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Low-dose lithium reduces side effects from most common treatment for Parkinson's disease

Buck Institute research provides further validation that low-dose lithium could be repurposed as a therapy for the incurable neurodegenerative disorder

Low-dose lithium reduced involuntary motor movements - the troubling side effect of the medication most commonly used to treat Parkinson's disease (PD) - in a mouse model of the condition that is diagnosed in about 60,000 Americans each year. The third in a series of studies from the Andersen lab involving PD and low-dose lithium, the results add to mounting evidence that low-doses of the psychotropic drug could benefit patients suffering from the incurable, degenerative condition.

This study, published online in Brain Research, involved Parkinsonian mice that were given Carbidopa/Levodopa (sold as Sinemet®), a drug used to boost levels of the neurotransmitter dopamine, which is lost in PD. While the medication remains the single most effective agent in the management of PD symptoms, long-term use causes its own side effects, among them abnormal involuntary movements or AIMS. Buck professor and senior scientist Julie Andersen, PhD, says AIMS become problematic for 30 percent of patients after four to six years of treatment with Sinemet, with 90 percent of patients suffering from the complication after nine years of chronic use. "For patients these side effects are just as devastating as the freezing that is associated with PD." "In our mice we saw significant behavioral improvement."

In this study, Andersen and her team dosed the mice with an amount of lithium equivalent to about a quarter of what humans receive for the treatment of psychiatric diseases. Researchers found that lithium boosted the expression of tyrosine hydroxylase which increases dopamine synthesis via the inhibition of calpain-1, an enzyme that normally reduces dopamine synthesis.

In earlier studies, Andersen's team found that low-dose lithium was protective in two different mouse models of PD. Treatment in mice with a human mutation for PD began when the animals reached late middle-age, the human equivalent of about 60, which is the average age of onset of Parkinson's in humans. "We clearly saw a prevention of the motor difficulties we would expect to see in the animals," said Andersen. "The treatment also protected the area of the brain that is normally damaged by Parkinson's."

Plans for a clinical trial of low-dose lithium for PD patients are in early stages. "This study suggests potential therapeutic benefit in PD," said David K. Simon, MD, PhD, Associate Professor of Neurology at Harvard Medical School in Boston. Simon chairs the Scientific Review Committee for the Parkinson's Study Group, a not-for-profit network of Parkinson's Centers. "One caveat is that other agents that have shown clear efficacy in this model of PD have subsequently failed to show benefit in clinical studies in PD (e.g. CoQ10, creatine, and pioglitazone). However, this study provides additional evidence on top of prior work from Dr. Andersen's lab and others that lithium may have therapeutic potential in PD, which is a hypothesis that should be tested in clinical trials," he said.

Lithium is a naturally occurring element, not a 'developed' molecule like most medications. It was approved by the FDA for the treatment of bipolar disorder in 1970 and has shown to be effective for treating mood disorders and suicidal thoughts. Previous studies suggest that at low doses lithium has a protective effect in other neurodegenerative diseases including Alzheimer's and Huntington's.

Citation: The combination of lithium and L-Dopa/Carbidopa reduces MPTP-induced abnormal involuntary movements (AIMs) via calpain-1 inhibition in a mouse model: relevance for Parkinson's disease therapy.

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Other Buck Institute contributors include: Rebecca R. Riley and Anand Rane. Corresponding author Y. Hwan Kim, a former member of the Andersen lab, is now in the Department of Biological Sciences, Delaware State University, Carol A. Lazzara, from Delaware State University also contributed to the work.

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A cataclysmic event of a certain age

Geologist James Kennett and an international team narrow the date of an anomalous cooling event most likely triggered by a cosmic impact

At the end of the Pleistocene period, approximately 12,800 years ago- -- give or take a few centuries -- a cosmic impact triggered an abrupt cooling episode that earth scientists refer to as the Younger Dryas.

New research by UC Santa Barbara geologist James Kennett and an international group of investigators has narrowed the date to a 100-year range, sometime between 12,835 and 12,735 years ago. The team's findings appear today in the Proceedings of the National Academy of Sciences.

The researchers used Bayesian statistical analyses of 354 dates taken from 30 sites on more than four continents. By using Bayesian analysis, the researchers were able to calculate more robust age models through multiple, progressive statistical iterations that consider all related age data.

"This range overlaps with that of a platinum peak recorded in the Greenland ice sheet and of the onset of the Younger Dryas climate episode in six independent key records," explained Kennett, professor emeritus in UCSB's Department of

Earth Science. "This suggests a causal connection between the impact event and the Younger Dryas cooling."

In a previous paper, Kennett and colleagues conclusively identified a thin layer called the Younger Dryas Boundary (YDB) that contains a rich assemblage of high-temperature spherules, melt-glass and nanodiamonds, the production of which can be explained only by cosmic impact. However, in order for the major impact theory to be possible, the YDB layer would have to be the same age globally, which is what this latest paper reports.

This map shows the Younger Dryas Boundary locations that provided data for the analysis. **UCSB**

"We tested this to determine if the dates for the layer in all of these sites are in the same window and statistically whether they come from the same event," Kennett said. "Our analysis shows with 95 percent probability that the dates are consistent with a single cosmic impact event."

All together, the locations cover a huge range of distribution, reaching from northern Syria to California and from Venezuela to Canada. Two California sites are on the Channel Islands off Santa Barbara.

However, Kennett and his team didn't rely solely on their own data, which mostly used radiocarbon dating to determine date ranges for each site. They also examined six instances of independently derived age data that used other dating methods, in most cases counting annual layers in ice and lake sediments.

Two core studies taken from the Greenland ice sheet revealed an anomalous platinum layer, a marker for the YDB. A study of tree rings in Germany also showed evidence of the YDB, as did freshwater and marine varves, the annual laminations that occur in bodies of water. Even stalagmites in China displayed signs of abrupt climate change around the time of the Younger Dryas cooling event.

"The important takeaway is that these proxy records suggest a causal connection between the YDB cosmic impact event and the Younger Dryas cooling event," Kennett said. "In other words, the impact event triggered this abrupt cooling.

"The chronology is very important because there's been a long history of trying to figure out what caused this anomalous and enigmatic cooling," he added. "We suggest that this paper goes a long way to answering that question and hope that this study will inspire others to use Bayesian statistical analysis in similar kinds of studies because it's such a powerful tool."

