http://www.eurekalert.org/pub_releases/2015-09/e-adc091615.php

Alzheimer's drug could prevent bone fractures

Research shows donepezil prevents bone loss in mice

The most common drug used to treat Alzheimer's disease increases bone mass in mice, according to one of the first research articles published in the new open access journal Heliyon. The authors of the study, from Saitama Medical University in Japan, say this means the drug could also be used to treat bone loss diseases like osteoporosis and periodontitis, following further clinical research.

Alzheimer's disease is the most common form of dementia and the incidence is increasing in our aging population. In the early stages of Alzheimer's disease, bone density decreases, putting patients at a higher risk of bone fractures.

The new Heliyon study suggests that treating Alzheimer's disease with a drug called donepezil not only improves cognitive function but also increases bone density, reducing the risk of fractures.

"We think that donepezil can improve cognitive function and increase bone mass, making it a very useful drug for patients with dementia and osteoporosis," said lead author Dr. Tsuyoshi Sato, Associate Professor in the Department of Oral and Maxillofacial Surgery, Saitama Medical University. "From the viewpoint of medical economics, this dual purpose could reduce the cost of treating these diseases."

Two different kinds of cell control the bone mass and density in our bodies: osteoblasts make bone and osteoclasts absorb it. A molecule called acetylcholine causes osteoclasts to die in vitro. Although an enzyme called acetylcholinesterase breaks this molecule down, the effect of this enzyme on osteoclasts remains unclear.

The most common drug used to treat Alzheimer's disease, donepezil, stops acetylcholinesterase from working, leading to an increase in the amount of acetylcholine in the brain. Recent retrospective clinical studies have suggested that patients being treated with donepezil for Alzheimer's disease have a lower risk of hip fracture, and that risk was dependent on the dose they were taking.

The researchers wanted to understand how donepezil prevents bone degradation. They looked at the drug's activity in vitro using mouse bone marrow cells, and found that more acetylcholinesterase is produced when osteoclasts are being made, which leads to even more osteoclasts being made. Donepezil stops acetylcholinesterase from working, therefore preventing osteoclasts from being made.

The team also looked at the effect of the drug in a mouse model with bone loss. They found that donepezil increases bone mass in mice by preventing the production of osteoclasts.

"We were surprised to see that donepezil directly inhibits the production of osteoclasts and subsequently increases bone mass in vivo," said Dr. Sato. "This is very surprising point - donepezil directly controls the molecule that is responsible for macrophages becoming osteoclasts."

Previous research has shown that acetylcholinesterase activity increases continuously with age, and may accelerate the risk of bone loss in elderly people. The researchers noted that the concentration of acetylcholinesterase in macrophages was higher when the tissue was inflamed. This suggests that inflammation causes bone to be degraded in part due to acetylcholinesterase production.

"Our findings are very promising and suggest that there is a role for donepezil in increasing bone mass in elderly patients with inflammation and dementia," said Dr. Sato. "There is still work to be done and we look forward to observing the effect of this drug in patients."

The team now plans to work with the Department of Neurology at Saitama Medical University on clinical research. They plan to study whether taking donepezil reduces patients' risk of bone fracture by looking at its effect in a group of patients compared to a control group.

"Donepezil prevents RANKL-induced bone loss via inhibition of osteoclast differentiation by downregulating acetylcholinesterase" by Sato et al. (doi: 10.1016/j.heliyon.2015.e00013). The article appears in Heliyon (September 2015), published by Elsevier.

http://bit.ly/1Jpk9T7

How Oklahoma went from two quakes a year to 585

The central US state of Oklahoma has gone from registering two earthquakes a year to nearly two a day and scientists point to a controversial culprit: wastewater injection wells used in fracking.

Located in the middle of the country, far from any major fault lines, Oklahoma experienced 585 earthquakes of a magnitude of 3.0 or greater in 2014. That's more than three times as many as the 180 which hit California last year. "It's completely unprecedented," said George Choy, a seismologist at the US Geological Survey. As of last month, Oklahoma has already experienced more than 600 quakes strong enough to rattle windows and rock cars. The biggest was a 4.5-magnitude quake that hit the small town of Crescent.

Sandra Voskuhl, 76, grew up in the rural oil boomtown and said she has never felt the earth shake like it did on July 27. First came a thunderous boom. Then the red earth shook hard, Voskuhl said. "You heard it coming," she said. "Everything shook." She recalled screaming as framed pictures toppled over in her home. Then, when things got quiet, she drove over to the town's Frontier Historical Museum to help clean up antique dishes that had crashed to the ground and shattered. "We

need the oil for our workers and our economy," she said. "But these earthquakes are a little scary."

– Could a 'Big One' hit? –

Hydraulic fracturing, or fracking, is the process of shooting water mixed with sand and chemicals deep into the earth to crack rock formations and bring up oil and natural gas trapped inside. The process has unlocked massive amounts of oil and gas in Oklahoma and other states over the past decade.

But along with the oil and gas comes plenty of that brackish water, which is disposed of by injecting it into separate wells that are dug as deep as a mile (less than two kilometers) below ground. The unnatural addition of the water can change pressure along fault lines, causing slips that make the earth shake, said Choy of the US Geological Survey.

There is debate among scientists over how large of a fault could be reawakened, and how hard that fault might shake. One camp believes Oklahoma won't see bigger than a 4.0 to 5.0-magnitude earthquake, which would be enough to break windows and knock things off shelves. Others believe a 7.0-magnitude earthquake could come about, which would be strong enough to topple buildings.

"What's at risk is that when you put water into the ground, it's never going to come back out. You're putting it in places it has never been before," Choy told AFP. "The bigger the volume, the greater the area will be affected. And we don't know what the long-term effect will be."

– 4,500 injection wells –

The pace at which earthquake activity has increased has rattled many in Oklahoma, who are also worried about groundwater contamination brought on by fracking. From 1975 to 2008, the state experienced anywhere from zero to three earthquakes a year which registered at 3.0 or higher. Then the numbers jumped: there were 20 in 2009, 35 in 2010, 64 in 2011, 35 in 2012, 109 in 2013 and 585 in 2014.

"We are the only state where once this problem came up, we just kept going (with fracking)," said Johnson Bridgwater, the executive director of the Oklahoma chapter of the Sierra Club, a prominent environmental group.

"We want public safety to come first, rather than treating this state as a giant lab."

The danger is particularly acute given that Oklahoma has such an enormous oil and gas industry, and its pipelines, refineries and storage facilities were not built to withstand constant quakes, Bridgwater said. Oklahoma has about 4,500 disposal wells, with about 3,200 operating on any given day.

State Governor Mary Fallin, a pro-business Republican, was slow to accept the link between fracking and earthquakes. She took action earlier this year after the science became clear, spokesman Alex Weintz said.

It appears that an area known as the Arbuckle rock formation is most vulnerable because of its "unique geological features," he noted. State regulators are now scrutinizing the operations of disposal wells in that area to ensure they don't go too deep or inject too much water. Some operators have been told to cut the amount of water they inject into their wells and the state has also stepped up its monitoring.

Three wells were shut down on Friday after two quakes – a 3.5 and a 4.1 – struck near Cushing, which has one of the largest crude oil storage facilities in the world.

"We are hopeful that the actions taken by the Corporation Commission will have a significant impact on seismicity, but the process is ongoing and we'll continue to evaluate the results that we're getting now and potential future actions," Weintz told AFP.

The Sierra Club insists that much more needs to be done and has called for a moratorium on wastewater injection wells in the 21 Oklahoma counties identified to be most at risk.

http://www.eurekalert.org/pub_releases/2015-09/acoc-sdn091715.php

Sex does not increase heart attack risk

Patients should be encouraged to resume sexual activity after heart attack

Sex is rarely the cause of a heart attack, and most heart disease patients are safe to resume sexual activity after a heart attack, according to a research letter published today in the Journal of the American College of Cardiology.

Sexual activity can be a concern for many heart attack patients who worry about exertion triggering another heart event, but data on the harms and benefits of sexual activity in heart disease patients is limited. According to the research letter, sexual activity generally involves moderate physical activity comparable to climbing two staircases or taking a brisk walk.

Researchers looked at 536 heart disease patients between 30 and 70 years old to evaluate sexual activity in the 12 months before a heart attack and estimate the association of frequency of sexual activity with subsequent cardiovascular events, including fatal heart attack, stroke or cardiovascular death.

In a self-reported questionnaire, 14.9 percent of patients reported no sexual activity in the 12 months before their heart attack, 4.7 percent reported sex less than once per month, 25.4 percent reported less than once per week and 55 percent reported one or more times per week. During 10 years of follow up, 100 adverse cardiovascular events occurred in patients in the study. Sexual activity was not a risk factor for subsequent adverse cardiovascular events.

Researchers also evaluated the timing of the last sexual activity before the heart attack. Only 0.7 percent reported sex within an hour before their heart attack. In

comparison, over 78 percent reported that their last sexual activity occurred more than 24 hours before the heart attack.

"Based on our data, it seems very unlikely that sexual activity is a relevant trigger of heart attack," said Dietrich Rothenbacher, M.D., M.P.H., lead author of the study and professor and chair of the Institute of Epidemiology and Medical Biometry at Ulm University in Ulm, Germany. "Less than half of men and less than a third of women are getting information about sexual activity after heart attack from their doctors. It is important to reassure patients that they need not be worried and should resume their usual sexual activity."

Researchers said that despite the benefits of sexual activity outweighing risks, the potential of erectile dysfunction as a side effect from various cardiovascular protective medications and the risk of a drop in blood pressure from combining certain heart medications with erectile dysfunction medications should be clearly communicated to patients.

http://bit.ly/1MICF0e

Can Napping Make Us Smarter?

Kimberly Cote, director of the Sleep Research Laboratory at Brock University in Ontario, answers:

By Jim Lohr | Aug 13, 2015

Daytime napping in healthy adults does indeed lead to benefits in terms of alertness, mood and cognitive functioning. Adults do not require shut-eye in the middle of the day—unlike infants and toddlers—but many grown-ups nap just the same. A 2008 National Sleep Foundation poll found that 460 out of 1,000 respondents had napped at least twice during the previous month.

People cite a variety of reasons for indulging in daytime siestas. Some take so-called replacement naps to make up for poor sleep the night before. Shift workers may take prophylactic naps in anticipation of needing to stay awake overnight. Many others, regardless of age and culture, habitually take appetitive naps—they sleep simply because it feels good.

Intuitively most of us think that a nap will refresh us and make us better able to take on the challenges of the day. In fact, research shows that healthy adults who take naps enjoy brighter moods, faster reaction times, and better performance on tasks involving logical reasoning, attention and memory.

How much we gain from napping, though, depends on a number of factors, including how and when we nap and for how long. A 20-minute nap appears to hit the sweet spot. Studies reveal that such brief sojourns boost both mood and cognitive performance. Shorter, 10-minute naps are also good for enhancing performance and cause less grogginess than longer naps do.

Naps lasting an hour or more are not recommended. During a longer nap, you fall into a deeper sleep, which makes it more difficult to awaken feeling refreshed. In other words, the longer the nap the greater the “hangover” effect afterward. Also, longer naps diminish the quality of nighttime sleep.

The best time of day to take a nap (assuming you keep a regular night sleep schedule) is midafternoon, between 2 and 4 P.M. Given the body's natural biological clock, it is generally easier to fall asleep during this window and to reap the full benefits of a good rest.

In one study from our sleep laboratory, we found that habitual nappers slept more lightly than nonhabitual nappers did, which may mean that the ability to nap lightly contributes to better alertness and performance after napping. Habitual nappers also reported feeling better than the nonhabitual nappers after the same amount of sleep.

Though generally beneficial, napping isn't for everyone. Poor sleepers who have difficulty falling and staying asleep at night might want to avoid daytime snoozing. For everyone else, though, a 20-minute midafternoon nap could be the secret to feeling sharp and happy throughout the day.

http://www.eurekalert.org/pub_releases/2015-09/uosc-sfd092115.php

Study: Fukushima disaster was preventable

Critical backup generators were built in low-lying areas at risk for tsunami damage -- despite warnings from scientists

The worst nuclear disaster since the 1986 Chernobyl meltdown never should have happened, according to a new study.

In the peer-reviewed Philosophical Transactions A of the Royal Society, researchers Costas Synolakis of the USC Viterbi School of Engineering and Utku Kânoğlu of the Middle East Technical University in Turkey distilled thousands of pages of government and industry reports and hundreds of news stories, focusing on the run-up to the disaster. They found that "arrogance and ignorance," design flaws, regulatory failures and improper hazard analyses doomed the costal nuclear power plant even before the tsunami hit.

"While most studies have focused on the response to the accident, we've found that there were design problems that led to the disaster that should have been dealt with long before the earthquake hit," said Synolakis, professor of civil and environmental engineering at USC Viterbi. "Earlier government and industry studies focused on the mechanical failures and 'buried the lead.' The pre-event tsunami hazards study if done properly, would have identified the diesel generators as the lynch pin of a future disaster. Fukushima Dai-ichi was a siting duck waiting to be flooded."

The authors describe the disaster as a "cascade of industrial, regulatory and engineering failures," leading to a situation where critical infrastructure - in this case, backup generators to keep the cooling the plant in the event of main power loss - was built in harm's way.

At the four damaged nuclear power plants (Onagawa, Fukushima Dai-ichi, Fukushimi Dai-ni, and Toka Dai-ni) 22 of the 33 total backup diesel generators were washed away, including 12 of 13 at Fukushima Dai-ichi. Of the 33 total backup power lines to off-site generators, all but two were obliterated by the tsunami.

Unable to cool itself, Fukushima Dai-ichi's reactors melted down one by one.

"What doomed Fukushima Dai-ichi was the elevation of the EDGs (emergency diesel generators)," the authors wrote. One set was located in a basement, and the others at 10 and 13 meters above sea level; inexplicably and fatally low, Synolakis said.

Synolakis and Kânoğlu report that the Tokyo Electric Power Company (TEPCO), which ran the plant, first reduced the height of the coastal cliffs where the plant was built, underestimated potential tsunami heights, relied on its own internal faulty data and incomplete modeling - and ignored warnings from Japanese scientists that larger tsunamis were possible.

Prior to the disaster, TEPCO estimated that the maximum possible rise in water level at Fukushima Dai-ichi was 6.1 meters - a number that appears to have been based on low-resolution studies of earthquakes of magnitude 7.5, even though up to magnitude 8.6 quakes have been recorded along the same coast where the plant is located.

This is also despite the fact that TEPCO did two sets of calculations in 2008 based on datasets from different sources, each of which suggested that tsunami heights could top 8.4 meters - possibly reaching above 10 meters.

During the 2011 disaster, tsunami heights reached an estimated 13 meters at Fukushimi Dai-ichi - high enough to flood all of the backup generators and wash away power lines.

Further, the 2010 Chilean earthquake (magnitude 8.8) should have been a wake-up call to TEPCO, said Synolakis, who describes it as the "last chance to avoid the accident." TEPCO conducted a new safety assessment of Fukushima Dai-ichi - but used 5.7 meters as the maximum possible height of a tsunami, against the published recommendations of some of its own scientists. TEPCO concluded in November 2010 that they had "assessed and confirmed the safety of the nuclear plants," presenting its findings at a nuclear engineering conference in Japan.

"The problem is that all of TEPCO's studies were done internally, there were no safety factors built in the analysis, which anyway lacked context. Globally, we lack standards for the tsunami-specific training and certification of engineers and scientists who perform hazard studies, and for the regulators who review them, who can in principle ensure that changes be made, if needed." Synolakis said. "How many licensing boards have tsunami-specific questions when granting professional accreditation?"

Lacking tsunami specific training, certification and licensing, the potential for similar mistakes to occur in hazard studies for other coastal nuclear power plants exists, he said. He points to recent studies around the world where lack of experience and context produced tsunami inundation projections with Fukushima size underestimation of the hazard.

Synolakis and Kânoğlu's paper was published on September 21. Their research as supported by ASTARTE Grant 603839 and the National Science Foundation, Award CMMI 1313839. In the same issue of the Philosophical Transactions, another review paper from the universities of Oxford, Cambridge and USC discusses hazards in the Eastern Mediterranean, where nuclear power plants are being planned for construction in the next few years.

http://www.eurekalert.org/pub_releases/2015-09/nu-ldb091715.php

Low dose beta-blockers as effective as high dose after a heart attack

Surprisingly, heart attack patients live as long -- or even longer -- on one-fourth the suggested dose

CHICAGO --- In a surprising new finding, heart attack patients treated with a substantially lower dosage of beta-blockers than used in earlier clinical trials showing their effectiveness survived at the same rate, or even better, than patients on the higher doses used in those trials.

In fact, patients who received one-fourth of the original clinical trial dose had up to a 20 to 25 percent decrease in mortality compared to the full dose group.

