor Sir Geoffrey Nice QC. The tribunal concluded that "in China forced organ harvesting from prisoners of conscience has been practiced for a substantial period of time involving a very substantial number of victims".

Mr Robertson said the study's findings were globally significant.

"China's much-heralded organ transplant reform program was supposed to be the culmination of over a decade of international pressure, where finally they were reforming and ceasing the use of organs from prisoners," Mr Robertson said.

"As a result of these promises to reform, the same Chinese officials who promulgated this data were welcomed into the World Health Organization's transplantation task force, and Chinese surgeons began presenting in medical conferences again. "Now we have found that the data was simply made up, based on an equation.

"With what our study shows, we think it is important the world take a closer look at China's organ transplantation system."

**Not affiliated with international aid organisation Red Cross*

http://bit.ly/33UXbWV

Researchers block metastasis-promoting enzyme, halt spread of breast cancer

Findings in mice point to new approach that may help make some metastatic breast cancers susceptible to immunotherapy

In a breakthrough with important implications for the future of immunotherapy for breast cancer, UC San Francisco scientists have found that blocking the activity of a single enzyme can prevent a common type of breast cancer from spreading to distant organs.

While studying a mouse model that replicates key features of early-stage human breast cancer, the researchers discovered that a ubiquitous enzyme called MMP9 is an essential component of the cancer's metastasis-promoting machinery, helping to create a hospitable environment for itinerant cancer cells to form new metastatic tumors.

"Metastasis is the biggest hurdle when it comes to successfully treating breast cancer, and solid tumors in general," said Vicki Plaks, PhD, now an assistant adjunct professor in the Department of Orofacial Sciences at UCSF. "Once a cancer becomes metastatic, there's really no cure, and the only option is to manage it as a chronic disease."

Plaks co-led the team that made the discovery when still a postdoctoral fellow in the laboratory of Zena Werb, PhD, a professor of anatomy and associate director for basic science at the UCSF Helen Diller Family Comprehensive Cancer Center.

When they examined lung tissue in their mouse model, the researchers found that MMP9 is involved in remodeling healthy tissue and transforming it into a kind of safe haven for migrating breast cancer cells. When the cancer cells colonize these sites with the help of MMP9, they're able to start growing into new tumors.

The new study, published Nov. 14 in the journal *Life Science Alliance*, shows that these metastases can be stopped before they are able to lay the foundations for tumor growth.

By administering an antibody that specifically targets and disrupts MMP9 activity, the scientists were able to prevent cancer from colonizing the lungs of mice. But interestingly, interfering with MMP9 had no effect on the primary tumor, which suggests that the enzyme's primary role in this scenario is helping existing malignancies metastasize and colonize other organs rather than promoting the growth of established primary tumors.

Prior to this study, Werb and others had found that MMP9 plays an important role in remodeling the extracellular matrix (ECM) -- a patchwork of biomolecules found outside of cells that provides structure and shape to organs, helps cells communicate with one another, and establishes a microenvironment that promotes cell health, among its many other functions.

Although MMP9 was known to be involved in cancer, specifically in remodeling the ECM to build tumor niches that are hospitable to malignancies, its role in the earliest stages of metastasis had not been fully explored.

"Lots of studies that examined metastatic niche formation in breast cancer have focused on late-stage cancers, when the tumors are fairly progressed. What sets our study apart is that we chose to focus on processes that alter the tumor and metastatic microenvironment early on. This approach enabled us to show that MMP9 really matters in the early stages," said Mark Owyong, co-lead author of the new study with Jonathan Chou, MD, PhD, a clinical fellow in the UCSF School of Medicine. Owyong, Chou and Plaks conducted the research as members of the Werb lab.

The first hint that MMP9 might be involved in early-stage metastasis came from publicly available gene expression data from clinical breast cancer biopsies. While sifting through this data, the researchers noticed that MMP9 levels were elevated in metastatic disease.

To further investigate MMP9's role in metastasis, the researchers turned to a unique mouse model of "luminal B" breast cancer, which is among the most frequently diagnosed forms of the disease. "We selected the model because it's one of the few that captures the natural progression of breast cancer, closely mimicking the progression of the disease experienced by patients," Owyong said.