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Some vaccines support evolution of more-virulent viruses

First confirmation of theory that some vaccines could allow more-virulent versions of a virus to survive

Scientific experiments with the herpesvirus such as the one that causes Marek's disease in poultry have confirmed, for the first time, the highly controversial theory that some vaccines could allow more-virulent versions of a virus to survive, putting unvaccinated individuals at greater risk of severe illness. The research has important implications for food-chain security and food-chain economics, as well as for other diseases that affect humans and agricultural animals.

"The challenge for the future is to identify other vaccines that also might allow more-virulent versions of a virus to survive and possibly to become even more harmful," said Andrew Read, an author of the paper describing the research, which will be published in the July 27, 2015 issue of the scientific journal PLoS Biology. Read is the Evan Pugh Professor of Biology and Entomology and Eberly Professor in Biotechnology at Penn State University.

"When a vaccine works perfectly, as do the childhood vaccines for smallpox, polio, mumps, rubella, and measles, it prevents vaccinated individuals from being sickened by the disease, and it also prevents them from transmitting the virus to others," Read said. These vaccines are a type that is "perfect" because they are designed to mimic the perfect immunity that humans naturally develop after having survived one of these diseases. "Our research demonstrates that another vaccine type allows extremely virulent forms of a virus to survive -- like the one for Marek's disease in poultry, against which the poultry industry is heavily reliant on vaccination for disease control," said Venugopal Nair, who led the research team in the United Kingdom where the experimental work related to this study was carried out. Nair is the head of the Avian Viral Diseases program at the Pirbright Institute, which also hosts the OIE Reference Laboratory on Marek's disease. "These vaccines also allow the virulent virus to continue evolving precisely because they allow the vaccinated individuals, and therefore themselves, to survive, Nair said.

Less-than-perfect vaccines create a 'leaky' barrier against the virus, so vaccinated individuals sometimes do get sick, but typically with less-virulent symptoms. Because the vaccinated individuals survive long enough to transmit the virus to others, the virus also is able to survive and to spread throughout a population. "In our tests of the leaky Marek's-disease virus in groups of vaccinated and unvaccinated chickens, the unvaccinated died while those that were vaccinated survived and transmitted the virus to other birds left in contact with them," Nair said. "Our research demonstrates that the use of leaky vaccines can promote the

evolution of nastier 'hot' viral strains that put unvaccinated individuals at greater risk."

The theory tested by the research team was highly controversial when it first was proposed over a decade ago. The team's experiments now show, for the first time, that the modern leaky vaccines, widely used in the agricultural production of poultry, can have precisely the effect on evolution of more-virulent strains of the virus that the controversial theory predicted.

Marek's disease used to be a minor disease that did not do much harm to chickens in the 1950s, but the virulence of the virus has evolved and today it even is capable of killing all the unvaccinated birds in poultry flocks, sometimes within 10 days. "Even though the Marek's disease virus is much nastier now than it was in the 1950s, it is becoming increasingly rare and now it causes relatively minor problems in the poultry industry because almost every chicken in agricultural production worldwide is vaccinated against the disease," Read said. If you can vaccinate all the individuals in a population against a virus, it does not matter if the virus has become super virulent so long as the vaccine continues to be effective."

The virus for Marek's disease is very virulent, but the virus causing avian influenza can be even worse. "The most-virulent strain of avian influenza now decimating poultry flocks worldwide can kill unvaccinated birds in just under three days," Read said. The vaccine against avian influenza is a leaky vaccine, according to Read. "In the United States and Europe, the birds that get avian influenza are culled, so no further evolution of the virus is possible," Read said. "But instead of controlling the disease by culling infected birds, farmers in Southeast Asia use vaccines that leak -- so evolution of the avian influenza virus toward greater virulence could happen."

The research has implications for human health, as well. The World Health Organization recently reported laboratory-confirmed cases in China of human infection with the avian influenza virus, including a number of deaths. "We humans never have experienced any contagious disease that kills as many unvaccinated hosts as these poultry viruses can, but we now are entering an era when we are starting to develop next-generation vaccines that are leaky because they are for diseases that do not do a good job of producing strong natural immunity -- diseases like HIV and malaria," Read said.

"Vaccines for human diseases are the least-expensive, most-effective public-health interventions we ever have had," Read said. "But the concern now is about the next-generation vaccines. If the next-generation vaccines are leaky, they could drive the evolution of more-virulent strains of the virus." He said it is critical now to determine as quickly as possible that the Ebola vaccines that now are in clinical trials are not leaky -- that they completely prevent the transmission of the Ebola virus among people. "We do not want the evolution of viral diseases as deadly as Ebola evolving in the direction that our research has demonstrated is possible with less-than-perfect, leaky vaccines," Read said.

The researchers recommend rigorous testing and vigilant monitoring of next-generation vaccines to prevent the runaway evolution of more-virulent strains of viruses that their research has confirmed can occur with leaky vaccines. "If some day we have a malaria vaccine or an HIV vaccine, of course we should use those vaccines, but we would be in significant danger if those vaccines turned out to be leaky and we had not developed effective ways to eradicate any strains that might become more virulent," Read said.

Read also recommends vaccination for individual protection. "When evolution toward more-virulent virus strains takes place as a result of vaccination practices, it is the unvaccinated individuals who are at the greatest risk. Those who are not vaccinated will be exposed, without any protection, to the hottest strains of a virus. Our research provides strong evidence for the importance of getting vaccinated."

In addition to Read, other members of the research team include Susan J. Baigent, Claire Powers, Lynda B. Kgosana, Luke Blackwell, Lorraine P. Smith, and Venugopal K. Nair at the Pirbright Institute in the United Kingdom; David A. Kennedy at Penn State University and the National Institutes of Health; and Stephen W. Walkden-Brown at the University of New England in Australia. The experiments were done in a specialized pathogen-containment facility at The Pirbright Institute in the United Kingdom.

Funding for this research was provided by the National Institutes of Health Institute of General Medical Sciences (R01GM105244) and by the U.K. Biotechnology and Biological Sciences Research Council as part of the joint NSF-NIH-USDA Ecology and Evolution of Infectious Diseases program.

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Who Were the First People to Eat Chickens?

A find in Israel shows evidence of chicken consumption from as early as 400 B.C.E.

Given the ubiquity of poultry on plates today, it may come as a surprised to learn that the first domesticated chicken was not for eating but for fighting. Humans raised fowl for cockfights starting in Southeast Asia and China as early as 10,000 years ago, but their meat wasn't enjoyed until later. Now researchers investigating an ancient city in Israel have found what they think is the earliest evidence that chickens were kept for food.