About 90 percent of patients who have had a heart attack currently receive beta-blockers, a class of drug commonly prescribed to improve survival and prevent future heart attacks. Beta-blockers block the effects of adrenaline on the heart, reduce irregular heartbeat (arrhythmia) and help prevent heart failure.

No one was more surprised at the results than lead investigator Dr. Jeffrey Goldberger. He launched the study when he discovered heart attack patients were being treated with much lower doses of beta-blockers than were used in clinical trials.

"I thought that was terrible quality of care," said Goldberger, a professor of medicine in cardiology at Northwestern University Feinberg School of Medicine and a cardiologist at Northwestern Memorial Hospital. "We set out on a mission

to show if you treat patients with the doses that were used in the clinical trials, they will do better. We expected to see patients treated with the lower doses to have worse survival. We were shocked to discover they survived just as well, and possibly even better."

New research should be conducted to determine the most appropriate beta-blocker dose for individual patients to get the optimal benefit, said Goldberger, also the director of the program in cardiac arrhythmias at the Center for Cardiovascular Innovation at Feinberg. The earlier clinical trials did not assess the effects of different doses.

The study will be published Sept. 21 in the Journal of the American College of Cardiology.

Patients are treated with lower doses for a variety of reasons. There may be concern about possible side effects that may include fatigue, sexual dysfunction and depression. In addition, when patients are started on conservative, low doses in the hospital after a heart attack, they return home so quickly, there is little time to adjust the dosage, Goldberger said.

The study examined data in a multicenter registry on 6,682 patients who had a heart attack. About 90 percent were receiving beta-blockers. All the patients on beta-blockers survived longer than those who did not receive the drugs. The raw, unadjusted data showed that of the people who received the full dose, 14.7 percent died within two years; of those receiving the half dose, 12.9 percent died; for the quarter dose, 9.5 percent died and for the one-eighth dose, 11.5 percent died.

OBTAIN (Outcomes of Beta-Blocker Therapy After Myocardial Infarction) is an observational multicenter registry in which beta-blocker dosing information was collected in patients with an acute heart attack at participating centers to assess the effect of dose on survival.

"There is probably not one right dose for every single patient," Goldberger said. "It doesn't make sense that the same dose will work for an 80-year-old frail man who had a small heart attack as a burly 40-year-old man with a huge heart attack."

"We now need to figure out how to dose it in individual patients," Goldberger said. "That's something no one has considered in the decades that we have been using this medication. This huge gap in knowledge has been completely unexplored. Since this is medicine we use in every single heart attack patient, we ought to figure out how to use it properly."

The paper is titled: "Effect of Beta-Blocker Dose on Survival After Acute Myocardial Infarction."

Other Northwestern authors include Dr. Robert O. Bonow, Lei Liu, and Haris Subačius.

The research was supported by grant 5U01HL080416 from the National Heart, Lung and Blood Institute at the National Institutes of Health.

http://www.eurekalert.org/pub_releases/2015-09/qi-odo091715.php

Old drug offers new hope to treat Alzheimer's disease

By repurposing a prescription drug used to treat rheumatoid arthritis, researchers successfully reversed tau-related symptoms in an animal model of dementia

Scientists from the Gladstone Institutes have discovered that salsalate, a drug used to treat rheumatoid arthritis, effectively reversed tau-related dysfunction in an animal model of frontotemporal dementia (FTD). Salsalate prevented the accumulation of tau in the brain and protected against cognitive impairments resembling impairments seen in Alzheimer's disease and FTD.

Salsalate inhibits tau acetylation, a chemical process that can change the function and properties of a protein. Published in Nature Medicine, the researchers revealed that acetylated tau is a particularly toxic form of the protein, driving neurodegeneration and cognitive deficits. Salsalate successfully reversed these effects in a mouse model of FTD, lowering tau levels in the brain, rescuing memory impairments, and protecting against atrophy of the hippocampus--a brain region essential for memory formation that is impacted by dementia.

"We identified for the first time a pharmacological approach that reverses all aspects of tau toxicity," says co-senior author Li Gan, PhD, an associate investigator at the Gladstone Institutes. "Remarkably, the profound protective effects of salsalate were achieved even though it was administered after disease onset, indicating that it may be an effective treatment option."

Although tau has been a target in dementia research for some time, there are no tau-targeted drugs available for patients. Additionally, how the protein builds up in the brain, causing toxicity and contributing to disease, still remains largely a mystery.

By investigating post-mortem brains with Alzheimer's disease, Dr. Gan's team found that tau acetylation is one of the first signs of pathology, even before tau tangles are detectable. The acetylated form of tau not only marked disease progression, it also served as a driver for tau accumulation and toxicity. What's more, in an animal model of FTD, when tau was acetylated, neurons had reduced ability to degrade the protein, causing it to build up in the brain. This in turn led to atrophy in the region and cognitive impairment in the mice on several different memory tests.

The Gladstone scientists discovered that salsalate can inhibit the enzyme p300 in the brain, which is elevated in Alzheimer's disease and triggers acetylation. Blocking tau acetylation in this way enhanced tau turnover and effectively reduced tau levels in the brain. This reversed the tau-induced memory deficits and prevented loss of brain cells.

"Targeting tau acetylation could be a new therapeutic strategy against human tauopathies, like Alzheimer's disease and FTD," says co-senior author Eric Verdin, MD, a senior investigator at the Gladstone Institutes. "Given that salsalate is a prescription drug with a long-history of a reasonable safety profile, we believe it can have immediate clinical implications."

The scientists say a clinical trial using salsalate to reduce tau levels in progressive supranuclear palsy, another tau-mediated neurological condition, has already been initiated.

Sang-Won Min, PhD, and Xu Chen, PhD, are co-first authors on the paper. Other investigators on the study from the Gladstone Institutes include Tara Tracy, Yaqiao Li, Yungui Zhou, Chao Wang, Kotaro Shirakawa, S. Sakura Minami, Peter Dongmin Sohn, Jeffrey Johnson, and Nevan Krogan. Scientists from Stanford University, University of California, San Francisco, Buck Institute for Research on Aging, and University of California, San Diego also took part in the research. Funding was provided by the Tau Consortium and the National Institutes of Health.

http://www.bbc.com/news/health-34312126

'No apology' tops patient complaints

Not getting a good enough apology when things go wrong is the most common complaint escalated by NHS patients in England, figures show.

It was the reason behind 34% of cases investigated by the Parliamentary and Health Service Ombudsman in 2014-15. Errors in diagnosing conditions, poor treatment and a lack of communication were also among the top reasons acute hospital trusts were referred.

The organisation upheld 726 complaints out of the 1,652 it investigated.

The PHSO is the final port of call for patients in England who are unhappy with a hospital's original handling of their complaint. The ombudsman has itself been criticised in recent years for not doing its job well enough by investigating too few cases and dragging its heels over decisions. Its latest report shows it has investigated more complaints than last year - 1,652 in 2014-15, compared with 852 in 2013-14. The investigations resulted in 36% of cases about the NHS being upheld, alongside 44% about acute hospital trusts.

Parliamentary and Health Service Ombudsman Julie Mellor said: "We know that there are many factors that influence the number of complaints hospitals receive, such as organisational size, demographics and whether they actively encourage feedback from patients.

"I strongly believe that NHS leaders should welcome feedback from patients and recognise the opportunities that good complaint handling offers to improve the services they provide. "We are publishing this data to help hospital trusts identify problems and take action to ensure trust in the healthcare system remains high."

Highest 10 acute trusts	Investigations	Investigations per 100,000 clinical episodes
London North West Healthcare NHS Trust	*25*	*17*
Basildon and Thurrock University Hospitals NHS Foundation Trust	*27*	*14.8*
Isle of Wight NHS Trust	*10*	*14.6*
The Clatterbridge Cancer Centre NHS Foundation Trust	*2*	*14.5*
Croydon Health Services NHS Trust	*18*	*14.1*
Weston Area Health NHS Trust	*8*	*12.7*
The Hillingdon Hospitals NHS Foundation Trust	*16*	*12.6*
Bedford Hospital NHS Trust	*13*	*12.5*
Liverpool Heart and Chest Hospital NHS Foundation Trust	*3*	*12.4*
Colchester Hospital University NHS Foundation Trust	*22*	*12.3*

http://bit.ly/1OX5BRR

Saturn’s largest moon Titan could have sun-warmed swirling seas

Saturn's largest moon Titan could have sun-warmed swirling seas

And now, the shipping forecast for Titan. Missions to explore the oceans on Saturn’s largest moon might have to contend with powerful currents driven by solar energy.

Titan is the only place in the solar system, besides Earth, that has large bodies of liquid on its surface, though its seas are composed of hydrocarbons such as methane rather than water. Now researchers have built simulations of currents in the large seas in the moon’s northern hemisphere, using maps created from radar data collected by the Cassini probe.

Because Titan’s seas are mostly methane, they behave rather differently to those on Earth. Water takes a lot of energy to heat, and our oceans are very deep, so sunlight only raises the temperature near the surface.

Deep heat

On Titan, if the seas aren’t too murky, sunlight could bring heat into the deep sea and make the methane less dense. The simulation predicts that a combination of the moon’s rotation and the way heat accumulates differently at the surface and at depth means this would cause anticlockwise currents near the sea surface and clockwise currents near the bottom, creating gyres. These large systems of rotating currents are common in Earth’s oceans, where they are driven by wind.

"This heating process is essentially insignificant on Earth," says Ralph Lorenz at the Johns Hopkins University Applied Physics Laboratory in Baltimore, Maryland, who was behind the simulations. "What we found is that it can cause currents that are comparable with tidal and wind-driven currents."

The work has practical implications for missions to explore Titan's seas. Proposed Titan submarines would also need to know how much power is required to navigate in the methane currents.

The major input parameters for the model were based on assumptions or other models, rather than observations, so it is unlikely to describe the true situation, says Sugata Tan, at the Planetary Science Institute in Tucson, Arizona. A more complete evaluation will be possible in the near future when more data from Titan's seas are available, he adds.

We won't know for sure what Titan's currents are like until we have a probe collecting data there. "Extraterrestrial oceanography is still in its early days," says Lorenz. *Journal reference: Icarus, DOI: 10.1016/j.icarus.2015.08.033*

http://www.eurekalert.org/pub_releases/2015-09/nioa-pdd092115.php

Prion disease detected soon after infection and in surprising place in mouse brains

Scientists report they can detect infectious prion protein in mouse brains within a week of inoculation

Prion diseases--incurable, ultimately fatal, transmissible neurodegenerative disorders of mammals--are believed to develop undetected in the brain over several years from infectious prion protein. In a new study, National Institutes of Health (NIH) scientists report they can detect infectious prion protein in mouse brains within a week of inoculation. Equally surprising, the protein was generated outside blood vessels in a place in the brain where scientists believe drug treatment could be targeted to prevent disease. The study, from NIH's National Institute of Allergy and Infectious Diseases (NIAID), appears in the Sept. 22 issue of mBio.

Scientists believe prion diseases potentially could be treated if therapy starts early in the disease cycle. However, identifying who needs treatment and pinpointing the optimal timeframe for treatment are open questions for researchers.

Human prion diseases include variant, familial and sporadic Creutzfeldt-Jakob disease (CJD). The most common form, sporadic CJD, affects an estimated one in one million people annually worldwide. Other prion diseases include scrapie in sheep, chronic wasting disease in deer, elk and moose, and bovine spongiform encephalopathy in cattle.

In their study, the NIAID scientists injected infectious scrapie prion protein into the brains of mice. After 30 minutes, they began observing whether the injected material generated new infectious protein at the injection site. By examining mouse brain tissue, the researchers measured and detected new infectious prion protein three days after infection on the outside walls of capillaries and other blood vessels at the injection site. Using Real-Time Quaking-Induced Conversion (RT-QuIC), a feasible testing method for people, the scientists detected newly generated prion protein after seven days. In prior studies, it took about six weeks to detect infectious prion protein. The new findings enhance scientific understanding of where infectious prion diseases might take hold in the brain and provide possible targets for treatment.

B Chesebro et al. Early generation of new PrPSc on blood vessels after brain microinjection of scrapie in mice. mBio. DOI: 10.1128/mBio.01419-15 (2015).

Bruce Chesebro, M.D., chief of the NIAID Laboratory of Persistent Viral Diseases, is available to comment on this study.

http://www.eurekalert.org/pub_releases/2015-09/bawh-adt092115.php

Androgen deprivation therapy associated with increased risk for fatal heart attack

Long term follow up indicates that men with comorbidity, predominately a prior heart attack, who received androgen deprivation therapy died earlier, due to a fatal heart attack

Long term follow up indicates that men with comorbidity, predominately a prior heart attack, who received androgen deprivation therapy(ADT) died earlier, due to a fatal heart attack.

Androgen deprivation therapy (ADT) and radiation therapy (RT) is known to prolong survival in men with unfavorable-risk prostate cancer and is considered a standard of care. However, in 2008, the FDA implemented a black box warning about ADT use for prostate cancer due to evidence that suggested an increased risk in non-fatal cardiovascular events. The association of ADT use and fatal heart attacks has remained uncertain until now. Specifically, long term follow up of a randomized clinical trial that compared ADT and radiation therapy (RT) to RT alone finds that men with significant comorbidity; most commonly prior heart attack, who received ADT died earlier, due to a fatal heart attack, compared to men who did not receive ADT.

These findings are published in a research letter in the September 22/29, 2015 issue of the Journal of the American Medical Association.

"These findings give us reason to rethink how we manage prostate cancer in men with known heart disease," said Anthony D'Amico, MD, lead author of the research paper and chief of genitourinary radiation oncology at Brigham and

Women's Hospital.. "Specifically, we should be cautious in prescribing ADT in all men who have had a prior heart attack. Men with significant heart disease that is not amenable to medical or surgical correction may be best served with RT alone."

Researchers compared overall survival and death due to prostate cancer, fatal heart attack and all other causes in a group of 206 men with unfavorable risk prostate cancer who were randomized to receive RT alone or RT and six months of ADT. They also categorized the men into subgroups based on extent of prior comorbidity, including prior heart attack. After a median follow up exceeding 16 years, researchers found that overall, survival did not differ between the two groups of men. When analyzing the subgroups of men by differing extent of comorbidity, researchers found that among men whose comorbidity included prior heart attack, treatment with RT and ADT shortened survival due to higher rates of fatal heart attacks, while prolonging survival in men with no or minimal comorbidity.

"While there is a growing body of evidence to support active surveillance for men with low risk prostate cancer, men who have unfavorable-risk cancer and significant comorbidity, notably heart disease, may be best served by considering RT alone or possibly active surveillance. For these men, the side effects of ADT may be life threatening. More research is needed to better understand the newer forms of hormone therapy that do not lower testosterone and how they impact survival," D'Amico said.

http://www.eurekalert.org/pub_releases/2015-09/cshl-gas092215.php

Genetic analysis supports prediction that spontaneous rare mutations cause half of autism

Quantitative study identifies 239 genes whose 'vulnerability' to devastating de novo mutation makes them priority research targets

Cold Spring Harbor, NY - A team led by researchers at Cold Spring Harbor Laboratory (CSHL) this week publishes in PNAS a new analysis of data on the genetics of autism spectrum disorder (ASD). One commonly held theory is that autism results from the chance combinations of commonly occurring gene mutations, which are otherwise harmless. But the authors' work provides support for a different theory.

They find, instead, further evidence to suggest that devastating "ultra-rare" mutations of genes that they classify as "vulnerable" play a causal role in roughly half of all ASD cases. The vulnerable genes to which they refer harbor what they call an LGD, or likely gene-disruption. These LGD mutations can occur "spontaneously" between generations, and when that happens they are found in the affected child but not found in either parent.

Although LGDs can impair the function of key genes, and in this way have a deleterious impact on health, this is not always the case. The study, whose first author is the quantitative biologist Ivan Iossifov, a CSHL assistant professor and on faculty at the New York Genome Center, finds that "autism genes" - i.e., those that, when mutated, may contribute to an ASD diagnosis - tend to have fewer mutations than most genes in the human gene pool.

This seems paradoxical, but only on the surface. Iossifov explains that genes with devastating de novo LGD mutations, when they occur in a child and give rise to autism, usually don't remain in the gene pool for more than one generation before they are, in evolutionary terms, purged. This is because those born with severe autism rarely reproduce.

The team's data helps the research community prioritize which genes with LGDs are most likely to play a causal role in ASD. The team pares down a list of about 500 likely causal genes to slightly more than 200 best "candidate" autism genes.

The current study also sheds new light on the transmission to children of LGDs that are carried by parents who harbor them but whose health is nevertheless not severely affected. Such transmission events were observed and documented in the families used in the study, comprising the Simons Simplex Collection (SSC). When parents carry potentially devastating LGD mutations, these are more frequently found in the ASD-affected children than in their unaffected children, and most often come from the mother.