In a key set of experiments, the researchers injected tumor cells into mice that had early stage breast cancer but no discernible metastases. They found that the cells colonized the lungs and formed new tumor growth sites. But when these cells were injected into genetically identical mice without breast cancer, no metastases formed.

When the experiment was repeated in mice with early stage breast cancer whose MMP9 gene had been knocked out, there was a significant reduction in the size of metastatic lung tumors, though there was no effect on the primary breast tissue tumor. These findings suggest that MMP9 is required to promote metastasis, but not essential for continued growth of the primary tumor.

Similar results were seen when the researchers disrupted the activity of MMP9 with a unique antibody that specifically targets the activated form of the enzyme. The researchers injected tumor cells into these mice, followed by injections of the antibody every two days. At the end of the treatment regimen, the researchers inspected the mice and saw a significant reduction in the number and size of lung metastases in mice who received the antibody compared with those that didn't.

"This was a very promising result and suggests that a therapeutic paradigm focused on intercepting metastasis early might offer a new route for treating certain kinds of breast cancer," said Plaks.

The researchers also discovered that interfering with MMP9 activity helped recruit and activate cancer-fighting immune cells to metastatic sites, a result with important implications for treating certain types of metastatic breast cancer with immunotherapy.

Immunotherapies work by enlisting the body's immune system to find and kill cancer cells. But certain cancers -- including luminal B breast cancer, the main focus of the new study -- don't succumb to immunotherapy.

According to Plaks, this is because, beyond their direct effects on metastatic growth, enzymes like MMP9 also play an important role in remodeling the ECM and building mesh-like barriers around metastatic sites that help to exclude immune cells. This may explain why some metastatic cancer cells are able to evade the immune onslaught triggered by immunotherapies.

But the new study shows that when MMP9 is incapacitated, metastatic sites may no longer be able to keep immune cells at bay. Plaks thinks that this represents an important step towards making breast cancer more susceptible to immunotherapies that have proven effective against other forms of cancer.

"These findings come at an exciting time in cancer immunology, with antibodies targeting MMP9 being actively explored for clinical use within the biotech industry," Plaks said. "There's been great interest in trying to use immunotherapy to treat metastatic breast cancers of the luminal B type, but so far, success has been limited. Our work indicates that a combination approach of immunotherapy with antibodies targeting MMP9 activity might actually succeed."

Authors: Additional authors include Renske JE van den Bijgaart, Niwen Kong, Gizem Efe, Carrie Maynard, Charlotte Koopman, Elin Hadler-Olsen, Mark Headley, Charlene Lin and Chih-Yang Wang from UCSF, and Dalit Talmi-Frank and Inna Solomonov from the Weizmann Institute of Science.

Funding: This study was supported by a Department of Defense Postdoctoral Fellowship, National Cancer Institute grants R01 CA057621 and U01 CA199315, the Parker Institute for Cancer Immuno- therapy, and funds from the Israel Science Foundation.

Disclosures: The authors declare that they have no conflict of interest.

http://bit.ly/2CRBwmp

Star Ejected from Milky Way's 'Heart of Darkness' Reaches Mind-Blowing Speed

"It is a visitor from a strange land."

By Chelsea Gohd - Space.com 3 days ago Space

As humankind's ancestors were learning to walk upright, a star was launched out of the supermassive black hole at the center of our galaxy at a staggering 3.7 million mph (6 million km/h).

Five million years after this dramatic ejection, a group of researchers, led by Sergey Koposov of Carnegie Mellon University's McWilliams Center for Cosmology, has spotted the star, known as S5-HVS1, in the Crane-shaped constellation Grus. The star was spotted traveling relatively close to Earth (29,000 light-years away) at unprecedented, searing speeds — about 10 times faster than most stars in our galaxy.

"The velocity of the discovered star is so high that it will inevitably leave the galaxy and never return," Douglas Boubert, a researcher at the University of Oxford and a co-author on the study, said in a statement.

Our sister publication All About Space magazine takes you on an awe-inspiring journey through our Solar System and beyond, from the amazing technology and spacecraft that enables humanity to venture into orbit, to the complexities of space science.