For *NPR*, Dan Charles reports on the find from Maresha, a city that enjoyed its peak during 400 to 200 BC. There, archeologists including Lee Perry-Gal, a doctoral student in archeology at the University of Haifa, found more than a thousand chicken bones bearing the marks of the knives used to butcher them.

Critically, they found twice as many female remains as male ones. The ladies don't fight, so all the signs point to chickens headed for dinner plates. Charles writes that something happened in Maresha to make the people think of chickens as food:

Maybe, in the dry Mediterranean climate, people learned better how to raise large numbers of chickens in captivity. Maybe the chickens evolved, physically, and became more attractive as food.

But Perry-Gal thinks that part of it must have been a shift in the way people thought about food. "This is a matter of culture," she says. "You have to decide that you are eating chicken from now on."

The researchers published their findings in *Proceedings of the National Academy of Sciences*. They write that the earliest evidence of large-scale chicken eating in Europe only pops up during the first century B.C.E., at least 100 years later than the finds in Israel.

From the streets and houses of Maresha, the chicken's popularity started to boom. In recent years, the popularity of chicken on U.S. plates has finally surpassed that of beef. Now, Americans eat more than 80 pounds of chicken per person every year.

http://nyti.ms/1Uh6xSf

Cellular 'Cheaters' Give Rise to Cancer

Review shows how cancer and similar kinds of cellular cheating arise not only in most, if not all, multicellular organisms

Maybe it was in "some warm little pond," Charles Darwin speculated in 1871, that life on Earth began. A few simple chemicals sloshed together and formed complex molecules. These, over great stretches of time, joined in various combinations, eventually giving rise to the first living cell: a self-sustaining bag of chemistry capable of dividing and spawning copies of itself.

While scientists still debate the specifics, most subscribe to some version of what Darwin suggested - genesis as a fortuitous chemical happenstance. But the story of how living protoplasm emerged from lifeless matter may also help explain something darker: the origin of cancer. As the primordial cells mutated and evolved, ruthlessly competing for nutrients, some stumbled upon a different course. They cooperated instead, sharing resources and responsibilities and so giving rise to multicellular creatures - plants, animals and eventually us.

Each of these collectives is held together by a delicate web of biological compromises. By surrendering some of its autonomy, each cell prospers with the whole. But inevitably, there are cheaters: A cell breaks loose from the interlocking constraints and begins selfishly multiplying and expanding its territory, reverting to the free-for-all of Darwin's pond. And so cancer begins.

Although we are getting better at preventing or controlling these rebellions, cancer is an inescapable consequence of multicellularity. A fascinating review, published last month in Philosophical Transactions B, shows how cancer and similar kinds of cellular cheating arise not only in mammals, birds, reptiles, fish, insects and other animals, but also in plants, fungi - in most, if not all, multicellular organisms.

In "Cancer Across the Tree of Life: Cooperation and Cheating in Multicellularity," researchers at the Institute for Advanced Study in Berlin show how maverick cells in species after species engage in the kind of pathological behavior that can bring down any society.

In a healthy organism, a cell replicates only as frequently as needed to maintain the population and allow for modest growth. Cancer cells begin reproducing wildly, consuming more than their share of resources and spewing poisons that degrade the environment and reshape it to their own advantage.

Through a process called differentiation, normal cells specialize, becoming skin cells, nerve cells, bone cells and so forth. There is a division of labor. But cancer cells "dedifferentiate," abandoning their assigned roles and pursuing a course beneficial only to themselves.

Under normal circumstances, a cell that goes berserk is quickly eliminated through a mechanism called programmed cell death, or cellular suicide. Cancer cells defeat this safeguard. They refuse to die.

No wonder cancer has become a metaphor for human excess - overpopulation and consumption, environmental pollution, the concentration of resources among a hyperacquisitive 1 percent.

The paper in Philosophical Transactions describes cancerlike phenomena in almost every niche of the biosphere. There is even a kind of growth, calicoblastic epithelioma, occurring among colonies of corals.

A photograph included in the paper shows a tumorous protrusion on the mushroom Agaricus bisporus. In another image, the top of a saguaro cactus erupts in elaborate curlicues of uncontrolled growth called fasciations - pathological but so visually arresting that "crested cacti" are valued by collectors.

The writhing distortions reminded me of those I've induced in weeds I sprayed with an herbicide called triclopyr. According to the manufacturer's literature, the chemical is believed to work by mimicking growth hormones called auxins, causing plant cells to crazily multiply. It's like chemotherapy in reverse, inducing something akin to cancer.

Not all biologists would agree that every instance described in the paper should be classified as cancerlike. What is clear from the abundance of examples is that multicellular life is a continual struggle between competition and cooperation. Tip the balance too far, and the result might be a malignancy.

Fasciation in saguaro cactus

In the long run of evolution, the trade-offs between cellular freedom and communalism have frequently paid off. Multicellularity, imperfect as it must be, can be so advantageous that it has evolved independently a number of times during the history of the biosphere.

Most of Earth's biomass still consists of individual actors - bacteria and other single-celled creatures.

Often, however, these microbes also cede some of their independence, banding into primitive collectives, like the invisible biofilms that coat surfaces of hospital equipment or thrive in our mouths as dental plaques. These mutual support societies can be all but invincible to antibiotics.

Yet here too, some research suggests, cooperation can give rise to cheating. Taking advantage of the sustenance and shelter provided by the biofilm, some bacteria will squander resources and thrive at the expense of the others - a microscopic tragedy of the commons.

Even cancer cells, once they gain the upper hand, may also begin cooperating with one another - to the benefit of the tumor and to the peril of its host.

As the cancerous cells divide and mutate, they diverge into separate lineages, or "subclones," each with different abilities.

In a deadly symbiosis, one family of cells might manufacture a substance that benefits the others, which in turn makes other chemicals the tumor needs to grow and colonize remote parts of the body.

Through a complex chemical dance, cancer cells can even beguile healthy cells into doing their bidding, acting in ways that promote the malignancy. It's a strategy all too familiar in life: cooperate just enough to gain your competitors' trust and then betray them for your own advantage.

In the end, there are no winners. The cancer destroys its own ecosystem and dies with its host.

http://www.eurekalert.org/pub_releases/2015-07/sjha-bs072815.php

Barrow scientists 'rewrite' history books

Brain surgery saved Russian general who helped defeat Napoleon

Researchers at Barrow Neurological Institute have spent years of medical sleuthing across three continents to uncover a brain surgery that changed history.