This result supports a theory first published in 2007 by senior author Michael Wigler, a CSHL professor, and Dr. Kenny Ye, a statistician at Albert Einstein College of Medicine. They predicted that unaffected mothers are "carriers" of devastating mutations that are preferentially transmitted to children affected with severe ASD. Females have an as yet unexplained factor that protects them from mutations which, when they occur in males, will be significantly more likely to cause ASD. It is well known that at least four times as many males as females have ASD.

Wigler's 2007 "unified theory" of sporadic autism causation predicted precisely this effect. "Devastating de novo mutations in autism genes should be under strong negative selection pressure," he explains. "And that is among the findings of the paper we're publishing today. Our analysis also revealed that a surprising proportion of rare devastating mutations transmitted by parents occurs in genes expressed in the embryonic brain." This finding tends to support theories suggesting that at least some of the gene mutations with the power to cause ASD occur in genes that are indispensable for normal brain development.

"Low load for disruptive mutations in autism genes and their biased transmission" appears in the Early Edition of Proceedings of the National Academy of Sciences the week of September 21, 2015. The authors are: Ivan Iossifov, Dan Levy, Jeremy Allen, Kenny Ye, Michael Ronemus, Yoon-ha Lee, Boris Yamrom and Michael Wigler. The paper can be obtained at: http://www.pnas.org/content/early/recent

http://www.bbc.com/news/world-us-canada-34332363

US drug company to cut 5,000% price rise after backlash

A US drug company that faced a backlash after raising the price of a drug used by Aids patients by over 5,000% has said it will lower the price.

Martin Shkreli, the head of Turing Pharmaceuticals, told US media he would drop the price following the outcry, but did not say by how much.

Turing Pharmaceuticals acquired the rights to Daraprim in August.

It then raised the cost of the drug, which treats a parasitic infection, from $13.50 (£8.70) to $750.

Amid criticism from medical groups - one called the cost "unjustifiable" - Mr Shkreli on Monday defended the increase, saying the profits would help research new treatments.

He accused critics of not understanding the pharmaceutical industry.

But he has now told ABC News: "We've agreed to lower the price on Daraprim to a point that is more affordable and is able to allow the company to make a profit, but a very small profit."

Earlier in the day, PhRMA, the pharmaceutical industry's main lobbying group, tweeted that Turing "does not represent the values of PhRMA member companies".

Agreeing a price for any drug is a tricky business.

In the UK, the National Health Service is the main buyer and prices are set through a voluntary scheme between manufacturers and the government, trying to strike the right balance of serving patients and generating money to keep the drug pipeline going.

Profits are capped to stop prices creeping too high.

In the US, the buyers are private insurance companies as well as the government through the Medicare and Medicaid system.

It's a market and prices can go up and down, depending on what people are willing to pay.

In recent years, pharmaceutical research and development has slowed and companies have to think carefully about what they invest in.

Blockbusters such as Viagra pull in money, but orphan drugs for rare diseases can be less attractive.

Not many patients use them, and so turning a profit may be difficult.

http://www.bbc.com/news/health-34322720

What's a fair price for a drug?

Agreeing a price for any drug is a tricky business.

By Michelle Roberts Health editor, BBC News online

In the UK, the NHS is the main buyer and prices are set through a voluntary scheme between manufacturers and the government, trying to strike the right balance of serving patients and generating money to keep the drug pipeline going.

Profits are capped to stop prices creeping too high. In the US, the buyers are private insurance companies as well as the government through the Medicare and Medicaid system. It's a market and prices can go up and down, depending on what people are willing to pay.

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Turing Pharmaceuticals says that is why it has hiked the price of Daraprim - a drug used for treating a rare but sometimes deadly infection called toxoplasmosis.

Greater good?

Turing's controversial founder and chief executive, former hedge-fund manager Martin Shkreli, who was fired from his last biotech venture, says he isn't doing this out of greed, but for justifiable business reasons. He says he has put systems in place to give the drug away free to those who really can't afford it and that some of the profit made will be ploughed into the research and development of new and better drugs. He hopes that by creating a market, other drug companies will join in on this innovation to find new treatments for rarer diseases.

For those who must buy it, the price tag is reported to be $750 (£485) a tablet, compared with $13.50 before the increase. It's thought to cost about $1 to produce, but Mr Shkreli says that does not include other costs such as distribution.

In the UK, the same drug is currently sold by GSK at a cost of £13 for 30 tablets.

Critics say the decision to allow such a massive price jump in the US is outrageous and is more about lining pockets than driving innovation.

The scrutiny of US drug prices is increasing. In the past few weeks, there was a similar outcry over a recent price increase of a drug for tuberculosis in the US. That company, Rodelis Therapeutics, quickly agreed to return the drug to its former owner, a non-profit organisation affiliated with a university.

On Wall Street, biotech shares fell sharply on Monday after Democratic presidential candidate Hillary Clinton accused Turing Pharmaceuticals of "price gouging" and pledged to take action against companies hiking prices for specialty

drugs. If money talks, hurting the profits of pharmaceutical companies would send a clear and loud message, but at what cost? Hopefully not drug innovation.

http://bit.ly/1OX61rq

Most Americans Will Be Misdiagnosed at Least Once

Alarming gaps in knowledge about diagnostic errors and medical delays require intense scrutiny, says an expert medical advisory group

By Dina Fine Maron | September 22, 2015

Most people in the U.S. will experience at least one misdiagnosis or delayed diagnosis in their lifetimes, according to a new report from the Institute of Medicine (IOM). Such mistakes—called diagnostic errors by physicians—could be as simple as failing to forward the results of a medical test showing that a patient recovered from a recent illness. Other errors can have devastating consequences: Perhaps a lung scan that reveals potentially cancerous tissue never makes it to a doctor's desk where it could receive further scrutiny. If the patient and health care provider discovered lung cancer, the patient could have received earlier treatment that might have saved his or her life.

Researchers know very little about the full extent of such errors or how to fix them. But they are pervasive—and deadly. Investigations over several decades have indicated that diagnostic errors contribute to around 10 percent of patient deaths. Recent work also concluded that some 5 percent of U.S. adults who seek outpatient care experience a diagnostic error—and that is a conservative estimate.

A health advisory committee with the private, nonprofit IOM is now calling for that to change. In a new September 22 report the group of experts recommends that federal agencies, including the Health and Human Services, Veterans Affairs and Defense departments, develop a coordinated research agenda on the diagnostic process and diagnostic errors by the end of 2016.

The committee's work builds on a 1999 IOM report that found up to 98,000 people a year die because of mistakes in hospitals. One respected estimate projected that medical errors nowadays could contribute to as many as 210,000 to 440,000 patient deaths annually.

To help avoid diagnostic errors going forward, the new IOM recommendations call for more medical school and continuing education training in making diagnoses and communicating them to patients. Medical providers can also help avert such problems by ensuring that patients have access to their electronic medical records and know how to read them.

More broadly, employers and federal agencies should encourage the reporting of diagnostic errors or "near misses" to help everyone learn about how to avoid them, the report notes. "Despite the pervasiveness of diagnostic errors and the risk for serious patient harm, diagnostic errors have been largely unappreciated," wrote report Chair John Ball, executive vice president emeritus of the American College of Physicians, along with his colleagues. "Without a dedicated focus on improving diagnosis, these errors will likely worsen as the delivery of health care and the diagnostic process continue to increase in complexity."

http://www.eurekalert.org/pub_releases/2015-09/uol-iys092115.php

If you're sitting down, don't sit still, new research suggests

New research suggests that the movements involved in fidgeting may counteract the adverse health impacts of sitting for long periods.

In a study published today in the American Journal of Preventive Medicine, a team of researchers, co-led by the University of Leeds and UCL, report that an increased risk of mortality from sitting for long periods was only found in those who consider themselves very occasional fidgeters. They found no increased risk of mortality from longer sitting times, compared to more active women, in those who considered themselves as moderately or very fidgety.

The study examined data from the University of Leeds' UK Women's Cohort Study, which is one of the largest cohort studies of diet and health of women in the UK.

Study co-lead author Professor Janet Cade, from the School of Food Science and Nutrition at the University of Leeds said: "While further research is needed, the findings raise questions about whether the negative associations with fidgeting, such as rudeness or lack of concentration, should persist if such simple movements are beneficial for our health." Even among adults who meet recommended physical activity levels and who sleep for eight hours per night, it is possible to spend the vast majority of the day (up to 15 hours) sitting down.

The study builds on growing evidence suggesting that a sedentary lifestyle is bad for your health, even if you are physically active outside work.

Breaks in sitting time have previously been shown to improve markers of good health, such as body mass index and your body's glucose and insulin responses. But until now, no study has ever examined whether fidgeting might modify an association between sitting time and death rates.

The University of Leeds' UK Women's Cohort Study gathered information on a wide range of eating patterns of more than 35,000 women aged 35 to 69 who are living in the UK. The new study analyses data from a follow-up survey sent to the same women, which included questions on health behaviours, chronic disease, physical activity levels and fidgeting. More than 14,000 responses were received.

Study co-lead author Dr Gareth Hagger-Johnson from UCL, who conducted the data analysis, said: "Our results support the suggestion that it's best to avoid sitting still for long periods of time, and even fidgeting may offer enough of a break to make a difference."

The research paper, 'Sitting-time, fidgeting and all-cause mortality in the UK Women's Cohort Study', is published online in the American Journal of Preventive Medicine.

http://lat.ms/1iQ76VP

Dinosaur discovery in Alaska: A duck-billed herbivore that didn't fear the snow

Dinosaurs in the snow? It happened.

By Deborah Netburn

In a remote area of northern Alaska, scientists have discovered a duck-billed dinosaur the size of a minibus that roamed above the Arctic Circle roughly 70 million years ago.

The newly described herbivore was dubbed *Ugrunaaluk kuukpikensis* (pronounced oo-GREW-na-luck KOOK-pik-en-sis), which means "ancient grazer of the Colville River" in the Inupiaq language. It was one of more than a dozen species of dinosaurs that lived surprisingly close to the North Pole.

Shown is an artist's depiction of the dinosaur Ugrunaaluk kuukpikensis, which lived in the Arctic Circle about 70 million years ago. **(James Havens / UFA)**

"When we think of dinosaurs, we think of them living in a tropical paradise," said Patrick Druckenmiller, a vertebrate paleontologist at the University of Alaska Fairbanks who described the new find this week in the journal Acta Palaeontologica Polonica. "For these dinosaurs, it was more like an Arctic paradise."

Ugrunaaluk kuukpikensis, which grew to 25 feet in length, had some interesting company. Other dinosaurs found in the same bone deposit include a pygmy tyrannosaur and a horned dinosaur with a fancy frill.

All of these creatures were discovered at a site known as the Prince Creek Formation. Paleontologists have been excavating the area since the 1980s.

When these dinosaurs were alive, the formation was at about 80 degrees latitude, well above the paleo-Arctic Circle. Over time, it has moved south to about 70 degrees latitude, due to the shifting of the Earth's crust.

If you're wondering how dinosaurs managed to survive in the Arctic temperatures we know today, the answer is, they didn't.

Back when *Ugrunaaluk kuukpikensis* roamed, the Arctic was a more hospitable place, with average temperatures around 45 degrees Fahrenheit. Evidence from fossilized pollen suggests these dinosaurs lived in a conifer forest with an understory that included flowering plants, ferns and horsetails.

"It was probably comparable to what you would find in Juneau, Alaska, down in the panhandle of the state," Druckenmiller said. "It wasn't a warm winter, but it was much warmer than it is today."

There are several ways a dinosaur could survive in those temperatures, experts said. The meat eaters might have been covered with feathers to provide insulation against the cold, while the plant eaters may have been good at storing fat.

It's also possible that the dinosaurs were able to slow their metabolism in the winter months to contend with a more limited food supply.

"Modern animals that live up there today like caribou and wolves don't hibernate, but they do adjust their metabolic rates," said Anthony Fiorillo, chief curator at the Perot Museum of Nature and Science in Texas who has worked in northern Alaska for 18 years. "I suspect we would see the same thing in dinosaurs."

Perhaps they already have.

"There is some suggestion that we are seeing seasonality in bone growth, which would support that hypothesis," said Fiorillo, who was not involved in the study.

It is also incorrect to assume that a dinosaur's internal temperature was entirely dependent on the external temperature, like some lizards today.

"They were definitely not like a typical lizard in their morphology," Druckenmiller said. "We all agree that they had some elevated metabolism and body temperature."

More than the cold, the big challenge for *Ugrunaaluk kuukpikensis* and its Arctic contemporaries may have been the long polar night. Between mid-October and mid-February, the sun never rose. "That's what is particularly intriguing about it all," Fiorillo said. "Sure, you can warm the place up, but you still have some profound seasonality in the form of light fluctuations."

Several lines of evidence suggest the community of northern dinosaurs did not migrate south during the winter. That means they would have needed to know how to move around in the dark and find food at a time when plants were scarce.

"Moose could be a good analogue," Druckenmiller said. "They fatten themselves up in the summer and survive on conifer needles in the winter. There's no reason these dinosaurs weren't doing the same thing."

Paleontologists said there is still a lot more to learn from the Prince Creek Formation, though the excavation work is treacherous and expensive. To get to the site, the researchers first have to take small planes or helicopters. Then they board inflatable boats and use the rivers like highways.

"People picture dinosaur digging taking place in the hot summer weather in some desert-y situation," Druckenmiller said. "We are totally dressed up in full winter gear, and it is 45 degrees and sleeting on us."

Despite these difficulties, small teams of paleontologists working for 10 to 14 days at a time have pulled thousands of bones from the fossil bed.

So far, they have found 6,000 bones from *Ugrunaaluk kuukpikensis* alone.

"It's the one we know better than any other," said Druckenmiller, who helped find some of the fossils. "We have every bone in its body."

Thomas Carr, a paleontologist at Carthage College in Kenosha, Wis., said the dinosaur species described so far are just the tip of the iceberg. "I expect that many new fossils will be found of the dinosaur species we know about, and that many hitherto undiscovered species will come to light," said Carr, who was not involved in the study. "We are currently enjoying a renaissance of Arctic dinosaurs."

Fiorillo agreed: "It would not surprise me to see more new animals coming out of the ancient Arctic."

http://www.eurekalert.org/pub_releases/2015-09/uow-utl091715.php

UW team links 2 human brains for question-and-answer experiment

First to show two brains can be linked to allow one person to guess what's on another person's mind

Imagine a question-and-answer game played by two people who are not in the same place and not talking to each other. Round after round, one player asks a series of questions and accurately guesses the object the other is thinking about.

Sci-fi? Mind-reading superpowers? Not quite.

University of Washington researchers recently used a direct brain-to-brain connection to enable pairs of participants to play a question-and-answer game by transmitting signals from one brain to the other over the Internet. The experiment, detailed today in PLOS ONE, is thought to be the first to show that two brains can be directly linked to allow one person to accurately guess what's on another person's mind.

"This is the most complex brain-to-brain experiment, I think, that's been done to date in humans," said lead author Andrea Stocco, an assistant professor of psychology and a researcher at UW's Institute for Learning & Brain Sciences.

"It uses conscious experiences through signals that are experienced visually, and it requires two people to collaborate," Stocco said.

Here's how it works: The first participant, or "respondent," wears a cap connected to an electroencephalography (EEG) machine that records electrical brain activity. The respondent is shown an object (for example, a dog) on a computer screen, and the second participant, or "inquirer," sees a list of possible objects and associated questions. With the click of a mouse, the inquirer sends a question and the respondent answers "yes" or "no" by focusing on one of two flashing LED lights attached to the monitor, which flash at different frequencies.

A "no" or "yes" answer both send a signal to the inquirer via the Internet and activate a magnetic coil positioned behind the inquirer's head. But only a "yes" answer generates a response intense enough to stimulate the visual cortex and cause the inquirer to see a flash of light known as a "phosphene." The phosphene -- which might look like a blob, waves or a thin line -- is created through a brief disruption in the visual field and tells the inquirer the answer is yes. Through answers to these simple yes or no questions, the inquirer identifies the correct item.

The experiment was carried out in dark rooms in two UW labs located almost a mile apart and involved five pairs of participants, who played 20 rounds of the question-and-answer game. Each game had eight objects and three questions that would solve the game if answered correctly. The sessions were a random mixture of 10 real games and 10 control games that were structured the same way.

The researchers took steps to ensure participants couldn't use clues other than direct brain communication to complete the game. Inquirers wore earplugs so they couldn't hear the different sounds produced by the varying stimulation intensities of the "yes" and "no" responses. Since noise travels through the skull bone, the researchers also changed the stimulation intensities slightly from game to game and randomly used three different intensities each for "yes" and "no" answers to further reduce the chance that sound could provide clues.