"This is super exciting, as we have long suspected that black holes can eject stars with very high velocities. However, we never had an unambiguous association of such a fast star with the galactic center," Koposov said in the statement.

The star was discovered with observations from the Anglo-Australian Telescope (AAT), a 12.8-foot (3.9-meter) telescope, and the European Space Agency's Gaia satellite. The discovery was made as part of the Southern Stellar Stream Spectroscopic Survey

(S5), a collaboration of astronomers from Chile, the U.S., the U.K. and Australia.

Now that the star has been spotted, researchers could track the star back to Sagittarius A*, the black hole at the center of the Milky Way. It also serves as an incredible example of the Hills Mechanism, proposed by astronomer Jack Hills 30 years ago, in which stars are ejected from the centers of galaxies at high speeds after an interaction between a binary-star system and the black hole at the center of the galaxy.

"This is the first clear demonstration of the Hills Mechanism in action," Ting Li, a fellow at the Carnegie Observatories and Princeton University who led the S5 collaboration, said in the statement. "Seeing this star is really amazing as we know it must have formed in the galactic center, a place very different to our local environment. It is a visitor from a strange land."

"While the main science goal of S5 is to probe the stellar streams — disrupting dwarf galaxies and globular clusters — we dedicated spare resources of the instrument to searching for interesting targets in the Milky Way, and voila, we found something amazing for 'free.' With our future observations, hopefully we will find even more!" Kyler Kuehn, deputy director of technology at the Lowell Observatory who is part of the S5 executive committee, added in the statement.

This discovery was published in a study on Nov. 4 in the journal the Monthly Notices of the Royal Astronomical Society.

http://bit.ly/2COzyDm

Dinosaur-Era Bird Preserved in 3D Could Rewrite History of Flight

The fossil dates to 120 million years ago.

By Mindy Weisberger

Around 120 million years ago, a bird about the size of a pigeon fluttered through Cretaceous forests in what is now Japan. The newly discovered fossil, preserved in three dimensions, is the first primitive Cretaceous bird found outside China. And it may force scientists to rethink some details in the evolution of flight.

The ancient avian, named *Fukuipteryx prima*, displays something found in modern birds that is absent in other early Cretaceous bird fossils: a bony plate near the tail.

Life restoration of Fukuipteryx prima. **(Image: © Masanori Yoshida)**

Known as a pygostyle, this triangular structure supports tail feathers and has been linked to the evolution of shorter tails for flying. But researchers now suspect that even though this plate emerged as tails became smaller, it isn't necessarily a flight adaptation, according to a new study.

Scientists found the partial skeleton of the bird in Kitadani Dinosaur Quarry, a Lower Cretaceous formation near the city of Katsuyama in central Japan.

What distinguishes birds such as *Fukuipteryx* from their nonavian dinosaur cousins? They have forelimbs longer than their hind limbs, unfused shoulder bones and a shortened tail with a pygostyle, said lead study author Takuya Imai, an assistant professor with the Dinosaur Research Institute at Fukui Prefectural University in Fukui, Japan.

Though some nonavian dinosaurs may have one of these features, only birds have all three, Imai told Live Science in an email.

Like *Archaeopteryx* — the oldest known bird, dating to 160 million to 140 million years ago — *Fukuipteryx* had an unfused pelvis and a U-shaped wishbone: hallmarks of primitive birds. Other intact bones in the fossil included ribs, vertebrae and limb bones, as well as the pygostyle, which was "long, robust and rod-shaped" and ended with "a paddle-like structure," the researchers reported.

In some aspects, *Fukuipteryx*'s pygostyle shape resembled that of **a domestic chicken**, the scientists wrote.

Previously, it was thought that birds' tails shortened as the animals adapted to flight. But *Fukuipteryx* is a more primitive bird than the last of the long-tailed flyers, a genus called *Jeholornis* that lived in China around 122 million to 120 million years ago, Imai said. This suggests that the loss of long tails, and the appearance of the pygostyle, may not be linked to flight.

"We still need more evidence to clarify this," he said.

Prior to this discovery, the only bird fossils from the early Cretaceous came from northeastern China, offering an incomplete view of how birds' distinctive adaptations emerged in the avian family tree, the study said.