After more than two-years of international investigation, the scientists have concluded that Napoleon likely would have conquered Russia in 1812 if not for the life-saving brain surgery performed on Russian general Mikhail Kutuzov by the French surgeon Jean Massot, who operated on Kutuzov after bullets twice passed through his head.

"It's a story of how medicine changed the course of civilization," says Mark C. Preul, MD, PhD, and chair of neurosurgery research director at Barrow, which is part of Dignity Health St. Joseph's Hospital and Medical Center.

Dr. Preul led the research team in collaboration with fellow Barrow Neurological Institute researchers Dr. Sergiy V. Kushchayev and Dr. Evgenii Belykh and five other researchers. The study, titled "Two bullets to the head and an early winter: fate permits Kutuzov to defeat Napoleon at Moscow," was published in the Journal of Neurosurgery.

For more than two centuries, history has focused on Kutuzov's incredible story. He survived being shot in the head in 1774 and 1788 and went on to become one of Russia's legendary heroes by repelling Napoleon's invaders. His story has been called a miracle. But by combing primary sources in Russian and French, the Barrow team found that Massot played a critical role in the drama, employing techniques that foreshadowed modern neurosurgery to help Kutuzov survive what appeared to be mortal wounds.

"We wanted to find out what really happened and basically identify this surgeon who saved Mikhail Kutuzov," Dr. Preul says. "Massot's facts were somewhat buried. He is at the vanguard of surgical technique. He uses incredibly modern techniques that we still use today."

What they found was evidence that the first bullet wound, sustained in a battle with the Turks in Crimea in 1774, had destroyed Kutuzov's frontal lobe. That explained Kutuzov's erratic behavior after the injury - but it also provided clues to the brilliant strategy he used to defeat Napoleon and his seemingly invincible Grande Armée.

Kutuzov's injury most likely impaired his ability to make decisions. Eye witnesseses remark about his altered personality after the first gun shot wound. So instead of challenging Napoleon's superior forces in the autumn of 1812, Kutuzov put off a confrontation. He ordered Moscow burned and fled with his army to safety east of Moscow. Napoleon's army pursued, invading Moscow, but lacking

food and supplies, succumbed to a horribly brutal early Russian winter. Napoleon abandoned the army in December and returned to Paris in defeat.

"The other generals thought Kutuzov was crazy, and maybe he was," Dr. Preul says. "The brain surgery saved Kutuzov's life, but his brain and eye were badly injured. However ironically the healing resolution of this situation allowed him to make what turned out to be the best decision. If he had not been injured, he may well have challenged Napoleon and been defeated."

Dr. Preul says some questions about Kutuzov's injuries - and Massot's operations on them - can't be completely answered without a medical examination. Kutuzov's body has not been examined since his autopsy shortly after his death in April 1813. But this much is clear: Kutuzov would not have been in command without Massot's efforts.

"Although some would say fate allowed the brilliant Russian general, who became the personification of Russian spirit and character, to survive two nearly mortal head wounds, the best neurosurgical technique of the day seems to have been overlooked as a considerable part of Kutuzov's success," the researchers wrote.

http://bit.ly/1OYLe4L

At Tiny Scales, a Giant Burst on Tree of Life

A new technique for finding and characterizing microbes has boosted the number of known bacteria by almost 50 percent, revealing a hidden world all around us.

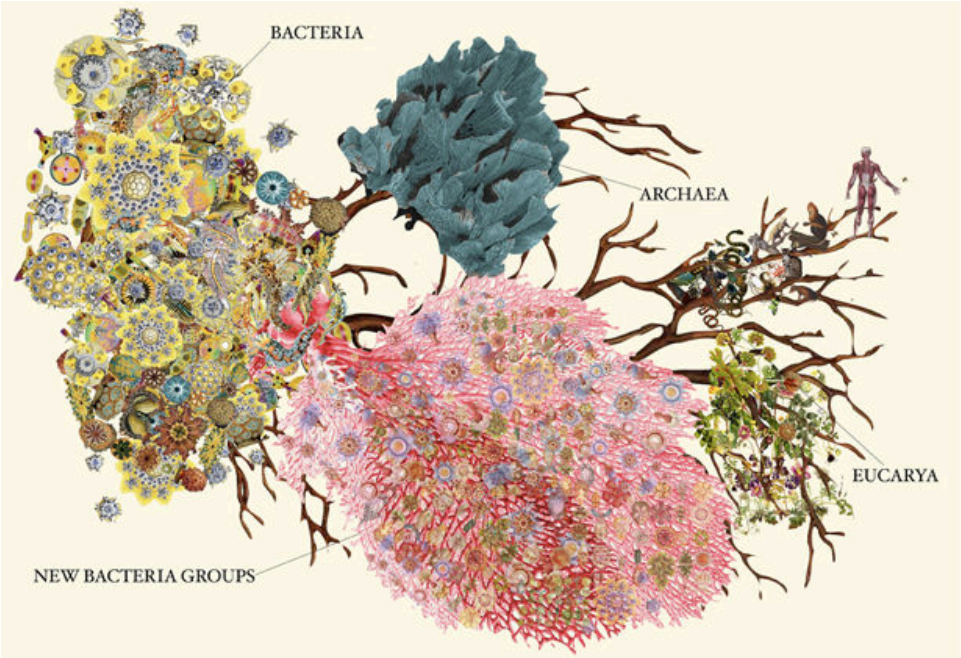
By: Kevin Hartnett

It used to be that to find new forms of life, all you had to do was take a walk in the woods. Now it's not so simple. The most conspicuous organisms have long since been cataloged and fixed on the tree of life, and the ones that remain undiscovered don't give themselves up easily. You could spend all day by the same watering hole with the best scientific instruments and come up with nothing.

Maybe it's not surprising, then, that when discoveries do occur, they sometimes come in torrents. Find a different way of looking, and novel forms of life appear everywhere.

A team of microbiologists based at the University of California, Berkeley, recently figured out one such new way of detecting life. At a stroke, their work expanded the number of known types - or phyla - of bacteria by nearly 50 percent, a dramatic change that indicates just how many forms of life on earth have escaped our notice so far.

"Some of the branches in the tree of life had been noted before," said Chris Brown, a student in the lab of Jill Banfield and lead author of the paper. "With this study we were able to fill in many gaps."