The researchers also repositioned the coil on the inquirer's head at the start of each game, but for the control games, added a plastic spacer undetectable to the participant that weakened the magnetic field enough to prevent the generation of phosphenes. Inquirers were not told whether they had correctly identified the items, and only the researcher on the respondent end knew whether each game was real or a control round.

"We took many steps to make sure that people were not cheating," Stocco said.

Participants were able to guess the correct object in 72 percent of the real games, compared with just 18 percent of the control rounds. Incorrect guesses in the real games could be caused by several factors, the most likely being uncertainty about whether a phosphene had appeared.

"They have to interpret something they're seeing with their brains," said co-author Chantel Prat, a faculty member at the Institute for Learning & Brain Sciences and a UW associate professor of psychology. "It's not something they've ever seen before."

Errors can also result from respondents not knowing the answers to questions or focusing on both answers, or by the brain signal transmission being interrupted by hardware problems.

"While the flashing lights are signals that we're putting into the brain, those parts of the brain are doing a million other things at any given time too," Prat said.

The study builds on the UW team's initial experiment in 2013, when it was the first to demonstrate a direct brain-to-brain connection between humans. Other scientists have connected the brains of rats and monkeys, and transmitted brain signals from a human to a rat, using electrodes inserted into animals' brains. In the 2013 experiment, the UW team used noninvasive technology to send a person's brain signals over the Internet to control the hand motions of another person.

The first experiment evolved out of research by co-author Rajesh Rao, a UW professor of computer science and engineering, on brain-computer interfaces that enable people to activate devices with their minds. In 2011, Rao began collaborating with Stocco and Prat to determine how to link two human brains together.

In 2014, the researchers received a $1 million grant from the W.M. Keck Foundation that allowed them to broaden their experiments to decode more complex interactions and brain processes. They are now exploring the possibility of "brain tutoring," transferring signals directly from healthy brains to ones that are developmentally impaired or impacted by external factors such as a stroke or accident, or simply to transfer knowledge from teacher to pupil.

The team is also working on transmitting brain states -- for example, sending signals from an alert person to a sleepy one, or from a focused student to one who has attention deficit hyperactivity disorder, or ADHD.

"Imagine having someone with ADHD and a neurotypical student," Prat said. "When the non-ADHD student is paying attention, the ADHD student's brain gets put into a state of greater attention automatically."

Many technological advancements over the past century, from the telegraph to the Internet, were created to facilitate communication between people. The UW team's work takes a different approach, using technology to strip away the need for such intermediaries.

"Evolution has spent a colossal amount of time to find ways for us and other animals to take information out of our brains and communicate it to other animals in the forms of behavior, speech and so on," Stocco said. "But it requires a translation. We can only communicate part of whatever our brain processes.

"What we are doing is kind of reversing the process a step at a time by opening up this box and taking signals from the brain and with minimal translation, putting them back in another person's brain," he said.

Other co-authors are UW computer science and neurobiology undergraduate student Darby Losey, UW bioengineering doctoral student Jeneva Cronin, UW bioengineering doctoral student Joseph Wu, and Justin Abernethy, a research assistant at the UW Institute for Learning & Brain Sciences.

http://www.eurekalert.org/pub_releases/2015-09/uu-eei092115.php

Enamel evolved in the skin and colonized the teeth much later

When did the enamel that covers our teeth evolve? And where in the body did this tissue first appear?

In the latest issue of the journal Nature, researchers from Uppsala University in Sweden and the Institute of Vertebrate Palaeontology and Palaeoanthropology (IVPP) in Beijing, China, combine data from two very different research fields - palaeontology and genomics - to arrive at a clear but unexpected answer to this question: enamel originated in the skin and colonized the teeth much later.

We are all familiar with enamel: shiny and white, this tissue gleams back at us from the bathroom mirror every morning when we brush our teeth. It is the hardest substance produced by the body, composed almost entirely of the mineral apatite (calcium phosphate) deposited on a substrate of three unique enamel matrix proteins.

Like other land vertebrates we only have teeth in the mouth, but certain fishes such as sharks also have "dermal denticles" - little tooth-like scales - on the outer surface of the body. In many fossil bony fishes, and a few archaic living ones such as the gar (Lepisosteus) from North America, the scales are covered with an enamel-like tissue called "ganoine". Tatjana Haitina, a researcher at the Department of Organismal Biology, Uppsala University, investigated the genome of Lepisosteus, which was sequenced by the Broad Institute, and found that it contains genes for two of our three enamel matrix proteins: the first to be identified from a ray-finned bony fish. Furthermore, these genes are expressed in the skin, strongly suggesting that ganoine is a form of enamel.

But where did enamel originate - in the mouth, in the skin, or both at once? The answer to that question is provided by two fossil fishes, Psarolepis from China and Andreolepis from Sweden, which are both more than 400 million years old and which have been studied by Qingming Qu and Per Ahlberg of Uppsala University in collaboration with Min Zhu from IVPP in Beijing. In Psarolepis the scales and the denticles of the face are covered with enamel, but there is no enamel on the teeth; in Andreolepis only the scales carry enamel.

"Psarolepis and Andreolepis are among the earliest bony fishes, so we believe that their lack of tooth enamel is primitive and not a specialization. It seems that enamel originated in the skin, where we call it ganoine, and only colonized the

teeth at a later point," explains Per Ahlberg, Professor of Evolutionary Organismal Biology at Uppsala University.

The study is the first to combine novel palaeontological and genomic data in a single analysis to explore tissue evolution. The research group plans to continue exploring the evolution of vertebrate hard tissues using this approach.

Qingming Qu, Tatjana Haitina, Min Zhu, Per Erik Ahlberg (2015) New genomic and fossil data illuminate the origin of enamel, Nature, DOI: 10.1038/nature15259

http://www.eurekalert.org/pub_releases/2015-09/wkh-iti092315.php

In terminally ill patients, some types of delirium are a sign of 'imminent death'

Hypoactive and "mixed" delirium-are strong indicators that death will come soon

In cancer patients nearing the end of life, certain subtypes of delirium--specifically, hypoactive and "mixed" delirium--are a strong indicator that death will come soon, reports a study in Psychosomatic Medicine: Journal of Biobehavioral Medicine, the official journal of the American Psychosomatic Society. The journal is published by Wolters Kluwer.

"Terminally ill patients with the hypoactive or mixed subtypes of delirium showed a higher probability of imminent death, with even earlier mortality among younger patients," according to the new research by Sung-Wan Kim, MD, and colleagues of Chonnam National University Medical School Gwangju, Republic of Korea. They believe their findings might help make more accurate predictions of survival in patients nearing the end of life.

Shorter Survival in Patients with Hypoactive/Mixed Delirium

The researchers looked at the relationship between delirium and survival time in 322 patients with terminal cancer entering palliative care. Delirium refers to confusion, altered awareness, or altered thoughts. It can result from many different illnesses, medications, and other causes.

Delirium was divided into subtypes according to standard DSM-5 criteria: hyperactive delirium, with increased motor activity, loss of control, and restlessness; hypoactive delirium, with decreased activity, decreased speech, and reduced awareness. Patients with normal psychomotor activity or fluctuating activity levels were classified as having "mixed" delirium.

About 30 percent of patients were diagnosed with delirium on entering palliative care. Of these, the delirium subtype was hyperactive in about 15 percent of patients, hypoactive in 34 percent, and mixed in 51 percent.

Survival time after entering palliative care was shorter for patients with delirium: median 17 days, compared to 28 days for those without delirium. However, the difference was significant only for patients with hypoactive or mixed delirium--with median survival times of 14 and 15 days, respectively.

These differences remained significant after adjustment for other factors. For patients with hyperactive delirium, survival was not different from that in patients without delirium.

While delirium was more common in older patients, the effects on time to death were actually stronger in younger patients. That was consistent with previous studies suggesting shorter survival times in younger patients diagnosed with delirium

Why are different delirium subtypes associated with differing survival times? It may have to do with differences in the underlying causes of and treatment responses. Hyperactive delirium is commonly related to reversible causes, such as medication side effects.

"In contrast, hypoactive delirium is generally related to hypoxia [decreased oxygen levels], metabolic disturbances, and multi-organ failure," Dr. Kim explains. "Therefore, hypoactive delirium could be associated with a higher mortality rate than hyperactive delirium."

Dr. Kim adds, "Also, the earlier mortality in younger patients overturns a conventional assumption for survival prediction of delirium. Although delirium was more prevalent in older patients, as known, the irony is that delirium predicted shorter survival in younger patients."

Accurate predictions of survival time in terminally ill patients are important for many reasons--"in terms of ensuring good clinical decision making, developing care strategies, and preparing for the end of life in a dignified manner." The researchers conclude, "Thus, the present findings could facilitate more precise predictions of survival, allowing families to prepare for the patient's death."

Click here to read "Differential Associations Between Delirium and Mortality According to Delirium Subtype and Age: A Prospective Cohort Study."

Articles: "Differential Associations Between Delirium and Mortality According to Delirium Subtype and Age: A Prospective Cohort Study." (doi: 10.1097/PSY.0000000000000239)

http://www.eurekalert.org/pub_releases/2015-09/hms-tfw092315.php

The final word on STAP

Researchers fail to replicate STAP study; computational analysis reveals genomic inconsistency

Tremendous controversy erupted in early 2014 when two papers published in Nature described how a technique called "stimulus-triggered acquisition of pluripotency," or STAP, could quickly and efficiently turn ordinary cells into pluripotent stem cells, that is, stem cells capable of developing into all the tissues in the body.

The simplicity of the approach--subjecting the cells to particular stresses like mild acid exposure--seemed too good to be true. And it was.

Almost immediately stem cell researchers around the world began questioning the results, as repeated attempts to replicate the findings failed. After an investigation by the journal revealed many problems and inconsistencies with the data, the papers were retracted.

Despite the retractions, claims persisted that the essential science of STAP was valid and that issues of replication could be solved through refined protocols. As a result, a group of scientists representing seven international laboratories and led by researchers at Harvard Medical School and Boston Children's Hospital pooled their collective efforts to replicate STAP, which included experiments conducted in the lab where STAP was first developed.

They also went beyond the original experiments and analyzed publicly available genomic sequence data with newly developed bioinformatics algorithms.

Collectively, researchers worldwide were unable to replicate the findings reported in the original STAP papers.

These negative results will be published in Nature, along with a companion paper that describes universal hallmarks of pluripotency, providing a roadmap that researchers can use to determine whether they have in fact created induced pluripotent stem cells, or iPS cells.

"The scientific process requires replicating and extending existing data," said George Q. Daley, HMS professor of biological chemistry and molecular pharmacology at Boston Children's and co-senior author on both papers addressing the STAP controversy. "We appreciate that can be difficult. We must strive for ever-higher standards of rigor up front, which can be at odds with the rush to publish in this increasingly competitive environment."

One experiment that the researchers sought to replicate involved a gene called Oct4, one of the most consistent markers of iPS cells. Most scientists agree that Oct4 is essential.

To test for Oct4, researchers use a green fluorescent protein that activates when Oct4 is present. In the original STAP studies, the researchers did in fact detect green fluorescence in the cells, leading them to believe that they had induced pluripotency.

However, when Alejandro De Los Angeles, a scientist in the Daley lab, repeated the protocol, he noticed what researchers call "autofluorescence," a tendency for some molecules in cells to emit light randomly when excited by lasers. The lasers used to detect green fluorescence require proper filters to separate random signal from noise.

After exposing cells to the original acid treatment and adjusting for the appropriate laser filters, the researchers detected no active presence of Oct4.

Another hallmark of pluripotent stem cells is their ability to form teratomas, benign tumors that arise when stem cells differentiate into multiple tissues when injected into mice.

While the original STAP papers claim to have found teratomas, researchers attempting to replicate teratomas from STAP preparations discovered adverse chemical reactions that could have been mistaken for teratoma formation. Aside from this, no teratomas were found.

In analyzing the original experiments, Peter Park, HMS associate professor of biomedical informatics, developed a set of algorithmic tools to analyze the original genomic data from the study. He refers to this approach as "forensic bioinformatics."

At first this was challenging because publicly available data sets from the original study were incomplete and poorly labeled. But once Park's team members had gathered enough data, they were able to determine in less than a month that the initial studies were problematic.

Inferring genetic variants in the DNA of the cells from gene expression data, Francesco Ferrari, a postdoctoral fellow in the Park lab, and his colleagues found that many of the cells described as STAP cells were genomically distinct from their predecessors.

In some cases, they were even different genders. In one critical experiment where STAP-derived cells were reported to behave like both embryonic and placental stem cells, it was found that the cell populations were in fact a mixture of embryonic and placental stem cells that pre-existed in the lab.

"At the very least, journals should enforce proper annotation and timely deposition of datasets into public databases," said Park. "It won't prevent this sort of thing from ever happening again, but it is an easily attainable safeguard."

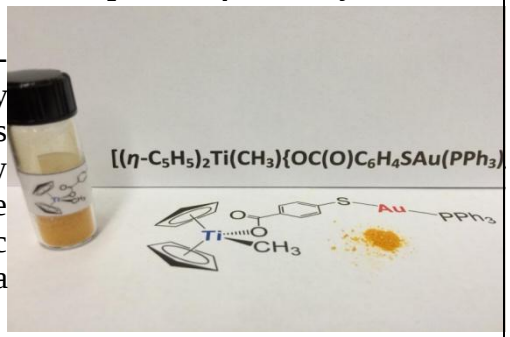
Furthermore, Park emphasized the importance of careful bioinformatic analysis in these studies, noting that "if the authors, their colleagues or the referees of the manuscripts had the right expertise in genomic data analysis, the STAP cell idea could have been discredited much earlier with the data they had already generated. That would have saved so much time and effort for researchers around the world who tried to replicate the findings."

"Ultimately, we need to have more checks and balances in science," said Daley, who is also an investigator of the Howard Hughes Medical Institute. "Incentives in the system are so stacked toward being productive and publishing and getting grants that it can lead even very well-intentioned people into too easily accepting their own cognitive biases."

http://www.eurekalert.org/pub_releases/2015-09/uohc-tag092315.php

Titanium and gold based compound fights kidney cancer cells

New research on metal-based compound shows promise for kidney cancer patients



A metal-based compound shows promise for kidney cancer patients. **Brooklyn College (The City University of New York)**

Researchers developed a promising metal-based compound that destroys kidney cancer cells, while leaving normal cells unharmed. The findings may provide a new way of treating kidney cancer, opening the potential for more potent and less toxic therapies that would give cancer patients a better quality of life.

"Kidney cancer is frequently diagnosed in the late stages when there are minimal options for treating the deadly disease. The hope is that this could potentially lead to new therapies that would extend the life-span of cancer patients who are diagnosed late," said Dr. Joe Ramos, PhD, a professor and the director of the Cancer Biology Program at the University of Hawaii Cancer Center.

Chemical Science published the findings by Dr. Maria Contel, an associate professor in the Department of Chemistry at Brooklyn College (The City University of New York) and Dr. Ramos. The study highlights the increased effectiveness and reduced toxicity of anti-cancer compounds containing the two metals, titanium and gold, called Compound 5 when used together. The research indicates that the improved anti-tumor activity may be due to the interaction of the different metals with multiple biological targets, or by the improved chemical and physical properties of the new compound.

"A gold based compound (called Auranofin) has been used to treat rheumatic diseases for years and has recently been used in clinical trials for the treatment of some cancers such as Chronic Lymphocytic Leukemia. However, that drug does not work well for kidney cancer. An important finding for us was that the incorporation of the titanium fragment into the similar gold based compound 5 increased the activity and specificity towards kidney cancer," said Contel.

Unlike previous metallic compounds known to fight cancerous cells, this titanium-gold compound does not attack DNA, but rather causes cancer cell death by blocking a group of enzymes that supports cancer cell survival and metastasis.

Compound 5 shrank tumors and performed better in pre-clinical models than the FDA approved platinum drug, Cisplatin, showing excellent promise for further clinical development. Researchers emphasize the necessity of having further studies to find how the compound affects other cancers and improve its potential for clinical use.

"To do the best cutting-edge cancer research you often need to work between disciplines and institutions. This work is the result of such a collaboration. This is the sort of work especially fostered by Cancer Centers like the UH Cancer Center, and is an important mission of NCI designated Cancer Centers like ours," said Ramos.

The UH Cancer Center will host The First International Organometallics Symposium in December 2015 where top researchers in the field will meet to share and discuss the latest findings of using metal-based compounds to fight cancer.

http://www.eurekalert.org/pub_releases/2015-09/cp-apb091715.php

Antidepressants plus blood thinners cause brain cancer cells to eat themselves in mice

Researchers find that antidepressants work against brain cancer by excessively increasing tumor autophagy

Scientists have been exploring the connection between tricyclic antidepressants and brain cancer since the early 2000s. There's some evidence that the drugs can lower one's risk for developing aggressive glioblastomas, but when given to patients after diagnosis in a small clinical trial, the antidepressants showed no effect as a treatment.