"New findings from Japan and other regions in the world may completely change the picture again about what we think of the evolution of flight in the birds," Imai added.

The findings were published online today (Nov. 14) in the journal Communications Biology.

http://bit.ly/2NVZ9AL

How likely do you think you are to develop dementia?

Bottom Line: ***A poll suggests almost half of adults ages 50 to 64 believe they're likely to develop dementia.***

The survey included 1,019 respondents who were asked what risk they perceived and what potential risk-reducing measures they took. Of the participants, 48.5% said they were at least somewhat likely to develop dementia during their lifetime. Many participants reported strategies to try to maintain or improve memory that aren't evidence based. Only a few participants (5.2%) had discussed potential ways to reduce dementia risk with their physician.

To access the embargoed study: Visit our For The Media website at this link https://media.jamanetwork.com/

Authors: Donovan T. Maust, M.D., M.S., University of Michigan, Ann Arbor, and coauthors. (doi:10.1001/jamaneurol.2019.3946)

This link will be live at the embargo time http://bit.ly/37dAWxc

Editor's Note: The article includes conflict of interest and funding/support disclosures. Please see the article for additional information, including other authors, author contributions and affiliations, financial disclosures, funding and support, etc.

Media Advisory: *To contact corresponding author Donovan T. Maust, M.D., M.S., email Kara Gavin at kegavin@med.umich.edu. The full study is linked to this news release.* ***This study is being released to coincide with presentation at the Gerontological Society of America 2019 Annual Scientific Meeting.***

http://bit.ly/33YOpHa

Ketogenic diet helps tame flu virus

A high-fat, low-carbohydrate diet like the Keto regimen has its fans, but influenza apparently isn't one of them.

Mice fed a ketogenic diet were better able to combat the flu virus than mice fed food high in carbohydrates, according to a new Yale University study published Nov. 15 in the journal Science Immunology.

The ketogenic diet -- which for people includes meat, fish, poultry, and non-starchy vegetables -- activates a subset of T cells in the lungs not previously associated with the immune system's response to influenza, enhancing mucus production from airway cells that can effectively trap the virus, the researchers report.

"This was a totally unexpected finding," said co-senior author Akiko Iwasaki, the Waldemar Von Zedtwitz Professor of Immunobiology and Molecular, Cellular and Developmental Biology, and an investigator of the Howard Hughes Medical Institute.

The research project was the brainchild of two trainees -- one working in Iwasaki's lab and the other with co-senior author Visha Deep Dixit, the Waldemar Von Zedtwitz Professor of Comparative Medicine and of Immunobiology. Ryan Molony worked in Iwasaki's lab, which had found that immune system activators called inflammasomes can cause harmful immune system responses

in their host. Emily Goldberg worked in Dixit's lab, which had shown that the ketogenic diet blocked formation of inflammasomes. The two wondered if diet could affect immune system response to pathogens such as the flu virus.

They showed that mice fed a ketogenic diet and infected with the influenza virus had a higher survival rate than mice on a high-carb normal diet. Specifically, the researchers found that the ketogenic diet triggered the release of gamma delta T cells, immune system cells that produce mucus in the cell linings of the lung -- while the high-carbohydrate diet did not.

When mice were bred without the gene that codes for gamma delta T cells, the ketogenic diet provided no protection against the influenza virus.

"This study shows that the way the body burns fat to produce ketone bodies from the food we eat can fuel the immune system to fight flu infection," Dixit said.

http://bit.ly/2XmVI9b

Here's a better way to convert dog years to human years, scientists say

Researchers say they have a new formula to convert dog years to human years—one with some actual science behind it

By Virginia Morell

Our Scotch collie, Buckaroo, is just shy of 14 years old. Following the long-debunked but still popular idea that one dog year equals seven human years, he's almost a centenarian. (This "formula" may be based on average life spans of 10 and 70 years for dogs and people, respectively.) Now, researchers say they have a new formula (see calculator below) to convert dog years to human years—one with some actual science behind it.