Travis Bedel **for Quanta Magazine**

Life's Finest Net

As an organizational tool, the tree of life has been around for a long time. Lamarck had his version. Darwin had another. The basic structure of the current tree goes back 40 years to the microbiologist Carl Woese, who divided life into three domains: eukaryotes, which include all plants and animals; bacteria; and archaea, single-celled microorganisms with their own distinct features. After a point, discovery came to hinge on finding new ways of searching.

"We used to think there were just plants and animals," said Edward Rubin, director of the U.S. Department of Energy's Joint Genome Institute. "Then we got microscopes, and got microbes. Then we got small levels of DNA sequencing."

Jill Banfield and collaborators at the University of California, Berkeley, have discovered new groups of very small bacteria, expanding the tree of life.

DNA sequencing is at the heart of this current study, though the researchers' success also owes a debt to more basic technology. The team gathered water samples from a research site on the Colorado River near the town of Rifle, Colo. Before doing any sequencing, they passed the water through a pair of increasingly fine filters - with pores 0.2 and 0.1 microns wide - and then analyzed the cells captured by the filters. At this point they already had undiscovered life on their

hands, for the simple reason that scientists had not thought to look on such a tiny scale before.

"Most people assumed that bacteria were bigger, and most bacteria are bigger," Rubin said. "[Banfield] has shown that there are whole populations that are very small."

The researchers extracted the DNA from the cellular material and sent it to the Joint Genome Institute for sequencing. What they got back was a mess. Imagine being handed a box of pieces from thousands of different jigsaw puzzles and having to assemble them without knowing what any of the final images look like. That's the challenge researchers face when performing metagenomic analysis - sequencing scrambled genetic material from many organisms at once.

The Berkeley team began the reassembly process with algorithms that assembled bits of the sequenced genetic code into slightly longer strings called contigs.

"You no longer have tiny pieces of DNA, you have bigger pieces," Brown said. "Then you figure out which of these larger pieces are part of a single genome."

This part of the process, in which contigs are combined to reconstruct the genome sequence, is called genome binning. To execute it, the researchers relied on another set of algorithms, customized for the task by Itai Sharon, a co-author of the study. They also assembled some of the genomes manually, making decisions about what goes where based on the fact that some characteristics are consistent for a given genome. For example, the percentage of Gs and Cs will be similar on any part of an organism's DNA.

When the assembly was complete, the researchers had eight full bacterial genomes and 789 draft genomes that were roughly 90 percent complete. Some of the organisms had been glimpsed before; many others were completely new.

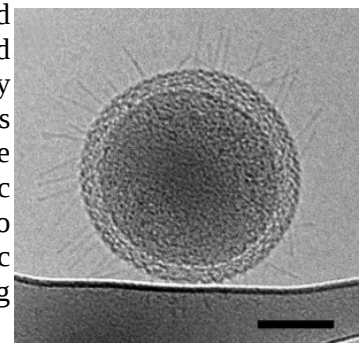
The reason no one had found these organisms before is that the traditional method used to search for small forms of life doesn't work for everything. That method involves the 16S rRNA gene, which is often compared to a fingerprint because the genetic code it contains is unique for every organism. When confronted with a DNA stew, like the one from the water samples in Rifle, scientists use substances called primers to draw out and amplify all the 16S rRNA genes. The problem is, not all 16S rRNA genes react with the primers, rendering some organisms effectively invisible.

"The primers don't work as well as people would like them to," Brown said. "We showed that many of the sequences we reconstructed would have been missed by the traditional 16S amplification-type method."

By reconstructing complete or nearly complete genomes, Brown and his collaborators were able to locate 16S rRNA genes and identify organisms without relying on primers. The group published their results in the July 9 issue of *Nature*.

Some of the newly discovered bacteria have hairlike structures on their surface.

The fuller genomic picture they created also allowed them to tease out traits of the life forms they'd discovered. All the organisms they found have very short genomes, about one million base pairs (compare that to *E. coli*, which has about five million), and they all have minimal metabolic function, requiring them to use fermentation to generate energy. They are also missing many basic biosynthetic pathways and need help making nucleotides and amino acids.



Courtesy of Jill Banfield

"They must be dependent on other organisms in some capacity to survive. This also explains why no one's been able to grow them in the lab," Brown said.

A New Domain?

The discovery of new organisms is fairly cut and dried: Either you've found one or you haven't. Cataloging organisms, fitting them into the tree of life, involves more judgment calls.

The researchers divided the 789 organisms into 35 phyla - 28 of which were newly discovered - within the domain bacteria. They based the sorting on the organisms' evolutionary history and on similarities in the code on the organisms' 16S rRNA genes - those with at least 75 percent of their code in common went into the same phylum.

With these new additions, there are now roughly 90 identified bacterial phyla. This is a lot more than there were a year ago, but also far fewer than the 1,300 to 1,500 phyla that microbiologists estimate we'll have once a complete accounting is finished. Recent advances in genetic sequencing and genome binning make Brown and Banfield optimistic, though, that it won't be long before we've mapped them all.

"I think that much of the tree of life will come into view in the next few years," Banfield wrote in an email.

Of course, no sooner do we think we've seen everything than we come up with a new way to see. Rubin thinks that the development of tools like the ones used in the new study make the search for life "a growth industry," and he thinks it's likely that growth will occur in surprising ways.

"Looking at things from a different angle may offer that possibility of a fourth domain," he said - an equal partner to bacteria, archaea and eukaryotes. "There will always be novel stuff that will teach us foundational info about how life operates."

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High intensity training helps ease arthritis pains

10 weeks of twice-weekly sessions eased arthritic inflammation

It's a disease that sneaks up on you. Fingers and toes slowly but surely become stiff and painful. A nice morning stretch is no longer all it takes to get your body moving. Arthritis is a chronic illness that sinks its claws into your body, and causes inflammation in your joints. Arthritis can destroy your joints, which causes weakness and loss of movement. Patients with arthritis often have reduced endurance, and an are at an increased risk of cardiovascular disease.

Affects three times as many women as men

Arthritis affects about one per cent of the population, and about three times as many women as men. Mostly adults are affected, but the disease can occur in children as well. Treatment helps to ease symptoms, but the disease is chronic.

"This is why it is especially important for arthritis patients to keep fit and work on their cardiovascular endurance," says Anja Bye, a researcher at the K. G. Jebsen Centre for Exercise in Medicine-- Cardiac Exercise Research Group (CERG) at NTNU. Until now, however, there has been little documentation of how exercise actually affects arthritic joints.

Hard work-out sessions are best

"Previously, studies have showed that moderate intensity work-out sessions can help improve endurance without inducing pain or inflammation, or damaging joints," says Bye.