In a study appearing in Cancer Cell on September 24, Swiss researchers find that antidepressants work against brain cancer by excessively increasing tumor autophagy (a process that causes the Cancer Cells to eat themselves). The scientists next combined the antidepressants with blood thinners--also known to increase autophagy--as a treatment for mice with the first stages of human glioblastoma. Mouse lifespan doubled with the drug combination therapy, while either drug alone had no effect.

"It is exciting to envision that combining two relatively inexpensive and non-toxic classes of generic drugs holds promise to make a difference in the treatment of patients with lethal brain cancer," says senior study author Douglas Hanahan, of the Swiss Federal Institute of Technology (EPFL). "However, it is presently unclear whether patients might benefit from this treatment. This new mechanism-based strategy to therapeutically target glioblastoma is provocative, but at an early stage of evaluation, and will require considerable follow-up to assess its potential."

Mice received the combination therapy 5 days a week with 10-15 minute intervals between drugs. The antidepressant was given orally, and the other drug (the blood

thinner or anti-coagulant) was injected. The data suggest that the drugs act synergistically by disrupting, in two different places, the biological pathway that controls the rate of autophagy--a cellular recycling system that at low levels enhances cell survival in stressful conditions. The two drugs work together to hyper-stimulate autophagy, causing the Cancer Cells to die.

"Importantly, the combination therapy did not cure the mice; rather, it delayed disease progression and modestly extended their lifespan," Hanahan says. "It seems likely that these drugs will need to be combined with other classes of anticancer drugs to have benefit in treating gliblastoma patients. One can also envision 'co-clinical trials' wherein experimental therapeutic trials in the mouse models of glioblastom are linked to analogous small proof-of-concept trials in GBM patients. Such trials may not be far off."

This work was supported by grants from Fondation S.A.N.T.É. and the School of Life Sciences at EPFL.

Cancer Cell, Shchors et al.: "Dual targeting of the autophagic regulatory circuitry in gliomas with repurposed drugs elicits cell-lethal autophagy and therapeutic benefit"

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http://www.eurekalert.org/pub_releases/2015-09/igdc-fbt092115.php

From brain, to fat, to weight loss

New study reveals neural mechanism responsible for fat breakdown

Weight is controlled by the hormone leptin, which acts in the brain to regulate food intake and metabolism. However, it was largely unknown until now, how the brain signals back to the fat tissue to induce fat breakdown. Now, a breakthrough study led by Ana Domingos at Instituto Gulbenkian de Ciência (IGC; Portugal), in collaboration with Jeffrey Friedman's group at Rockefeller University (USA), has shown that fat tissue is innervated and that direct stimulation of neurons in fat is sufficient to induce fat breakdown. These results, published in the latest issue of the prestigious journal Cell*, set up the stage for developing novel anti-obesity therapies.

Fat tissue constitutes 20 to 25% of human body weight being an energy storage container, in the form of triglycerides. Twenty years ago Jeffrey Friedman and colleagues identified the hormone leptin, which is produced by fat cells in amounts that are proportional to the amount of fat, and informs the brain about how much fat is available in the body. Leptin functions as an "adipostat" neuro-endocrine signal that preserves body's fat mass in a relatively narrow range of variation. Low leptin levels increase appetite and lower basal metabolism, whereas high leptin levels blunt appetite and promote fat breakdown. However, until now it was largely unknown what circuits close the neuroendocrine loop, such that leptin action in the brain signals back to the fat.

Now, the research team led by Ana Domingos, combined a variety of techniques to functionally establish, for the first time, that white fat tissue is innervated. "We dissected these nerve fibers from mouse fat, and using molecular markers identified these as sympathetic neurons", explains Ana Domingos. But most remarkable, "when we used an ultra sensitive imaging technique, on the intact white fat tissue of a living mouse, we observed that fat cells can be encapsulated by these sympathetic neural terminals".

Next, researchers used genetic engineered mice, whose sympathetic neurons could be activated by blue light, to assess the functional relevance of these fat projecting neurons. Roksana Pirzgalska, a doctorate student in Domingos' laboratory and co-first author of the study explains: "We used a powerful technique called optogenetics, to locally activate these sympathetic neurons in fat pads of mice, and observed fat breakdown and fat mass reduction". Ana Domingos adds: "The local activation of these neurons, leads to the release of norepinephrine, a neurotransmitter, that triggers a cascade of signals in fat cells leading to fat hydrolysis. Without these neurons, leptin is unable to drive fat-breakdown". The conclusions and future directions are clear according to Ana Domingos: "This result provides new hopes for treating central leptin resistance, a condition in which the brains of obese people are insensitive to leptin." Senior co-author Jeffrey Friedman adds: "These studies add an important new piece to the puzzle that enables leptin to induce fat loss".

This work was funded by Fundação para a Ciência e Tecnologia (FCT), European Molecular Biology Organization (EMBO) and the JPB Foundation.

**Zeng, W., Pirzgalska, R.M., Pereira, M.A.M., Kubasova, N., Barateiro, A., Seixas, E., Lu, Y., Kozlova, A., Voss, H., Martins, G.G., Friedman, J.M., Domingos, A.I. (2015). Sympathetic Neuro-Adipose Connections Mediate Leptin-Driven Lipolysis. Cell.*

http://dx.doi.org/10.1016/j.cell.2015.08.055

http://www.eurekalert.org/pub_releases/2015-09/w-rft092115.php

Researchers find ticks linked with Lyme disease in south London parks

Visitors to 2 popular parks in South London are at risk of coming into contact with ticks that can transmit Lyme disease to humans, according to a new study in Medical and Veterinary Entomology.

Researchers studied 4 parks to see how prevalent ticks were and whether they carried the Borrelia burgdorferi bacterial parasite that causes Lyme borreliosis (Lyme disease). A total of 1109 ticks (532 larvae, 568 nymphs, 6 adult male, 3 adult female) were collected at Richmond Park and 9 ticks (all nymphs) were collected at Bushy Park. The team found no evidence of ticks in Wimbledon Common or Hampton Court.

When the investigators analyzed ticks for the presence of B. burgdorferi, they estimated the presence of 0.22 infected ticks per 40-m transect in Richmond Park. The researchers advise the public to take preventative measures to avoid tick bites in Bushy, and especially Richmond, parks.

"The overall the risk of Lyme disease in London parks is very low, but precautions should be taken. Check yourself and your pets after frequenting parkland areas and remove ticks as quickly as possible, if you find any, using a tick removal tool," said Dr. James Logan, senior author of the Medical and Veterinary Entomology study. "To minimize the risk, stick to footpaths and wear an insect repellent. "

http://www.eurekalert.org/pub_releases/2015-09/dlnl-nto092415.php

New theory of stealth dark matter may explain universe's missing mass

Lawrence Livermore scientists have come up with a new theory that may identify why dark matter has evaded direct detection in Earth-based experiments.

A group of national particle physicists known as the Lattice Strong Dynamics Collaboration, led by a Lawrence Livermore National Laboratory team, has combined theoretical and computational physics techniques and used the Laboratory's massively parallel 2-petaflop Vulcan supercomputer to devise a new model of dark matter. It identifies it as naturally "stealthy" (i.e. like its namesake aircraft, difficult to detect) today, but would have been easy to see via interactions with ordinary matter in the extremely high-temperature plasma conditions that pervaded the early universe.

"These interactions in the early universe are important because ordinary and dark matter abundances today are strikingly similar in size, suggesting this occurred because of a balancing act performed between the two before the universe cooled," said Pavlos Vranas of LLNL, and one of the authors of the paper, "Direct Detection of Stealth Dark Matter through Electromagnetic Polarizability". The paper appears in an upcoming edition of the journal Physical Review Letters and is an "Editor's Choice."

Dark matter makes up 83 percent of all matter in the universe and does not interact directly with electromagnetic or strong and weak nuclear forces. Light does not bounce off of it, and ordinary matter goes through it with only the feeblest of interactions. Essentially invisible, it has been termed dark matter, yet its interactions with gravity produce striking effects on the movement of galaxies and galactic clusters, leaving little doubt of its existence.

The key to stealth dark matter's split personality is its compositeness and the miracle of confinement. Like quarks in a neutron, at high temperatures, these electrically charged constituents interact with nearly everything. But at lower temperatures they bind together to form an electrically neutral composite particle. Unlike a neutron, which is bound by the ordinary strong interaction of quantum chromodynamics (QCD), the stealthy neutron would have to be bound by a new and yet-unobserved strong interaction, a dark form of QCD.

"It is remarkable that a dark matter candidate just several hundred times heavier than the proton could be a composite of electrically charged constituents and yet have evaded direct detection so far," Vranas said.

Similar to protons, stealth dark matter is stable and does not decay over cosmic times. However, like QCD, it produces a large number of other nuclear particles that decay shortly after their creation. These particles can have net electric charge but would have decayed away a long time ago. In a particle collider with sufficiently high energy (such as the Large Hadron Collider in Switzerland), these particles can be produced again for the first time since the early universe. They could generate unique signatures in the particle detectors because they could be electrically charged.

"Underground direct detection experiments or experiments at the Large Hadron Collider may soon find evidence of (or rule out) this new stealth dark matter theory," Vranas said.

The LLNL lattice team authors are Evan Berkowitz, Michael Buchoff, Enrico Rinaldi, Christopher Schroeder and Pavlos Vranas, who is the lead of the team. The LLNL Laboratory Directed Research and Development and Grand Challenge computation programs supported this research. Other collaborators include researchers from Yale University, Boston University, Institute for Nuclear Theory, Argonne Leadership Computing Facility, University of California, Davis, University of Oregon, University of Colorado, Brookhaven National Laboratory and Syracuse University.

http://www.eurekalert.org/pub_releases/2015-09/uog-wwm092415.php

Women with moderate beer consumption run lower risk of heart attack

Women who drink beer at most once or twice per week run a 30 per cent lower risk of heart attack, compared with both heavy drinkers and women who never drink beer.

These are the findings of a Swedish study which has followed 1,500 women over a period of almost 50 years. In the study, researchers at the Sahlgrenska Academy, University of Gothenburg, have followed a representative selection of the middle-aged female population from 1968 to 2000 (when the women in the study were between 70 and 92 years old).

Now, with the help of data from the study, the researchers have attempted to chart the relationship between the intake of different types of alcoholic beverages and the incidence of heart attacks, stroke, diabetes and cancer.

Beer consumption

In the study in question, the 1,500 women were asked about the frequency of their consumption of beer, wine or spirits (from 'daily' to 'nothing in the past 10 years'), and about various physical symptoms.

The results reveal that over the 32-year follow-up period, 185 women had a heart attack, 162 suffered a stroke, 160 developed diabetes and 345 developed cancer.

Higher cancer risk

The study shows a statistically significant connection between high consumption of spirits (defined as more frequent than once or twice per month) and an almost 50 per cent higher risk of dying of cancer, compared with those who drink less frequently.

Lower risk of heart attack

The study also reveals that women who reported that they drank beer once or twice per week to once or twice per month ran a 30 per cent lower risk of a heart attack than women who drank beer several times per week/daily or never drank beer. Moderate consumption of beer thus seems to protect women from heart attacks.

"Previous research also suggests that alcohol in moderate quantities can have a certain protective effect, but there is still uncertainty as to whether or not this really is the case. Our results have been checked against other risk factors for cardiovascular disease, which substantiates the findings. At the same time, we were unable to confirm that moderate wine consumption has the same effect, so our results also need to be confirmed through follow-up studies," explains Dominique Hange, researcher at Sahlgrenska Academy.

The article A 32-year longitudinal study of alcohol consumption in Swedish women: Reduced risk of myocardial infarction but increased risk of cancer was published online in Scandinavian Journal of Primary Health Care in July 2015.

FACTS

The women study ("Kvinnostudien") in Gothenburg began in the late 1960s, when around 1,500 middle-aged women representative of the female population of Gothenburg were surveyed and were asked to answer a series of questions regarding their health and any medical conditions that they might have. The women have been followed continuously since then, with regular follow-ups, from 1968-1969 right up until the most recent survey which is currently underway.

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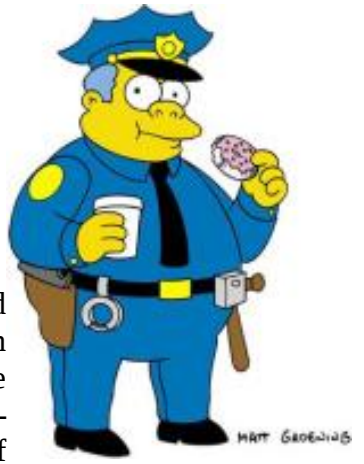
How Doughnut-Loving Cops Became a Stereotype

A sugar-sweet symbol for beat cops around the country

By Danny Lewis smithsonian.com



From *The Simpsons'* Chief Wiggum to the Twin Peaks sheriff's department, in pop culture police officers and doughnuts go together like peanut butter and jelly. There are few, if any, other professions that are so associated with a specific food as cops and doughnuts that it begs the question of how the sugary snack became a staple for the stereotypical cop's diet? As Cara Giamo writes for *Atlas Obscura*, cops around the United States began to be associated with doughnuts back in the 1950's, when they were some of the only snacks available to police walking the late-night beat. Back then, doughnut shops were some of the only stores open late at night because they needed to get ready for the morning rush. As a result, they were some of the best options for cops who needed a quick bite to eat, a place to fill out paperwork or make a call, or to simply sit and take a breather, Michael Krondl writes in his book, *The Donut*.

The History of the Doughnut

"When it came to [meals], graveyard cops in the forties and fifties had few choices," former Seattle Chief of Police Norm Stamper once wrote, Krondl reports. "They could pack lunch, pray for an all-night diner on their beat, or fill up on doughnuts. Doughnuts usually won out. They were, to most palates, tasty, and they were cheap and convenient."

At the time, Giamo writes, the relationship between doughnut and police officer wasn't a joke: in fact, it was a point of pride for some shops:

...having officers around made the shop workers feel safe—as early as 1950, one small-time inn owner threatened a larger, litigious hotel chain by boasting, "our High Sheriff and our local troop of state police… help themselves to coffee and doughnuts in my kitchen when the spirit so moves them, which seems about every day."

In some cases, according to Giamo, police departments had to step in and remind their officers that accepting free doughnuts could give an impression of favor to a person or business that could undermine their roles as impartial law enforcement. Even so, the doughnut had already become married to police in popular culture, as well as the cops walking or driving their nightly beats.

For more on the history of the long relationship between police officer and doughnut, make sure to read Giamo's article.

http://bit.ly/1KXEzYF

Scientists Manipulate Common Plants to Produce Cancer Drugs

Stanford researchers have figured out how to transfer a rare plant's chemical "assembly line" into a cheap, common lab plant

By Emily Matchar smithsonian.com

Mayapple plant (Susan Quinlan)

Many commonly used medicines are still derived from plants. Scopolamine, used for motion sickness and to treat post-surgical nausea, is made from plants in the nightshade family. Digoxin, a heart medication, comes from the foxglove plant. Codeine and other opioid painkillers are derived from opium poppies.

But plants used to make medications are sometimes endangered or expensive. A poor growing season or geopolitical instability in the region where a plant is cultivated could cause a decline in medication supply. Now, a Stanford scientist has figured out how to isolate the molecular "factory" within an endangered plant and assemble it within another, more widely available plant.

"This was a challenge, because plants are pretty complicated," says Elizabeth Sattely, a professor of chemical engineering. "They're pretty difficult to work with. Their genomes are very complicated."

Sattely and her team worked with a Himalayan plant called the mayapple, which produces precursors to a commonly used chemotherapy drug called etoposide. Etoposide is used to treat a variety of cancers, including lymphoma, lung cancer, testicular cancer and some types of leukemia and brain cancer. It's on the World Health Organization's list of essential medicines—drugs considered crucial for medical system functioning. But mayapple is slow-growing, and supply has been in decline for years due to high demand.

Sattely realized that mayapple's chemical assembly line starts up in response to its leaves being injured. Once this injury occurs, the plant starts producing a number of proteins. Some of these proteins eventually produce etoposide's precursor. But the big question was which proteins? There were more than 30 present, but not all of them were involved in making the precursor. "What was crucial here was really narrowing down our candidate list," Sattely says.

She and her team tried out various combinations of proteins until they figured out which 10 constituted the assembly line. Then, they put the genes that made these 10 proteins into a different plant. The plant they chose was *Nicotiana benthamiana*, a wild relative of tobacco, chosen because it's widely available and easy to grow in a lab. The *Nicotiana* plant began producing the etoposide precursor, just like mayapple. Sattely and her graduate student, Warren Lau, published their discovery in the journal *Science*.

"This is a very nice proof of concept," says Sattely.