The work is based on a relatively new concept in aging research: that chemical modifications to a person's DNA over a lifetime create what is known as an epigenetic clock. Scientists have built a case that one such modification, the addition of methyl groups to specific DNA sequences, tracks human biological age—that is, the toll that disease, poor lifestyle, and genetics take on our bodies. As a result, some groups have converted a person's DNA methylation status to an age estimate—or even a prediction of life expectancy (worrying ethicists, who say the data could be misused by forensic investigators and insurance companies).

Other species also undergo DNA methylation as they age. Mice, chimpanzees, wolves, and dogs, for example, all seem to have epigenetic clocks. To find out how those clocks differ from the human version, geneticist Trey Ideker of the University of California, San Diego, and colleagues started with dogs. Even though man's best friends diverged from humans early in mammalian evolution, they're a good group for comparison because they live in the same environments and many receive similar healthcare and hospital treatments.

All dogs—no matter the breed—follow a similar developmental trajectory, reaching puberty around 10 months and dying before age 20. But to increase their chances of finding genetic factors associated with aging, Ideker's team focused on a single breed: Labrador retrievers.

They scanned DNA methylation patterns in the genomes of 104 dogs, ranging from 4 weeks to 16 years of age. Their analysis revealed that dogs (at least Labrador retrievers) and humans do have similar age-related methylation of certain genomic regions with high mutation rates; those similarities were most apparent when the scientists looked at young dogs and young humans or old dogs and old humans. Most importantly, they found that certain groups of genes involved in development are similarly methylated during aging in both species. That suggests at least some aspects of aging are a continuation of development rather than a distinct process—and that at least some of these changes are evolutionarily

conserved in mammals, Ideker and colleagues report in a preprint posted online at bioRxiv.

"We already knew that dogs get the same diseases and functional declines of aging that humans do, and this work provides evidence that similar molecular changes are also occurring during aging," says Matt Kaeberlein, a biogerontologist at the University of Washington in Seattle, who was not involved with this research. "It's a beautiful demonstration of the conserved features of the epigenetic age clocks shared by dogs and humans."

The research team also used the rate of the methylation changes in dogs to match it to the human epigenetic clock, although the resulting dog age conversion is a bit more complex than "multiply by seven." The new formula says a canine's human age = *16* ***ln****(dog age) + 31*. (That's the natural logarithm of the dog's real age, multiplied by 16, with 31 added to the total.)

Dog age calculator

Using that formula, dogs' and humans' life stages seem to match up. For example, a 7-week-old puppy would be equivalent to a 9-month-old human baby, both of whom are just starting to sprout teeth. The formula also nicely matches up the average life span of Labrador retrievers (12 years) with the worldwide lifetime expectancy of humans (70 years). Overall, the canine epigentic clock ticks much faster initially than the human one--that two year old Lab may still act like a puppy but it is middle-aged, the methylation-based formula suggests--and then slows down.

"They've shown that there's a gradual increase in DNA methylation in both species with age," says Steve Austad, an evolutionary biologist and aging expert at the University of Alabama in Birmingham. He doesn't find that especially surprising, but he thinks the technique could reveal far more interesting results if applied to issues like the different life spans among different dog breeds.

That's one goal of Kaeberlein, whose group's new Dog Aging Project (open to all breeds) will include epigenetic profiles of its canine subjects. He hopes to find out why some dogs develop disease at younger ages or die earlier than normal, whereas others live long, disease-free lives.

So, how does our Buckaroo fare? Happily, the epigenetic clock calculation goes in his favor. He's now only 73 in human years—and a spry 73 at that.

https://wb.md/2CPLftk

Family History Is Never 'Noncontributory'

Family has a lot of meaning for me because family and illness in the family are the reasons I studied medicine

Mark A. Lewis, MD

Hi. I am Mark Lewis, director of gastrointestinal oncology at Intermountain Healthcare in Salt Lake City, Utah, and a contributor to Medscape.

I want to talk about the importance of family. This has a lot of meaning for me because family and illness in the family are the reasons I studied medicine, and oncology in particular.

Tolstoy had a saying that all happy families are alike, but every unhappy family is unhappy in their own way. And I would say that every healthy family appears similar, but every family grappling with illness has to do so in their own way. That is why I believe it is so important to take a good and detailed family history.