She explains that numerous studies show that high-intensity interval training is much more effective for improving endurance than moderate intensity training.

"This is true regardless if you're sick or healthy, young or old. We wanted to see if patients with arthritis could handle high intensity training and see the same positive effects," says Bye.

Lost weight as well

After ten weeks of hard training on a spinning bike twice a week, Bye saw no adverse effects on her study's participants, a group of women with arthritis.

"Rather, we saw a tendency for there to be less inflammation, at least as measured by the inflammation marker CRP, and the participants of the study experienced a solid increase maximum oxygen intake, meaning that they reduced their risk of cardiovascular disease," Bye said. The participants also saw a small reduction in BMI, body fat per cent and waist measurement, as well as an increase in muscle mass as a result of the training period.

The study took place at CERG's training studio at St. Olavs Hospital. The participants warmed up for ten minutes at 70 per cent of their maximum pulse, and then did four repetitions of high intensity (85-95 per cent of max pulse) four-minute intervals. The break between each interval was about three minutes, at 70 per cent of max pulse. The total work-out session lasted about 35 minutes.

Several participants are continuing training

"The women who participated in the study found this to be a good, effective method of training, and are mostly very motivated to continue because of the progress they've seen," Bye says.

The study was a pilot to see if the idea was worth researching in depth, and consequently included only 18 women between the ages of 20-49. The study's small size means it is too early to conclude if recommended training programmes for arthritis patients should be changed, but the study suggests it might be a good idea. The Department of Rheumatology at St. Olavs Hospital is working on a study of high intensity training for different patient groups with CERG.

European Journal of Applied Physiology, May 2015.

The effects of high intensity interval training in women with rheumatic disease: a pilot study. Janne Sandstad, Dorthe Stensvold, Mari Hoff, Bjarne M. Nes, Ingerid Arbo, Anja Bye

http://www.eurekalert.org/pub_releases/2015-07/dnnl-tgo072915.php

Tiny grains of rice hold big promise for greenhouse gas reductions, bioenergy

Discovery delivers high starch content, virtually no methane emissions

Rice serves as the staple food for more than half of the world's population, but it's also the one of the largest manmade sources of atmospheric methane, a potent greenhouse gas. Now, with the addition of a single gene, rice can be cultivated to emit virtually no methane from its paddies during growth. It also packs much more of the plant's desired properties, such as starch for a richer food source and biomass for energy production, according to a study in Nature.

In addition to a near elimination of greenhouse gases associated with its growth, SUSIBA2 rice produces substantially more grains for a richer food source. The new strain is shown here (right) compared to the study's control. **Swedish University of Agricultural Sciences**

With their warm, waterlogged soils, rice paddies contribute up to 17 percent of global methane emissions, the equivalent of about 100 million tons each year. While this represents a much smaller percentage of overall greenhouse gases than carbon dioxide, methane is about 20 times more effective at trapping heat.

SUSIBA2 rice, as the new strain is dubbed, is the first high-starch, low-methane rice that could offer a significant and sustainable solution.

Researchers created SUSIBA2 rice by introducing a single gene from barley into common rice, resulting in a plant that can better feed its grains, stems and leaves while starving off methane-producing microbes in the soil.

The results, which appear in the July 30 print edition of Nature and online, represent a culmination of more than a decade of work by researchers in three countries, including Christer Jansson, director of plant sciences at the Department of Energy's Pacific Northwest National Laboratory and EMSL, DOE's Environmental Molecular Sciences Laboratory. Jansson and colleagues hypothesized the concept while at the Swedish University of Agricultural Sciences and carried out ongoing studies at the university and with colleagues at China's Fujian Academy of Agricultural Sciences and Hunan Agricultural University.

"The need to increase starch content and lower methane emissions from rice production is widely recognized, but the ability to do both simultaneously has eluded researchers," Jansson said. "As the world's population grows, so will rice production. And as the Earth warms, so will rice paddies, resulting in even more methane emissions. It's an issue that must be addressed."

Channeling carbon

During photosynthesis, carbon dioxide is absorbed and converts to sugars to feed or be stored in various parts of the plant. Researchers have long sought to better understand and control this process to coax out desired characteristics of the plant. Funneling more carbon to the seeds in rice results in a plumper, starchier grain. Similarly, carbon and resulting sugars channeled to stems and leaves increases their mass and creates more plant biomass, a bioenergy feedstock.

In early work in Sweden, Jansson and his team investigated how distribution of sugars in plants could be controlled by a special protein called a transcription factor, which binds to certain genes and turns them on or off.

"By controlling where the transcription factor is produced, we can then dictate where in a plant the carbon - and resulting sugars - accumulate," Jansson said.

To narrow down the mass of gene contenders, the team started with grains of barley that were high in starch, then identified genes within that were highly active. The activity of each gene then was analyzed in an attempt to find the specific transcription factor responsible for regulating the conversion of sugar to starch in the above-ground portions of the plant, primarily the grains.

The master plan

Upon discovery of the transcription factor SUSIBA2, for SUgar SIgnaling in BArley 2, further investigation revealed it was a type known as a master regulator. Master regulators control several genes and processes in metabolic or regulatory pathways. As such, SUSIBA2 had the ability to direct the majority of carbon to the grains and leaves, and essentially cut off the supply to the roots and soil where certain microbes consume and convert it to methane.

Researchers introduced SUSIBA2 into a common variety of rice and tested its performance against a non-modified version of the same strain. Over three years of field studies in China, researchers consistently demonstrated that SUSIBA2 delivered increased crop yields and a near elimination of methane emissions.

Next steps

Jansson will continue his work with SUSIBA2 this fall to further investigate the mechanisms involved with the allocation of carbon using mass spectrometry and imaging capabilities at EMSL. Jansson and collaborators also want to analyze how roots and microbial communities interact to gain a more holistic understanding of any impacts a decrease in methane-producing bacteria may have.

Reference: J. Su, C. Hu, X. Yan, Y. Jin, Z. Chen, Q. Guan, Y. Wang, D. Zhong, C. Jansson, F. Wang, A. Schnurer, C. Sun. Expression of barley SUSIBA2 transcription factor yields high-starch low-methane rice, Nature July 22 (online), 2015, DOI: 10.1038/nature14673

http://www.eurekalert.org/pub_releases/2015-07/sdsu-cwr072815.php

Can we restart the heart?

SDSU heart researchers mash up the incredible proliferating and survival powers of cancer cells with broken-down cardiac cells to rejuvenate the heart.