Sattely hopes to ultimately make microbes, such as yeast, produce the same molecules, skipping plants entirely. If she succeeds, she'll be joining a number of scientists who have figured out how to turn microorganisms into drug-producing factories. Just this week, German scientists announced they'd made genetically modified yeast produce THC, the compound in marijuana that produces the "high" and can help treat side effects from chemotherapy and other illnesses. Last month, Stanford researchers published results showing how they had made yeast produce hydrocodone, an opioid painkiller similar to morphine. The breakthrough has potential to make such drugs cheaper and more accessible. In 2013, chemical engineers at Berkeley coaxed genetically modified yeast into producing anti-malaria drugs.

Making drugs with yeast is even simpler and less expensive than using common lab plants. The supplies are incredibly cheap and easy to produce, take little space or special care, and can be endlessly manipulated.

"The promise of the field of synthetic biology is that you can get cells to make or do anything you want," Sattely says.

But there's still much to learn from plants and the chemicals they produce. As plants' molecular production pathways become better understood, scientists can learn to manipulate them, potentially producing better drugs with fewer side effects. "Plants are some of the best molecular factories in nature," Sattely says. "We have a lot to learn about these molecules that are so important for human health and also for plant health."

http://www.eurekalert.org/pub_releases/2015-09/chla-tnf092415.php

Tumor necrosis factor in colitis -- bad actor or hero?

Common therapeutic target for the treatment of inflammatory bowel disease may actually protect against intestinal inflammation

Investigators at Children's Hospital Los Angeles have found that a common therapeutic target for the treatment of inflammatory bowel disease (IBD) may actually protect against intestinal inflammation by inhibiting pathogenic T-cells. This discovery, reported in the October 2015 issue of Gastroenterology, could lead to new treatment options for the 65 percent of individuals with IBD who do not respond or become resistant to anti-TNF medications.

According to lead author Shivesh Punit of The Saban Research Institute, discovering that tumor necrosis factor receptor 2 (TNFR2) mitigates inflammation

in mice was surprising, given that therapies that target tumor necrosis factor (TNF) are the primary treatments for individuals with IBD.

"Understanding this mechanism allows us to target new therapeutic approaches for patients who don't respond to current therapies," said principal investigator Brent Polk, MD, who was senior author on this study. Polk is a pediatric gastroenterologist and director of The Saban Research Institute of Children's Hospital Los Angeles, and is also professor and chairman of pediatrics at the Keck School of Medicine of the University of Southern California.

An autoimmune disorder that causes inflammation of the intestinal tract, IBD is a broad term that includes ulcerative colitis and Crohn's disease. Characterized by severe gastrointestinal symptoms that get worse over time, IBD negatively affects quality of life and increases risk of colon cancer. In the United States, more than one million people are living with IBD with a cost of treatment of over one billion dollars each year. Currently, there is no cure for IBD. For patients with moderate to severe disease, one current therapy acts to blocks TNF. Although anti-TNF medications represent a significant breakthrough in treatment, they are effective in only one third of individuals suffering from IBD. Recent research suggests there are various conditions that lead to the disease including a microbial imbalance in the gut, dysregulated immunity and alterations in the epithelial cells that line the intestinal tract.

In the current study, investigators examined the role of TNFR2 in mice with IBD. Biological activity of TNF is mediated by two cell surface receptors--TNFR1 and TNFR2. TNFR2 is located primarily on immune cells and during inflammation increases in the intestinal epithelial cells. When the investigators blocked this receptor-mimicking the effect of anti-TNF treatment-they noted an increase in severity and decrease in time to onset of colitis. To verify that the effect was mediated by TNFR2, they did bone marrow transfers, and the mice that got TNFR2-deficient bone marrow developed severe disease.

The investigators also noted that loss of TNFR2 increased cytotoxic CD8 T-cells two-fold. When they specifically inhibited CD8 cells, IBD resolved. They also showed that loss of TNFR2 on CD8 cells alone worsened IBD. These observations led the investigators to conclude that CD8 T-cells worsen IBD in this model, and that TNFR2 alleviates IBD by inhibiting these cells.

Additional contributors to the study include Philip E. Dube, Cambrian Y. Liu and Nandini Girish, of The Saban Research Institute of Children's Hospital Los Angeles and the Keck School of Medicine of the University of Southern California; and M. Kay Washington, Vanderbilt University. Funding was provided in part by the National Institutes of Health R01DK056008, R01DK54993 and P30DK058404, the Canadian Institutes of Health Research, the Crohn's and Colitis Foundation of America, and the California Institute for Regenerative Medicine (CIRM).

http://www.eurekalert.org/pub_releases/2015-09/uoia-sat092115.php

Study adds to evidence that viruses are alive

New analysis supports the hypothesis that viruses are living entities

CHAMPAIGN, Ill. -- A new analysis supports the hypothesis that viruses are living entities that share a long evolutionary history with cells, researchers report. The study offers the first reliable method for tracing viral evolution back to a time when neither viruses nor cells existed in the forms recognized today, the researchers say. The new findings appear in the journal Science Advances.

Until now, viruses have been difficult to classify, said University of Illinois crop sciences and Carl R. Woese Institute for Genomic Biology professor Gustavo Caetano-Anollés, who led the new analysis with graduate student Arshan Nasir. In its latest report, the International Committee on the Taxonomy of Viruses recognized seven orders of viruses, based on their shapes and sizes, genetic structure and means of reproducing.

"Under this classification, viral families belonging to the same order have likely diverged from a common ancestral virus," the authors wrote. "However, only 26 (of 104) viral families have been assigned to an order, and the evolutionary relationships of most of them remain unclear."

Part of the confusion stems from the abundance and diversity of viruses. Less than 4,900 viruses have been identified and sequenced so far, even though scientists estimate there are more than a million viral species. Many viruses are tiny - significantly smaller than bacteria or other microbes - and contain only a handful of genes. Others, like the recently discovered mimiviruses, are huge, with genomes bigger than those of some bacteria.

The new study focused on the vast repertoire of protein structures, called "folds," that are encoded in the genomes of all cells and viruses. Folds are the structural building blocks of proteins, giving them their complex, three-dimensional shapes. By comparing fold structures across different branches of the tree of life, researchers can reconstruct the evolutionary histories of the folds and of the organisms whose genomes code for them.

The researchers chose to analyze protein folds because the sequences that encode viral genomes are subject to rapid change; their high mutation rates can obscure deep evolutionary signals, Caetano-Anollés said. Protein folds are better markers of ancient events because their three-dimensional structures can be maintained even as the sequences that code for them begin to change.

Today, many viruses - including those that cause disease - take over the protein-building machinery of host cells to make copies of themselves that can then spread to other cells. Viruses often insert their own genetic material into the DNA of their hosts. In fact, the remnants of ancient viral infiltrations are now

permanent features of the genomes of most cellular organisms, including humans. This knack for moving genetic material around may be evidence of viruses' primary role as "spreaders of diversity," Caetano-Anollés said.

The researchers analyzed all of the known folds in 5,080 organisms representing every branch of the tree of life, including 3,460 viruses. Using advanced bioinformatics methods, they identified 442 protein folds that are shared between cells and viruses, and 66 that are unique to viruses.

"This tells you that you can build a tree of life, because you've found a multitude of features in viruses that have all the properties that cells have," Caetano-Anollés said. "Viruses also have unique components besides the components that are shared with cells."

In fact, the analysis revealed genetic sequences in viruses that are unlike anything seen in cells, Caetano-Anollés said. This contradicts one hypothesis that viruses captured all of their genetic material from cells. This and other findings also support the idea that viruses are "creators of novelty," he said.

Using the protein-fold data available in online databases, Nasir and Caetano-Anollés used computational methods to build trees of life that included viruses.

The data suggest "that viruses originated from multiple ancient cells ... and co-existed with the ancestors of modern cells," the researchers wrote. These ancient cells likely contained segmented RNA genomes, Caetano-Anollés said.

The data also suggest that at some point in their evolutionary history, not long after modern cellular life emerged, most viruses gained the ability to encapsulate themselves in protein coats that protected their genetic payloads, enabling them to spend part of their lifecycle outside of host cells and spread, Caetano-Anollés said. The protein folds that are unique to viruses include those that form these viral "capsids."

"These capsids became more and more sophisticated with time, allowing viruses to become infectious to cells that had previously resisted them," Nasir said. "This is the hallmark of parasitism."

Some scientists have argued that viruses are nonliving entities, bits of DNA and RNA shed by cellular life. They point to the fact that viruses are not able to replicate (reproduce) outside of host cells, and rely on cells' protein-building machinery to function. But much evidence supports the idea that viruses are not that different from other living entities, Caetano-Anollés said.

"Many organisms require other organisms to live, including bacteria that live inside cells, and fungi that engage in obligate parasitic relationships - they rely on their hosts to complete their lifecycle," he said. "And this is what viruses do."

The discovery of the giant mimiviruses in the early 2000s challenged traditional ideas about the nature of viruses, Caetano-Anollés said.

"These giant viruses were not the tiny Ebola virus, which has only seven genes. These are massive in size and massive in genomic repertoire," he said. "Some are as big physically and with genomes that are as big or bigger than bacteria that are parasitic."

Some giant viruses also have genes for proteins that are essential to translation, the process by which cells read gene sequences to build proteins, Caetano-Anollés said. The lack of translational machinery in viruses was once cited as a justification for classifying them as nonliving, he said.

"This is no more," Caetano-Anollés said. "Viruses now merit a place in the tree of life. Obviously, there is much more to viruses than we once thought."

The paper "A phylogenomic data-driven exploration of viral origins and evolution" is available to members of the media from vancepak@aaas.org.

http://www.eurekalert.org/pub_releases/2015-09/asop-toa092515.php

The origin and spread of 'Emperor's rice'

Scientists solve the mystery of black rice

Traditional rice balls prepared from white, red, and black varieties of rice.

Black rice has a rich cultural history; called "Forbidden" or "Emperor's" rice, it was reserved for the Emperor in ancient China and used as a tribute food. In the time since, it remained popular in certain regions of China and recently has become prized worldwide for its high levels of antioxidants.

Despite its long history, the origins of black rice have not been clear. Black rice cultivars are found in locations scattered throughout Asia.

However, most cultivated rice (species Oryza sativa) produces white grains, and the wild relative Oryza rufipogon has red grains. The color of rice grains is determined by which colored pigments they accumulate (or fail to accumulate, in the case of white rice). For instance, the pro-anthocyanidins that give wild rice grains their characteristic red color are not produced in white rice due to a mutation in a gene controlling pro-anthocyanidin biosynthesis. The color in black rice is known to be due to anthocyanin pigments, but how these came to be made in the grains was not known.

A paper to be published this week in The Plant Cell reveals the answer to the long-standing question of how black rice became black and, moreover, traces the history of the trait from its molecular origin to its spread into modern-day

varieties of rice. Researchers from two institutions in Japan collaborated to meticulously examine the genetic basis for the black color in rice grains.

They discovered that the trait arose due to a rearrangement in a gene called Kala4, which activates the production of anthocyanins. They concluded that this rearrangement must have originally occurred in the tropical japonica subspecies of rice and that the black rice trait was then transferred into other varieties (including those found today) by crossbreeding.

According the study's lead scientist, Dr. Takeshi Izawa, "The birth and spread of novel agronomical traits during crop domestication are complex events in plant evolution." This new work on black rice helps explain the history of domestication of rice by ancient humans, during which they selected for desirable traits including grain color.

http://www.eurekalert.org/pub_releases/2015-09/ind-sis092515.php

Should I stay or should I go? On the importance of aversive memories and the endogenous cannabinoid

Cannabinoid receptors of the brain control aversive memories crucial for survival

Memory is not a simple box of souvenirs; it is also, and most importantly, a safety system for organisms. With the help of negative memories, known as "aversive" memories, we can avoid a threat that we have already confronted. Researchers from Inserm and University of Bordeaux have just discovered that the cannabinoid receptors of the brain control these memories that are crucial for survival. This study is published in Neuron.

When confronted by danger, every individual has to make a crucial choice. This type of "simple" decision may determine his/her destiny: if the fire alarm goes off, we have learned to heed it and flee, and not to ignore it. In the same way, we avoid food and drinks that might have made us sick in the past.

The body is thus equipped with neurological mechanisms that help it to adjust its behaviour in response to a stimulus. Such is the case with aversive memories, a key survival process, which prepares the body to avoid these potential dangers effectively. These memories are accompanied by physiological responses (fright and flight) that enable one to get away from a dangerous situation.

Although the role of the habenula, a central region of the brain, in this phenomenon has received a great deal of attention in recent years, the same is not true of the endogenous cannabinoid system of the habenular neurons, on which Giovanni Marsicano and his team (particularly Edgar Soria-Gomez) have focused. This system involves the type 1 cannabinoid receptors. These receptors, the activity of which is normally regulated by endocannabinoids - the body's own molecules - are the target of the main psychoactive components of cannabis.

The researchers conditioned mice so that they reacted to certain danger signals (sounds or smells). When they exposed them to these signals, mice that were deficient in cannabinoid receptors in the habenula expressed neither the fear nor the repulsion observed in normal mice. Interestingly, this impaired reaction did not apply to neutral or positive memories, which remained unchanged in these mice.

At molecular level, the scientists observed that, although the functioning of the habenula normally involves two molecules (acetylcholine and glutamate), the defect observed in these mice is caused by an imbalance in neurotransmission involving only acetylcholine.

"These results demonstrate that the endogenous cannabinoid system in the habenula exclusively controls the expression of aversive memories, without influencing neutral or positive memories, and does so by selectively modulating acetylcholine in the neural circuits involved," explains Giovanni Marsicano, Inserm Research Director.

The control of these particular memories is an integral part of diseases associated with the emotional process, such as depression, anxiety or drug addiction. As a consequence, the endogenous cannabinoid system of the habenula might represent a new therapeutic target in the management of these conditions.

Habenular CB1 receptors control the expression of aversive memories

Edgar Soria-Gómez1,2, Arnau Busquets-Garcia1,2, Fei Hu3, Amine Mehidi1,2, Astrid Cannich1,2, Liza Roux1,2, Ines Louit1,2, Lucille Alonso1,2, Theresa Wiesner1,2, François Georges2,4, Danièle Verrier1,2, Peggy Vincent1,2, Guillaume Ferreira2,5, Minmin Luo3 and Giovanni Marsicano1,2

1INSERM, U862 Neurocentre Magendie, Bordeaux 33077, France; 2University of Bordeaux, France; 3National Institute of Biological Sciences, Beijing 100875, China; 4French National Centre for Scientific Research (CNRS), UMR 5297, Interdisciplinary Institute for Neuroscience, Bordeaux 33077, France; 5French National Agronomic Research Institute (INRA), Nutrition and Integrative Neurobiology Laboratory, UMR 1286, Bordeaux 33077, France.

http://bit.ly/1LUILoI

The Nose Job Dates Back to the 6th Century B.C.

But for a long time, the nose was built up instead of shaved down

By Marissa Fessenden smithsonian.com

The rhinoplasty, colloquially known as the nose job, is now popular enough that it's considered minor plastic surgery. Still, the procedure earns comment when the proboscis being shaped is a famous one and plenty of people express concern over the current boom in cosmetic surgeries. Yet nose jobs aren't new. The earliest recorded schnoz shaping happened in ancient India in the 6th century B.C., reports Tiffany Hearsey for *The Atlantic*.

The report is related by Elizabeth Harken in her book *Venus Envy: A History of Plastic Surgery*. The ancient Indian procedure included taking a flap of skin from

the patient's cheek and reshaping it into a new nose. The ayurvedic physician Sushruta describes that procedure in his *Sushruta samhita*, now considered a foundational Sanskrit text on medicine.

Still, rhinoplasty didn't enter the limelight in the West until syphilis struck Europe in the late 16th century. Hearsey writes:

One of the unfortunate symptoms of advanced syphilis is soft-tissue decay, which affects the nose and leaves a gaping hole in the middle of one's face. Such a disfigurement carried the social stigma of disease and infection, even if the afflicted had lost their nose by another means. Different methods were employed to recreate noses. One of the most popular procedures involved taking skin from the patient's arm and grafting it to their face in an effort to make a new nose (or something resembling one, anyway).

People have long sought out rhinoplasties to address cosmetic concerns and conform to society's beauty ideals. Some of that led people to try and make their features look less like that of a racial minority in America (still a motivating factor today).

But not all nose jobs were motivated by beauty standards. Facial surgery experienced a real boom during the two World Wars, as soldiers with injuries to their jaws, lips and noses became the proving ground for surgeons experimenting with reconstruction techniques.

In her book, excerpted by *The New York Times*, Harken writes of surgeons who pioneered ways to build up noses, rather than reduce them. So-called "saddle-nose" could be caused by syphilis, but also could be inherited or caused by trauma or infection. A surgeon observed in 1926 that ""Many persons with a saddle nose ... are suspected of having inherited disease and are greatly handicapped, both in their social and business relations."