This may happen only once during the entire longitudinal encounter with the patient. It tends to happen at the first visit when there are so many other things to talk about, especially if you're dealing with a new cancer diagnosis. The family history, frankly, can be swept aside. But the word that I particularly hate to see in the medical record is "noncontributory." There is hardly any medical condition where it's not important to know about that illness in relatives of the patient.

For example, my father was 42 years old when he was told that he had a form of lung cancer. Before that, his only medical problem had been kidney stones. And despite his strong efforts to reduce calcium intake in his diet, he continued to develop those kidney stones. That was really a bothersome problem for him, but it was then superseded by this lung malignancy. He died when I was 14, and I didn't have any access to his medical records at that point.

Later, I got sick in a similar way. I was just about to start my oncology training. I had abdominal pain and I found that I, too, had a high calcium level. That was actually the key clue I needed to see a pattern in my family; very few conditions cause high calcium levels in consecutive generations. That's when I started to ask questions about my father's medical history. I learned that the form of lung cancer he had was quite specifically a neuroendocrine tumor that grew out of his thymus. That fact plus two consecutive generations of family members with high calcium levels allowed me to see a pattern: That pattern is MEN-1, or multiple endocrine neoplasia type 1. Being able to see this pattern has cast my entire family in a different light. It turns out that my paternal uncle died of a pituitary tumor, and my paternal grandfather also died of a mysterious tumor in his chest. It's only in hindsight that I can see that all of these men actually shared a diagnosis and all died from the same root cause, which was MEN-1.

So when I see a family history taken either without sufficient detail or not taken at all, that really bothers me. Please, please, please take the time when you're first meeting a patient to ask about the health status of their relatives. Record it in as much detail as you can, including details that may seem inconsequential. It may later turn out, as in my case and my family's case, to actually have great importance. Particularly when it comes to cancer, try to note the site and the age at diagnosis. Again, this may allow you to see patterns of hereditary tumor syndromes.

I have one last person to introduce to you. [*Speaking to son.*] Tell the audience your name.

"Alan."

Who are you named after?

"My grandfather."

That's right. You have your grandfather's name. And what else did he share with us? He had the same medical problem we have, right?

My son is 8 years old, and my wife and I are able to take care of him in a completely different and proactive manner, knowing that he also carries MEN-1. I believe that his health is likely to be better than mine, and I certainly hope that he will live longer, better, and with fewer complications than me, my dad, or my grandfather.

Alan and I have a secret handshake that we do as Lewis men, to share our linkage. Obviously, we can't do that on camera or you'll know our secret. But from him and me, and my dad who's no longer with us, we want to remind you that family matters. This is what a family history looks like.

Thank you for listening.

Mark A. Lewis, MD, is director of gastrointestinal oncology at Intermountain Healthcare in Salt Lake City, Utah. He has an interest in neuroendocrine tumors, hereditary cancer syndromes, and patient-physician communication.

https://nyti.ms/2NUlOxi

How Narwhal the 'Unicorn' Puppy May Have Grown a Tail on His Head

The likeliest explanation is not all that cute, some scientists say. But Narwhal is still very cute.

By Elizabeth Preston

A puppy with a tail on his face gained viral fame this week. "I would die for Narwhal," a number of Twitter commenters pledged. The rescue mutt was named for a marine mammal with a single tusk that sticks out of its face. But instead of a tusk, Narwhal the

puppy has a miniature tail flopping between his eyes. Scientists don't agree on how the unusual heart-stealer came to exist.

A Missouri shelter called Mac's Mission, which specializes in what it calls "janky" dogs, took in the abandoned puppy. Staff were disappointed that Narwhal's extra tail didn't wag. But the appendage didn't seem to bother the otherwise normal, healthy puppy, and a veterinarian said there was no need to remove it. An X-ray showed no bones.

Narwhal, the 10-week-old puppy with a tail growing between his eyes, at Mac's Mission animal rescue in Cape Girardeau, Mo., on Wednesday.
Tyler Graef/The Southeast Missourian, via Associated Press

The likeliest explanation for how Narwhal got his face tail is not all that cute, said Margret Casal, a professor at the University of Pennsylvania School of Veterinary Medicine. The tail is probably Narwhal's parasitic twin.