SAN DIEGO, Calif. - In a way, trying to repair age-related heart damage and trying to fight cancer are opposite problems. Your heart cells' ability to regenerate themselves and proliferate into new, young cells degrades as you get older. They simply lose their proficiency at cell division. Cancer cells, on the other hand, are too good at proliferating. They don't know when to stop, and the overgrowth results in tumors.

This is all very simplified, of course, but it's the basic model described by Mark Sussman, chief research scientist at the San Diego State University Heart Institute, who was recently selected by the American Heart Association's Basic Cardiovascular Science division to receive this year's Distinguished Achievement Award. The heart in particular seems to be resistant to developing cancerous cells.

"When's the last time you heard of anyone having heart cancer? It's almost unheard of," said Sussman.

That's not surprising from an evolutionary standpoint. If heart cells make a grave transcription error during cell division and your ticker ticks its last tock, there's no fixing the problem. So it makes sense that heart cells are incredibly careful when it comes to proliferating.

But it's this very meticulousness that makes heart disease such an intractable problem, Sussman explained. Over time, the cells burn themselves out. Their ability to repair themselves and generate fresh replacements gets progressively worse. By the time you reach old age and start experiencing symptoms of age-related heart disease, your cardiac cells are running on fumes and aren't able to properly divide into new cells.

"There's a razor's edge balancing cellular aging and cancer risk," he said.

What if you could use biotechnology to walk that razor's edge? To use the proliferative and survival properties of cancer-prone cells to rejuvenate cardiac progenitor cells -- a rare type of stem cell that replicates indefinitely into new heart cells--and get them dividing again, without forming tumors?

That's the aim of one arm of Sussman's research at SDSU. Sussman and his colleagues published a paper in the May 29 issue of the Journal of Biological Chemistry exploring the results of taking an enzyme, Pim, known to be associated with growth and survival of certain types of cancer cells, and causing it to be overexpressed in cardiac progenitor cells in mice. In healthy cells, Pim helps facilitate chromosome splitting, a key part of the cellular division process.

The gene that encodes the production of this enzyme, PIM1, is what's known as a proto-oncogene. That means that by itself, the gene doesn't cause cancer. But when it teams up with another gene, Myc, tumors are likely to form.

Fortunately, the Pim/Myc combination isn't an issue in heart progenitor cells, meaning you could tweak those cells to overexpress the PIM1 gene without raising the risk of cancer.

That's exactly what Sussman's team did. They modified mouse heart progenitor cells to overexpress PIM1 in specific locations within the cell, targeting specific locations with more of the critical Pim enzyme in hopes that it would protect against aging-related heart disease.

And it worked. Compared to controls, the mice with overexpressed PIM1 lived longer and showed stronger cell proliferation. But interestingly, the way it worked was different depending on where in the cell the gene was overexpressed.

If the researchers caused PIM1 to be overexpressed in the progenitor cell's nucleus, they saw increased proliferation into new cells. If they overexpressed the gene in a different region of the cell, the mitochondria, they found that the enzyme inhibited the cell's natural self-destruct signals, causing them to live longer.

One technique enhanced cell division, the other warded off cell death. In humans, depending on a person's individual circumstance, either or both of these effects might help restore their cardiac cells to a younger, healthier state.

Sussman and his colleagues have replicated the results with human tissue obtained from people whose hearts have failed and who are living on a ventricular assist device that pumps their blood for them. The research team is currently trying to obtain funding to do human clinical trials wherein they obtain a patient's own cardiac progenitor cells, modify them to overexpress PIM1, then put them back into the patient's heart in hopes of rejuvenating the tissue and spurring the heart to repair itself.

"We're trying to dial back the clock to when their cells had more regenerative potential," Sussman said. "By understanding how and where Pim affects these cells, we can create specialized Pim molecules that get you all the benefits of youthfulness without the risk of cancer."

http://www.eurekalert.org/pub_releases/2015-07/cru-pci072915.php

Prostate cancer is 5 different diseases

Cancer Research UK scientists have for the first time identified that there are five distinct types of prostate cancer and found a way to distinguish between them, according to a landmark study* published today in EBioMedicine.

The findings could have important implications for how doctors treat prostate cancer in the future, by identifying tumours that are more likely to grow and spread aggressively through the body.

The researchers, from the Cancer Research UK Cambridge Institute and Addenbrooke's Hospital, studied samples of healthy and cancerous prostate tissue from more than 250 men. By looking for abnormal chromosomes and measuring the activity of 100 different genes linked to the disease they were able to group the tumours into five distinct types, each with a characteristic genetic fingerprint.

This analysis was better at predicting which cancers were likely to be the most aggressive than the tests currently used by doctors - including the PSA test** and Gleason score. But, the findings need to be confirmed in clinical trials with larger groups of men.

Study author Dr Alastair Lamb, from the Cancer Research UK Cambridge Institute, said: "Our exciting results show that prostate cancer can be classified into five genetically-different types. These findings could help doctors decide on the best course of treatment for each individual patient, based on the characteristics of their tumour.

"The next step is to confirm these results in bigger studies and drill down into the molecular 'nuts and bolts' of each specific prostate cancer type. By carrying out more research into how the different diseases behave we might be able to develop more effective ways to treat prostate cancer patients in the future, saving more lives."

Prostate cancer is the most common cancer in men in the UK, with around 41,700 cases diagnosed every year. There are around 10,800 deaths from the disease each year in the UK.

Professor Malcolm Mason, Cancer Research UK's prostate cancer expert, said: "The challenge in treating prostate cancer is that it can either behave like a pussycat - growing slowly and unlikely to cause problems in a man's lifetime - or a tiger - spreading aggressively and requiring urgent treatment. But at the moment we have no reliable way to distinguish them. This means that some men may get treatment they don't need, causing unnecessary side effects, while others might benefit from more intensive treatment.

"This research could be game-changing if the results hold up in larger clinical trials and could give us better information to guide each man's treatment - even helping us to choose between treatments for men with aggressive cancers. Ultimately this could mean more effective treatment for the men who need it, helping to save more lives and improve the quality of life for many thousands of men with prostate cancer."