Building up the nose presented the challenge of figuring out what to build with. The human body rejects many substances, such as ivory, that were used for other protheses. For a time, surgeons in the early 20th century settled on paraffin, but over time the substance tended to move, especially if people spent time in the sun and frequently caused cancer.

Then, as now, plastic surgery was sometimes ridiculed, but still the demand increased. A pioneer in the field, Charles Conrad Miller, noted that the serious surgeon should not turn away patients seeking facial surgery. The rise of unskilled, untrained "surgeons" to fill that need presented a professional dilemma. Harken writes:

For this, Miller blamed neither the charlatans nor the gullible patients, but the physicians who did not take seriously their patients' needs. "Physicians cannot longer disregard the effect of the `Beauty Columns,'" Miller insisted. "Every practitioner who laughs at the patient who questions him regarding an operation for improving the appearance of the face takes the chance of seeing that patient return from the advertiser disfigured for life." Although he voiced some sympathy for those physicians whose refusal to perform cosmetic operations reflected their personal convictions, Miller believed that it was too late to turn the tide of public interest: "the demand for featural surgeons is too great on the part of the public."

As surgeons have perfected the procedure, its popularity has only grown. The American Society of Plastic Surgeons reports that rhinoplasty is the second most popular procedure, with 217,000 noses reshaped in 2014 out of a total 15.6 million cosmetic procedures. The surgery that earned the top spot? Breast augmentation.

http://bit.ly/1iU1WIA

Ancient Human Ancestors Heard Differently

Early human species may have had sharper hearing in certain frequencies than we enjoy, to facilitate short-range communication in an open environment. Cynthia Graber reports.

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Imagine the evolutionary advantage of being able to hear a predator rustling in the tall grass nearby—or in the ability to hear a comrade making a (make a titch sound, like calling a horse) sound to warn you about that predator. Now a study finds that early human species may have had sharper hearing in certain frequencies than we enjoy. The finding is in the journal *Science Advances*. [Rolf Quam et al, Early hominin auditory capacities]

"We've been able to reconstruct an aspect of sensory perception in a fossil human ancestor known as* Australopithecus africanus *and* Paranthropus robustus *from South Africa."

Binghamton University anthropologist Rolf Quam.

"Both of these fossil forms lived about two million years ago and represent early human ancestors. We took CT scans of the skulls. We created virtual reconstructions on the computer of the internal structures of the ear that will predict how an organism hears based on these measurements of its ear."

And the reconstructed physiology reveals that those early hominins likely heard differently than both modern chimps and modern humans.

Specifically, the hominins were probably more sensitive to frequencies associated with sounds like t, k, f, and s.

"We're not arguing they had language, but we think our results do have implications for how they communicated. And the finding is that this hearing pattern would have been beneficial if you were engaging in short-range vocal communication in an open environment."

The estimation of the hearing abilities of the hominins complements previous research suggesting that these species spent more time in open environments such

as the savannah—where a hasty, short-range consonant from a comrade might convey important information—than they spent in dense rainforests, where sound travels farther. Could be that (make a few consonant sounds) were survival tools that also paved the way for the evolution of full-fledged human language. Even if we can't hear those sounds quite as well as those ancient hominins did.

http://www.bbc.com/news/health-34360868

Two new kidney cancer drugs 'work'

Two new therapies for hard to treat advanced kidney cancer could change treatment of the disease, say experts at the European Cancer Congress.

Both drugs increased survival in trials which are also published in the New England Journal of Medicine. One drug takes the brakes off the immune system while the other stops growth signals in the tumour. Cancer Research UK said the developments will "greatly expand the arsenal" of available drugs.

Kidney cancer is the eighth most common cancer in the UK and survival rates plummet if it is caught late. Once the tumour has spread to other parts of the body then only one-in-10 people live for five years after diagnosis.

The first trial, called Checkmate 025, used the immunotherapy drug nivolumab.

It is one of a suite of "checkpoint inhibitors" being developed by pharmaceutical companies that stop cancers turning off the immune system.

They have already been proven effective in skin and lung cancers.

The trial on 821 patients showed average survival was increased from 19.6 months with standard therapy, to 25 months with nivolumab.

Dr James Larkin, a consultant at the Royal Marsden Hospital, told the BBC News website: "It's another big day for immunotherapy for cancer and one of the biggest days for kidney cancer for some time. "We've known for two to three years that these drugs have efficacy in multiple types of cancer, but it's the randomised control trials that are important."

Strong results

The second trial, Meteor, used the targeted therapy cabozantinib on a trial of 658 patients. It doubled survival from 3.8 months to 7.4 months.

Prof Toni Choueiri, from Harvard Medical School, said: "An early evaluation of overall survival from the ongoing Meteor trial has shown a strong trend indicating that survival may be improved in patients receiving cabozantinib compared to standard therapy."

Commenting on the findings, Dr Alan Worsley from Cancer Research UK, said: "Advanced kidney cancer has been hard to treat for far too long and it has been particularly difficult to find drugs that work after first-line treatment has failed.

"The drugs tested in these two trials both appear to work better than everolimus - one of the options available if the first treatment fails - and with fewer side-effects. "Cabozantinib, a targeted therapy, and nivolumab, an immunotherapy, fight cancer in very different ways, so making either available for use in the clinic will greatly expand the arsenal for clinicians to treat kidney cancer patients."

Prof Peter Naredi, the scientific co-chair of the Congress, said he was "excited over the advances" and that the results "most likely will be practice-changing".

http://bit.ly/1KGa6K9

A Short History of the Rise, Fall and Rise of Subliminal Messaging

People have believed in subliminal influences for hundreds of years—but the last few decades have taken a far more scientific look at these ideas

By Victoria Stern | Aug 13, 2015

The idea that people can be subliminally influenced is ancient—historical evidence suggests that in the fifth century B.C., Greek thinkers attempted to employ subtle yet persuasive language to sneakily influence people. In the mid-20th century the idea famously captured popular attention, but science has only recently begun to parse the actual effects of subliminal messages.

1943: Subliminal messages were occasionally embedded in radio, film and television programs. In an animated short featuring Daffy Duck in 1943, for example, the words "BUY BONDS" appear briefly on screen. Nobody knew whether these messages would influence people, but they figured it couldn't hurt to try.

1957: James Vicary, a market researcher, claimed that by flashing the words "Eat Popcorn" and "Drink Coca-Cola" during a movie for a fraction of a second, he significantly increased the sale of these snacks. Five years later he admitted he had faked the study. By that time, however, the public had grown concerned—and advertisers and government agencies intrigued—about the manipulative power of these messages.

Late 1960s–1980s: Scientific studies throughout the 1960s, 1970s and 1980s tended to discredit the claims that subliminal messages could subtly influence behavior. One study, for instance, showed that flashing the words "Hershey's Chocolate" on a series of slides during a lecture did not influence whether students purchased Hershey's products during a 10-day period.

1990s: Although many studies continued to discredit the claim that subliminal messages carried any psychological weight, other research started to uncover subtle effects. In one such study from 1992, participants viewed images of a person engaged in a normal daily activity. After each image, researchers quickly flashed a photograph: half the viewers saw positive, uplifting content, and half saw negative content. Those who saw negative messages reported thinking of the photographed person in a more damaging light.

Early 2000s: Research continued to show that subliminal messages do influence our perceptions; the effect is just subtler than we thought.

2006: Studies have shown subliminal messages may work in advertising after all, in certain situations. For example, a 2006 study found that participants flashed an image of a brand-name drink, in this case Lipton Ice Tea, were more likely to choose that brand to quench their thirst. This association only held up, however, if participants were already thirsty. (Another provocative study showed that embedding images related to thirst in an episode of *The Simpsons* actually made people thirstier.)

2007: Subliminal messages may also enhance academic performance. In a 2007 study, researchers flashed students hidden words related or unrelated to intelligence, such as "talent" and "grass," respectively, before a practice exam. Those who saw the intelligence words performed better on a midterm one to four days later.

2010–2015: Imaging studies have shown that our brain responds to subliminal messages in measurable ways. Activity levels change in the amygdala, which processes emotions, the insula (involved in conscious awareness), the hippocampus (involved in processing memories) and the visual cortex.

http://nyti.ms/1WqmEOf

Smaller, Faster, Cheaper, Over: The Future of Computer Chips

At the inaugural International Solid-State Circuits Conference held on the campus of the University of Pennsylvania in Philadelphia in 1960, a young computer engineer named Douglas Engelbart introduced the electronics industry to the remarkably simple but groundbreaking concept of "scaling."

By JOHN MARKOFF SEPT. 26, 2015

Dr. Engelbart, who would later help develop the computer mouse and other personal computing technologies, theorized that as electronic circuits were made smaller, their components would get faster, require less power and become

Sitting in the audience that day was Gordon Moore, who went on to help found the Intel Corporation, the world's largest chip maker. In 1965, Dr. Moore quantified the scaling principle and laid out what would have the impact of a computer-age Magna Carta. He predicted that the number of transistors that could be etched on a chip would double annually for at least a decade, leading to astronomical increases in computer power.

His prediction appeared in Electronics magazine in April 1965 and was later called Moore's Law. It was never a law of physics, but rather an observation about the economics of a young industry that ended up holding true for a half-century.

One transistor, about as wide as a cotton fiber, cost roughly $8 in today's dollars in the early 1960s; Intel was founded in 1968. Today, billions of transistors can be squeezed onto a chip the size of a fingernail, and transistor costs have fallen to a tiny fraction of a cent.

That improvement — the simple premise that computer chips would do more and more and cost less and less — helped Silicon Valley bring startling advances to the world, from the personal computer to the smartphone to the vast network of interconnected computers that power the Internet.

In recent years, however, the acceleration predicted by Moore's Law has slipped. Chip speeds stopped increasing almost a decade ago, the time between new generations is stretching out, and the cost of individual transistors has plateaued.

Technologists now believe that new generations of chips will come more slowly, perhaps every two and a half to three years. And by the middle of the next decade, they fear, there could be a reckoning, when the laws of physics dictate that transistors, by then composed of just a handful of molecules, will not function reliably. Then Moore's Law will come to an end, unless a new technological breakthrough occurs.

To put the condition of Moore's Law in anthropomorphic terms, "It's graying, it's aging," said Henry Samueli, chief technology officer for Broadcom, a maker of communications chips. "It's not dead, but you're going to have to sign Moore's Law up for AARP."

In 1995, Dr. Moore revised the doubling rate to two-year intervals. Still, he remains impressed by the longevity of his forecast: "The original prediction was to look at 10 years, which I thought was a stretch," he said recently at a San Francisco event held to commemorate the 50th anniversary of Moore's Law.

But the ominous question is what will happen if that magic combination of improving speeds, collapsing electricity demand and lower prices cannot be sustained.

The impact will be felt far beyond the computer industry, said Robert P. Colwell, a former Intel electrical engineer who helped lead the design of the Pentium microprocessor when he worked as a computer architect at the chip maker from 1990 to 2000.

"Look at automobiles, for example," Dr. Colwell said. "What has driven their innovations over the past 30 years? Moore's Law." Most automotive industry innovations in engine controllers, antilock brakes, navigation, entertainment and security systems have come from increasingly low-cost semiconductors, he said.

These fears run contrary to the central narrative of an eternally youthful Silicon Valley. For more than three decades the industry has argued that computing will get faster, achieve higher capacity and become cheaper at an accelerating rate. It

has been described both as “Internet time” and even as the Singularity, a point at which computing power surpasses human intelligence, an assertion that is held with near religious conviction among many in Silicon Valley.

When you’re thinking that big, bumping into the limits of physics could be a most humbling experience.

“I think the most fundamental issue is that we are way past the point in the evolution of computers where people auto-buy the next latest and greatest computer chip, with full confidence that it would be better than what they’ve got,” Dr. Colwell said.

The Limits of Physics

Chips are made from metal wires and semiconductor-based transistors — tiny electronic switches that control the flow of electricity. The most advanced transistors and wires are smaller than the wavelength of light, and the most advanced electronic switches are smaller than a biological virus.

Chips are produced in a manufacturing process called photolithography. Since it was invented in the late 1950s, photolithography has constantly evolved. Today, ultraviolet laser light is projected through glass plates that are coated with a portion of a circuit pattern expressed in a metal mask that looks like a street map.

Each map makes it possible to illuminate a pattern on the surface of the chip in order to deposit or etch away metal and semiconducting materials, leaving an ultrathin sandwich of wires, transistors and other components.

The masks are used to expose hundreds of exact copies of each chip, which are in turn laid out on polished wafers of silicon about a foot in diameter.

Machines called steppers, which currently cost about $50 million each, move the mask across the wafer, repeatedly exposing each circuit pattern to the surface of the wafer, alternately depositing and etching away metal and semiconducting components.

A finished computer chip may require as many as 50 exposure steps, and the mask must be aligned with astonishing accuracy. Each step raises the possibility of infinitesimally small errors.

“I’ve worked on many parts of the semiconductor process,” said Alan R. Stivers, a physicist whose career at Intel began in 1979 and who helped introduce a dozen new semiconductor generations before retiring in 2007. “By far, lithography is the hardest.”

To build devices that are smaller than the wavelength of light, chip makers have added a range of tricks like “immersion” lithography, which uses water to bend light waves sharply and enhance resolution. They also have used a technique called “multiple pattern” lithography, which employs separate mask steps to sharpen the edges and further thin the metal wires and other chip components.

As the size of components and wires have shrunk to just a handful of molecules, engineers have turned to computer simulations that require tremendous computational power. “You are playing tricks on the physics,” said Walden C. Rhines, chief executive of Mentor Graphics, a Wilsonville, Ore., design automation software firm.

If that scaling first described by Dr. Engelbart ends, how can big chip companies avoid the Moore’s Law endgame? For one, they could turn to software or new chip designs that extract more computing power from the same number of transistors.

And there is hope that the same creativity that has extended Moore’s Law for so long could keep chip technology advancing.

If silicon is, in the words of David M. Brooks, a Harvard University computer scientist, “the canvas we paint on,” engineers can do more than just shrink the canvas.

Silicon could also give way to exotic materials for making faster and smaller transistors and new kinds of memory storage as well as optical rather than electronic communications links, said Alex Lidow, a physicist who is chief executive of Efficient Power Conversion Corporation, a maker of special-purpose chips in El Segundo, Calif.

There are a number of breakthrough candidates, like quantum computing, which — if it became practical — could vastly speed processing time, and spintronics, which in the far future could move computing to atomic-scale components.

Recently, there has been optimism in a new manufacturing technique, known as extreme ultraviolet, or EUV, lithography. If it works, EUV, which provides light waves roughly a tenth the length of the shortest of the light waves that make up the visible spectrum, will permit even smaller wires and features, while at the same time simplifying the chip-making process.

But the technology still has not been proved in commercial production.

Earlier this year ASML, a Dutch stepper manufacturer partly owned by Intel, said it had received a large order for EUV steppers from a United States customer that most people in the industry believe to be Intel. That could mean Intel has a jump on the rest of the chip-making industry.

Intel executives, unlike major competitors such as Samsung and Taiwan Semiconductor Manufacturing Company, or TSMC, insist the company will be able to continue to make ever-cheaper chips for the foreseeable future. And they dispute the notion that the price of transistors has reached a plateau.

Yet while Intel remains confident that it can continue to resist the changing reality of the rest of the industry, it has not been able to entirely defy physics.

"Intel doesn't know what to do about the impending end of Moore's Law," said Dr. Colwell.

In July, Intel said it would push back the introduction of 10-nanometer technology (a human hair, by comparison, is about 75,000 nanometers wide) to 2017. The delay is a break with the company's tradition of introducing a generation of chips with smaller wires and transistors one year, followed by adding new design features the next.

"The last two technology transitions have signaled that our cadence is closer to two and a half years than two years," Brian Krzanich, Intel's chief executive, said in a conference call with analysts.

No More 'Free Ride'

The glass-is-half-full view of these problems is that the slowdown in chip development will lead to more competition and creativity. Many semiconductor makers do not have the state-of-the-art factories now being designed by four chip manufacturers, GlobalFoundries, Intel, Samsung and TSMC.

The delays might allow the trailing chip makers to compete in markets that don't require the most bleeding-edge performance, said David B. Yoffie, a professor at Harvard Business School.

And even if shrinking transistor size doesn't make chips faster and cheaper, it will lower the power they require.

Ultra-low-power computer chips that will begin to appear at the end of this decade will in some cases not even require batteries — they will be powered by solar energy, vibration, radio waves or even sweat. Many of them will be sophisticated new kinds of sensors, wirelessly woven into centralized computing systems in the computing cloud.

What products might those chips lead to? No one knows yet, but product designers will be forced to think differently about what they're building, rather than play a waiting game for chips to get more powerful. Thanks to Moore's Law, computers have gotten smaller and smaller but have essentially followed the same concept of chips, hardware and software in a closed box.