Regular identical twins form when an embryo splits in half very soon after fertilization. Sometimes, this split happens too late in a pregnancy and the halves don't fully separate, leading to conjoined twins. Even more rarely, Dr. Casal said, the late split is asymmetrical, meaning one side of the embryo grows into a fully formed individual and the other becomes an extra body part.

Dr. Casal highlighted a little mohawk of backward-growing fur above Narwhal's face tail, similar to the crest on a dog such as a Rhodesian Ridgeback. She said this could suggest a twin's rear end on Narwhal's face.

David Kilroy's first impression of Narwhal was different.

"At first I thought that it was a bit of clever computer work and not real," said Dr. Kilroy, who specializes in head anatomy and development at the University College Dublin School of Veterinary Medicine. But after looking at the photos and X-ray, he said, "It looks like some weird outgrowth of skin. Although something so large and strange would be most unusual."

Dr. Casal, though, said the bottom of a spine can't develop bones without signals from the top. So if Narwhal's appendage is a parasitic twin, it might make sense that it never grew bones.

Unlike in humans, identical twins are very rare in dogs, which are typically born in litters, Dr. Casal said. So a dog with a parasitic twin is "really super, super rare."

But it's not unheard-of. In one case, a puppy had an extra pair of hind legs growing from its belly. Parasitic twins, like conjoined twins, can occur in humans, too.

Animals are sometimes born with more extreme spare parts, like an entire second head. Two-headed calves occasionally show up in headlines, though they usually die soon after birth.

Snakes, too, can hatch with two heads. In a 2007 paper, a herpetologist, Van Wallach, summarized nearly a thousand reported cases of two-headed snakes. The two heads are almost always next to each other, he found, but occasionally stacked. Many factors can lead to two-headed snakes, including cold temperatures when eggs are incubating. Most two-headed snakes die right away, but a few live to adulthood.

Dr. Wallach had a pet two-headed snake named Brady & Belichick that grew to healthy adulthood. Both heads ate normally. But the head that finished eating its mouse first would then attack and chew on the other head, as Dr. Wallach described in his 2012 paper, "Two-headed Snakes Make High Maintenance Pets."

A calf or snake's second head can arise from a parasitic twin. Or an extra head can form when something goes wrong during a single individual's development. For example, certain genes act like stage directors in a developing embryo, making sure everything ends up in the right place.

"If you get a mutation in one of those genes then you can get bizarre duplications," like two heads, Dr. Casal said. "Or, what we see every once in a while in dogs or cats is they can have, for example, two penises."

Michael Levin, who directs the Allen Discovery Center at Tufts University, said that while Narwhal is a cute example of development gone awry, "I've seen a lot weirder."

Dr. Levin studies how signals between cells, especially electrical signals, help to organize a whole animal into the correct shape. Researchers in his lab have created worms with four heads, tadpoles with eyes on their backs and six-legged frogs.

While Dr. Levin thinks a parasitic twin might explain Narwhal, he said it's impossible to know for sure because of the complex processes that organize bodies even in simple creatures, like flatworms. Chemicals and other factors in a developing animal's environment can make these processes go wrong in countless ways.

"There are massive gaps in our understanding," he said.

Scientists are still trying to answer major questions about how a blob of cells turns into a complete animal of just the right size and shape, with different kinds of parts in all the correct places.

"It's a miracle it comes out right most of the time."

http://bit.ly/2qZG1ss

Egyptian Vats 5,600 Years Old Were For Beer Brewing

Archaeologists working in the ancient city of Hierakonpolis discovered five ceramic vats containing residues consistent with brewing beer.

By Susanne Bard

Some 5,600 years ago, people in the Egyptian city of Hierakonpolis did something that's still a very popular activity today: they brewed and drank beer. We know this, because archaeologists examining the area near the ruins of a cemetery for the elite discovered a structure containing five ceramic vats that would have been heated from below. Residues in the vats confirmed that they had once made beer.

Remains of two of the vats. **Credit: Masahiro Baba in *Scientific Reports***

"And it's estimated that if these five vats were operating at the same time, 325 liters would have been produced, which is equal to 650 cans of Budweiser."