"In the past, designers were lazy," said Tony Fadell, an electrical engineer who headed the team that designed the original iPod, and led the hardware design of the iPhone before founding Nest Labs, a maker of smart home devices like thermostats and smoke alarms.

Carver Mead, the physicist who actually coined the term Moore's Law, agrees. "We've basically had a free ride," he said. "It's really nuts, but that's what paid off."

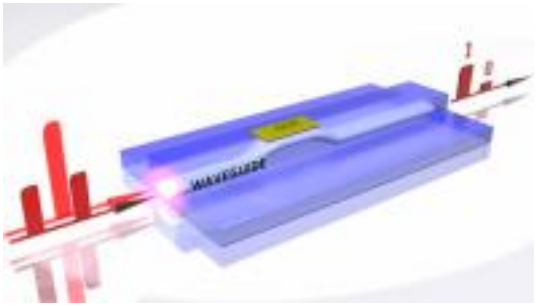
Indeed, a graying Moore's Law could be alive and well for at least another decade. And if it is not, humans will just have to get more creative.

http://bit.ly/1YI8fPD

Light-based memory chip is first to permanently store data

Creation of the first permanent optical memory on a chip

By Robert F. Service



Today's electronic computer chips work at blazing speeds. But an alternate version that stores, manipulates, and moves data with photons of light instead of electrons would make today's chips look like proverbial horses and buggies. Now, one team of researchers reports that it has created the first permanent optical memory on a chip, a critical step in that direction.

Intense light pulses (pink) write data in a patch of GST, which can be read out as digital 1s and 0s with lower intensity light (red). **C. Rios et al., Nature Photonics, Advance Online Publication (2015)**

"I am very positive about the work," says Valerio Pruneri, a laser physicist at the Institute of Photonic Sciences in Barcelona, Spain, who was not involved in the research. "It's a great demonstration of a new concept."

Interest in so-called photonic chips goes back decades, and it's easy to see why. When electrons move through the basic parts of a computer chip—logic circuits that manipulate data, memory circuits that store it, and metal wires that ferry it along—they bump into one another, slowing down and generating heat that must be siphoned away. That's not the case with photons, which travel together with no resistance, and do so at, well, light speed. Researchers have already made photon-friendly chips, with optical lines that replace metal wires and optical memory circuits. But the parts have some serious drawbacks. The memory circuits, for example, can store data only if they have a steady supply of power. When the power is turned off, the data disappear, too.

Now, researchers led by Harish Bhaskaran, a nanoengineering expert at the University of Oxford in the United Kingdom, and electrical engineer Wolfram Pernice at the Karlsruhe Institute of Technology in Germany, have hit on a solution to the disappearing memory problem using a material at the heart of rewritable CDs and DVDs. That material—abbreviated GST—consists of a thin layer of an alloy of germanium, antimony, and tellurium. When zapped with an intense pulse of laser light, GST film changes its atomic structure from an ordered crystalline lattice to an "amorphous" jumble. These two structures reflect light in different ways, and CDs and DVDs use this difference to store data. To read out

the data—stored as patterns of tiny spots with a crystalline or amorphous order—a CD or DVD drive shines low-intensity laser light on a disk and tracks the way the light bounces off.

In their work with GST, the researchers noticed that the material affected not only how light reflects off the film, but also how much of it is absorbed. When a transparent material lay underneath the GST film, spots with a crystalline order absorbed more light than did spots with an amorphous structure.

Next, the researchers wanted to see whether they could use this property to permanently store data on a chip and later read it out. To do so, they used standard chipmaking technology to outfit a chip with a silicon nitride device, known as a waveguide, which contains and channels pulses of light.

They then placed a nanoscale patch of GST atop this waveguide. To write data in this layer, the scientists piped an intense pulse of light into the waveguide. The high intensity of the light's electromagnetic field melted the GST, turning its crystalline atomic structure amorphous. A second, slightly less intense pulse could then cause the material to revert back to its original crystalline structure.

When the researchers wanted to read the data, they beamed in less intense pulses of light and measured how much light was transmitted through the waveguide. If little light was absorbed, they knew their data spot on the GST had an amorphous order; if more was absorbed, that meant it was crystalline.

Bhaskaran, Pernice, and their colleagues also took steps to dramatically increase the amount of data they could store and read. For starters, they sent multiple wavelengths of light through the waveguide at the same time, allowing them to write and read multiple bits of data simultaneously, something you can't do with electrical data storage devices. And, as they report this week in Nature Photonics, by varying the intensity of their data-writing pulses, they were also able to control how much of each GST patch turned crystalline or amorphous at any one time. With this method, they could make one patch 90% amorphous but just 10% crystalline, and another 80% amorphous and 20% crystalline. That made it possible to store data in eight different such combinations, not just the usual binary 1s and 0s that would be used for 100% amorphous or crystalline spots. This dramatically boosts the amount of data each spot can store, Bhaskaran says.

Photonic memories still have a long way to go if they ever hope to catch up to their electronic counterparts. At a minimum, their storage density will have to climb orders of magnitude to be competitive.

Ultimately, Bhaskaran says, if a more advanced photonic memory can be integrated with photonic logic and interconnections, the resulting chips have the potential to run at 50 to 100 times the speed of today's computer processors.

http://www.bbc.com/news/health-34351798

The tale of the dog behind the 'kiss of life' discovery

'Kiss of life' has an intriguing history stretching back over 100 years and, in part, it owes its discovery to the fate of an unnamed lab dog

By Lizzie Crouch and Chris Pitt Health Check

There are about 30,000 cardiac arrests every year in the UK and ten times that number in the US. It is one of the most common ways to die.

It is also one of the most common scenarios in which a bystander can save a life through CPR or cardiopulmonary resuscitation, the technique used to keep blood and oxygen pumping round the body until emergency help arrives.

This 'kiss of life' has an intriguing history stretching back over 100 years to when electricity was first being installed in domestic homes and, in part, it owes its discovery to the fate of an unnamed lab dog.

Throughout the early 1900s an electrical revolution hit America, and homes became populated with electrical appliances - everything from light bulbs to refrigerators.

But, on the down side, electrocution was a major risk to people working on the newly-installed power lines. Many died of cardiac arrests.

Shock tactics

As a result, external defibrillators had been invented to shock the heart back into rhythm without opening the chest - but they were too big and cumbersome to use outside of hospitals.

In the 1950s, the Edison Electric Institute in the US decided to sponsor researchers to investigate the effects of electrical currents on the heart.

Enter Guy Knickerbocker, a fastidious, 29-year-old graduate working under electrical engineer William Kouwenhoven in one of the labs at Johns Hopkins University in Maryland.

They were trying to improve the external defibrillator, which Kouwenhoven had invented a few years earlier.

In 1958, before the ethical treatment of animals became a serious consideration, their experiments involved testing on laboratory dogs. Knickerbocker, now 86 years old, remembers working with a colleague one day when, suddenly, one of the dogs went into cardiac arrest, or ventricle fibrillation (VF).

Normally when this happened, they would use a defibrillator to shock the dog's heart back into rhythm - but that day they were in the lab on the 12th floor and the equipment was on the fifth floor.

The notoriously slow lifts in the building meant they would never get the defibrillator to the dog in time. "There is very little chance of survival after cardiac arrest that goes on longer than five minutes," says Knickerbocker.

'Sprang to life'

Knickerbocker had a brainwave. Only a few weeks earlier he had observed that just the pressure of the defibrillator paddles on the dog's chest caused a change in blood pressure. Did this change in pressure mean that the blood was moving around the body?

He took a chance: "We started to pump the dog's chest because it seemed to be the right thing to do." Knickerbocker raced along the stairs to the fifth floor to get the defibrillator while his colleagues pressed the dog's chest for 20 minutes - four times longer than any previous successful attempt. When he arrived back with the defibrillator and administered two shocks, the dog sprang back to life.

The importance of their discovery cannot be overstated; the experiment established beyond doubt that rhythmic pressing of the chest could sustain life.

Knickerbocker says: "We had found a way to slow down the dying process, and give people time to receive defibrillation".

From pooch to people

Knickerbocker excitedly shared his discovery with cardiac surgeon, Dr Jim Jude, who worked in the next-door lab. Dr Jude immediately realised its potential, and along with Kouwenhoven, set about working out exactly where to push, how often, and how much force to apply - and found they could extend a dog's life for more than an hour

"I didn't believe the chest compression technique would ever translate to humans, and neither did a lot of my colleagues," he says today.

This included the head of surgery at Johns Hopkins at that time who wanted the team to provide a lot of evidence before he let them publish their findings.

However Dr Jude was convinced the dog-saving technique could work on people.

The chest compression technique, he realised, could be used to simulate up to 40% of normal cardiac activity. The only problem was that there was no-one to test it on.

A little over a year later, a 35-year-old woman, who was admitted for a gall bladder operation at Johns Hopkins, reacted badly to the anaesthetic and went into cardiac arrest. Dr Jude immediately began applying rhythmic, manual pressure to her chest. Within two minutes her heart started again and she went on to have the operation and make a full recovery.

'Happy and proud'

This led Kouwenhoven, Jude and Knickerbocker to publish their discovery in a paper in 1960.

"Anyone, anywhere, can now initiate cardiac resuscitative procedures," the authors concluded. "All that is needed are two hands."

In collaboration with another research group who were looking at ventilation techniques, they developed modern CPR.

Now it is taught across the world and in some countries it is also taught in schools.

The American Heart Association estimates that CPR provided immediately after sudden cardiac arrest can double or triple a victim's chance of survival.

How to save someone's life with CPR

Using chest compressions and mouth-to-mouth resuscitation is the best way to increase someone's chances of survival, but hands-only CPR is always a good option on its own.

- ***Place the heel of your hand on the breastbone at the centre of the person's chest. Place your other hand on top of your first hand and interlock your fingers.***
- ***Position yourself with your shoulders above your hands.***
- ***Using your body weight (not just your arms), press straight down by 5-6 cm on their chest, then repeat until an ambulance arrives.***
- ***Try to perform 100-120 chest compressions a minute.***

Knickerbocker is philosophical about their achievement. "After everything died down, I never dwelled on our work in the lab that often. But I was happy and proud that it had worked out so well." "Then, recently, I saw some statistics on the internet, counting up the number of people successfully resuscitated using CPR. It was over five million. I was astounded, of course."

He adds wryly, "This doesn't take into account numerous pets around the country that have also benefited from chest compressions. But it's still a lot."

http://bit.ly/1LUTeQT

NASA to announce a 'major science finding' about Mars in anticipated press conference Monday

NASA is teasing a major discovery on Mars, but they're being tight-lipped and won't reveal the mystery until a Monday press conference, CNN reports.

The space agency will announced a "major science finding" at 11:30 a.m. Eastern, and will broadcast the event live on NASA TV and from its website, in a press release that announces a "Mars mystery solved."

Some are speculating based on the list of experts slated to make the announcement that NASA has found evidence of water, Business Insider reports. And where there's water, there's the prospect of life.

NASA currently has two rovers on Mars — Spirit and Opportunity. One of the goals of the agency is to determine whether life on Mars ever existed.

In July, NASA called a press conference to announce that one of its spacecrafts found a planet similar to Earth, dubbed Kepler-452b, CNN reports. The agency hopes to send humans to Mars in the 2030s.

http://bit.ly/1LGYaNz

Ground zero for climate change: the tropics were first to feel the definite effects in the 1960s

Places near the equator, with less natural climate variation, were the first to see humanity's climate fingerprint.

Over the past century our climate has changed as greenhouse gas concentrations in the atmosphere have increased. Today we see the impacts of climate change in increasing numbers of extreme heat records while cold records decline, as well as in rising sea levels, the disappearance of land-based ice, and a host of other phenomena.

It is through these multiple lines of evidence that a scientific consensus has emerged telling us the impacts of climate change are not just a problem for the future – they have already arrived. But the question remains: at what point over the past century did climate change first make its presence felt?

Our new study goes some way to answering this question. Using state-of-the-art climate models, we and our colleagues investigated the statistical properties of temperatures and precipitation to see if they have been radically altered by climate change and, if so, when this disruption became evident. We focused on extremes in climate, as these can have large impacts on society and ecosystems.

We call the point in time when the human influence on climate becomes clear the "time of anthropogenic emergence".

Climate scientists have long expected to see the earliest detectable evidence of the influence of climate change in temperatures. By contrast, increases in rainfall extremes driven by climate change are expected, but it has been difficult to determine when they would appear.

The results of our study were intriguing. For temperature extremes, we found that the earliest simulated emergence occurred in the tropics in the mid-to-late 20th century, generally from 1960 onwards. This is because there is less year-to-year variation in temperature extremes near the equator than at higher latitudes, so less warming is required before the human fingerprint becomes clear.

The models suggested that the impact on average temperatures appeared even earlier in some regions of Africa and in the Pacific nations north of Australia, first becoming apparent in the 1940s.

As flora and fauna in this region are adapted to a narrow range of temperatures, even a small amount of warming can have large impacts.

Closer to the poles, the time of anthropogenic emergence arrived later. In terms of average temperatures, most countries show clear climate change impacts today, although parts of the United States and Russia are only seeing their first clear indications this decade.

Still waiting for the rain to change

Interestingly, our models showed that the emergence of a human fingerprint on precipitation extremes has not happened yet. But there are indications that it will emerge in winter over much of Russia, Canada and Northern Europe during this decade and the next. To ensure confidence in our findings, we tested them using several climate models and simulations.

There was strong agreement that the fingerprint in both average and extreme temperatures has already emerged across the globe. Fewer models indicated that a clearly discernible climate signal had emerged in heavy precipitation up to 2014, although there was a simulated increase in such events.

In summary, our analysis suggests that a human fingerprint on temperature extremes has already emerged and that it appeared in equatorial regions first. We can expect the effects of human-induced climate change on winter precipitation extremes in mid-to-high northerly latitudes to become clear soon, with an increase in the intensity of heavy precipitation days expected.

This work shows where the effects of human-induced climate change are being felt earliest. This provides a guide to where adaptation to climate change is needed. Of course, risk exposure to climate change impacts is made up of multiple factors including the ability to adapt to the change, as well as the relative magnitude of the change in the climate.

The tropics, where the earliest effects of climate change on temperatures seem to have occurred, also tend to be less economically developed and have less capability to adapt to climate change. These countries may require assistance in adapting to the warmer climate they are already experiencing.

http://cnet.co/1FsI6OE

Want to upgrade your dreams to HD? A psychologist needs your brain

A psychology student wants to see if a certain vitamin can make sleepy people have more lucid dreams. Is it Vitamin Zzzzzzzzzzz?

Everyone has at least one dream they wish they could rewind and play back in their head. Unfortunately, TiVo's not working on any brain implants as far as we know. You may, however, be able to make your dreams more lucid and vivid simply by taking a vitamin.

That is, at least, the premise Denholm Aspy, a psychology student at the University of Adelaide in Australia, wants to explore. Aspy, who's studying lucid dreaming for his Ph.D., plans to conduct a study to see how people can gain more

control over their dreams by recruiting some local dreamers, according to the university. Lucid dreaming means being aware that you're dreaming while you're dreaming.

Some think the secret to having more lucid dreams may lie in Vitamin B6, which is found in foods such as cereal grains, vegetables, fish and eggs and helps the brain produce the neurotransmitters serotonin and norepinephrine. Aspy's experiment is based on several previous studies, including a preliminary one published in 2002 in the journal Perception and Motor Skills that examines the effects of Vitamin B6 on dreams.

The scientists behind the 2002 study recruited 12 college-age students -- a very small sample -- and gave them either a dose of the vitamin or a placebo to take before they went to sleep. They also interviewed the students during the five days the experiment lasted and asked them to recall and describe their dreams. Those who had taken higher doses of the vitamin had more salient dreams with increased vividness and more of an ability to observe emotions than those who had taken small doses or no vitamins at all, according to the study's summary.

The study theorizes that Vitamin B6 transforms the amino acid tryptophan into serotonin during REM (rapid eye movement) sleep. Serotonin then wakes up the brain while the person is still sleeping and makes them more aware of their imaginary surroundings.

Aspy's study will replicate that experiment, but it will also compare the effects other B vitamins may have on people's dreams using a larger pool of participants. Aspy is looking for 150 people to participate in the study, according to a story from the Australian Broadcasting Company. Aspy says the vitamin may be able to do more than just give people's dreams a high-definition upgrade.

"Previous research suggests that lucid dreaming has many potential benefits," Aspy said in the statement. "For example, it may be possible to use lucid dreaming for overcoming nightmares, treating phobias, creative problem solving, refining motor skills and even helping with rehabilitation from physical trauma."

This treatment might be able to get rid of nightmares? That's terrific news! You hear that, grizzly bear with my ex-girlfriend's face that chases in me in my dreams and always tries to kill me with its switchblade lobster claws? You're about to find yourself on the unemployment line!