Texas Tech University microbiologist Moamen Elmassry. He says this ancient beer would have tasted very different from what our modern palates are used to. The Egyptian beer makers did use malted wheat and barley in the brewing process. But no one had mastered carbonation yet. So the resulting brew was a flat, unfiltered malt beverage with a low alcohol content.

Elmassry's colleagues recently sampled thick dark deposits from the Hierakonpolis vats. The chemical analysis confirmed that they were indeed the product of beer making and not some other fermented food. The tests also revealed other ingredients ancient Egyptians put in their beer. The researchers found a high concentration of the amino acid proline, which is abundant in dates and some other fruits. "This result suggests that dates could have been used or incorporated in the beer, for flavor." And maybe to add some sweet notes.

Hops—which act as both a flavoring and a preservative—weren't added to beer until medieval times. "The use of hops was unknown to the ancient Egyptians, and we think that they used phosphoric acid to preserve their beer." The residues were indeed high in phosphoric acid, a product of barley grains added during the fermentation process. Phosphoric acid is often used today to prolong the shelf life of alcoholic beverages.

Phosphoric acid via barley would have made it possible to mass produce beer, store it for extended periods, and even transport it—

all consistent with the important role beer played in ancient Egyptian society. It not only provided hydration and nutrition, but was also part of religious rituals among the elite.

The study is in the journal *Scientific Reports*. [Mohamed A. Farag et al. Revealing the constituents of Egypt's oldest beer using infrared and mass spectrometry] Studying ancient beer has allowed Elmassry to reflect on the intersection of science and history.

"I teach a microbiology lab and we brew beer in the lab and the students see the whole fermentation process. And thinking about how ancient Egyptians were able to do a similar thing thousands of years ago, it's kind of very special feeling."

http://bit.ly/37dIKiB

Climate Change Is Going to Supercharge Waves

We know about sea level rise, but new research shows that in some places waves will get bigger and stronger, too.

For surfers looking to ride them, and inhabitants of low-lying coasts hoping to avoid them, waves are best when they are predictable. While it has long been possible to forecast the surf a few days out, projecting how climate change will affect the behavior of waves in the coming decades has been much more difficult—until now.

New research by scientists with the Coordinated Ocean Wave Climate Project (COWCLIP) reduces some of this uncertainty and shows that, if unchecked, climate change will significantly alter how and where waves break—with potentially catastrophic consequences.

Since the 1990s, scientists have been projecting changes in global sea level, but they have not been able to accurately predict trends in waves. Joao Morim, an environmental scientist at Griffith University in Australia, and the COWCLIP team, however, have integrated more than 150 wave and climate models to create the first unified wave model covering the entire planet.

The improved model found that if the climate warms beyond 2 °C, the size, intensity, and direction of waves across half of the world's oceans will drastically change by the end of the century. In some parts of the northern hemisphere, like the east coast of the United States, waves will get smaller and weaker; in other areas, such as on Australia's southern coast, waves will get bigger and more powerful. If countries fail to rein in carbon emissions, the waves that break on southern Australia will be 15 percent larger by 2100.

In some places, these changes are already happening. Since the 1980s, 28,000 square kilometers of coastline have eroded due in part to sea level rise, while larger swells have led to severe storm inundation in some Pacific islands. By 2100, flooding to coastal areas could cause up to US $14-trillion in annual damage.

Morim says the shifts in wave behavior stem in large part from the effect warming has on global weather systems, such as the movement of the wind belt that circles Antarctica. By influencing the latitude of this circular current, increases in atmospheric greenhouse gases have been linked to more frequent periods of low atmospheric pressure over Antarctica, which lead to a strengthening of wave-generating winds. If climate change is not curbed, this trend will be reinforced in the coming decades.

Combining bigger waves with rising sea levels could be disastrous for coastal communities, says Morim. Higher sea levels could "lead to more wave energy being carried to shore, meaning faster erosion and dangerous storms."

Stephen Flood, a coastal adaptation researcher with the MaREI Centre for Marine and Renewable Energy in Ireland who was not involved in the study, agrees that changing wave behavior has dangerous consequences for coastal areas. "Any increases in mean wave height could certainly make coastal communities more vulnerable."