**Ross-Adams et al. Integration of copy number and transcriptomics provides risk stratification in prostate cancer: a discovery and validation cohort study. EBioMedicine. DOI: 10.1016/j.ebiom.2015.07.017.*

This work was funded by Cancer Research UK with support from Prostate Cancer UK.

http://bit.ly/1K0nt82

We've Modified Our Behavior So We Can Text and Walk

Texting while walking might no longer be the annoyance it once was

By Krystal D'Costa | July 29, 2015

Texting - or checking social media or reading/responding to email or reading the news or checking the weather or watching a video - while walking is a pretty ubiquitous phenomenon. It's so common that it might no longer be the annoyance it once was. Who's left to find it a nuisance? We've all been captivated by the notification icons on our phones, so virtually no one is paying attention to where they're going.

Our mobile devices are heavily integrated in our lives. In my case, it's one of the first things I reach for in the morning, and when I get out of the car my phone is often in my hand as I walk through the parking lot to the store I'm visiting. I do try to put it away while I walk to the office from the train station though. I'm very aware that the distraction may make me a target--or put me in front of a moving vehicle. Plus, I've just been reading emails on the entire train ride in. The ten minute walk to my building is a welcome break. Usually.

Another reason we may not be complaining about texting walkers is that they're less awkward. Why don't they fall down or walk into other people? Research suggests that these texters adopt protective measures to minimize the risk of accidents when walking. They're less likely to trip because they shorten their step length, reduce step frequency, lengthen the time during which both feet are in contact with the ground, and increase obstacle clearance height. Taken together this creates an exaggerated image of walking, but it apparently slows the walker enough so that he registers some of what is happening around him and can compensate for it.

The impact of texting and walking is that it slows the walker down. So we're all connected but it may take us longer to get to each other--which may be okay since we've likely texted the person that we're on our way and then given a play-by-play of our progress as we update social media along the way.

The study suggests that this outcome may be partially due to familiarity. The participants were between the ages of 18-50 years old and had owned a mobile phone for at least a month. According to data from Pew Internet, 90% of American adults own a cell phone, and two-thirds of American adults own a smart phone. Between the age group and the ownership requirement for this study, the people surveyed very likely fell into these categories.

We may be driving technology to respond to our needs in various areas, but this is one instance where we've definitely demonstrated that we're also adapting to accommodate technological change.

Licence S, Smith R, McGuigan MP, Earnest CP (2015) Gait Pattern Alterations during Walking, Texting and Walking and Texting during Cognitively Distractive Tasks while Negotiating Common Pedestrian Obstacles. PLoS ONE 10(7): e0133281. doi:10.1371/journal.pone.0133281

http://www.eurekalert.org/pub_releases/2015-07/esoc-guf072915.php

Get up for your heart health and move for your waistline

Time spent standing rather than sitting could improve your blood sugar, fats in the blood and cholesterol levels

More time spent standing rather than sitting could improve your blood sugar, fats in the blood and cholesterol levels, according to a new study published today (Friday) in the European Heart Journal [1]. The study also shows that replacing time spent sitting with time walking could have additional benefits for your waistline and body mass index (BMI).

Researchers in Australia gave activity monitors to 782 men and women, aged 36-80 years, who were taking part in the Australian Diabetes, Obesity and Lifestyle Study. The monitors were capable of determining, very accurately, how long each participant spent sleeping, sitting or lying down, standing and stepping (which includes walking and running). After providing blood samples and measurements of their blood pressure, height, weight and waist circumference, participants each wore an activity monitor on their thigh for 24 hours a day over a seven-day period. The researchers then used a statistical technique called isotemporal analysis [2] to

estimate the potential impact on health of reallocating time from sitting to standing or stepping.

Dr Genevieve Healy, senior research fellow at the School of Public Health, The University of Queensland, Australia, who led the study, said: "We found that time spent standing rather than sitting was significantly associated with lower levels of blood sugar and blood fats. Replacing sitting time with stepping was also associated with a significant reduction in waistline and BMI. While the study cannot show that less time spent sitting causes the improvements in these markers of health, the associations it reveals are consistent with what is known already about the benefits of a non-sedentary lifestyle. More work is needed to understand cause and effect."

An extra two hours per day spent standing rather than sitting was associated with approximately 2% lower average fasting blood sugar levels and 11% lower average triglycerides (fats in the blood). Extra standing time was also associated with 0.06 mmol/L higher average levels of the "good" type of cholesterol, HDL, and a 6% lower average total/HDL cholesterol ratio, which indicates an improvement in the total amount of HDL cholesterol in relation to "bad" LDL cholesterol [3].

Replacing two hours a day of sitting time with stepping was associated with an approximately 11% lower average BMI and a 7.5cm smaller average waist circumference. In addition, average blood sugar levels fell by approximately 11% and average triglycerides by 14% for every two hours spent walking rather than sitting, while HDL cholesterol was 0.10 mmol/L higher. There was no significant effect on BMI or waistline of replacing sitting time with standing.

"These findings provide important preliminary evidence that strategies to increase the amount of time spent standing or walking rather than sitting may benefit the heart and metabolism of many people," said Dr Healy. "Get up for your heart health and move for your waistline.

"This has important public health implications, given that standing is a common behaviour that usually replaces sitting, and that can be encouraged in the workplace with interventions such as sit-stand desks.

"However, it is important to say that not all sitting is bad; but if people can incorporate alternatives to sitting wherever possible, it may benefit their heart and metabolic health. Our message is to 'Stand Up, Sit Less, Move More'."

She said the study had also produced evidence of how common standing is during the waking day. "Standing takes up nearly a third of waking hours, and among this group of participants who could choose when they sat, stood or walked, the standing had health benefits. Notably, we did not measure upper body movement, so someone could be standing up doing the dishes, which involves some extra physical activity."

While the benefits to health of walking have been well established, until now the potential benefits (or harms) of replacing sitting with standing have been less well understood. The study is one of the first to look at the estimated associations between replacing time in one activity with another and its effect on markers of health, such as blood pressure, blood sugar and cholesterol levels, BMI and waist circumference. The researchers say more, larger studies are needed to confirm their findings and they hope to follow up the study participants for longer, as well as studying participants from a wider age range.

In the meantime, Dr Healy and her colleagues are working to encourage people to stand up more in their workplaces. "We are also looking to increase the amount of time spent stepping as well," she concluded.

In an accompanying editorial [4], Professor Francisco Lopez-Jimenez (MD, MSc) of the Mayo Clinic and Mayo College of Medicine (Minnesota, USA) writes that the study "provides an important addition to the wealth of scientific evidence highlighting the importance of avoiding sedentary behaviour". He writes that "the fight against sedentary behaviour cannot be won based only on the promotion of regular exercise" and that while exercise should continue to be recommended, it is important to promote non-sedentary behaviour in everyday life. "A person walking while at work for two hours, standing for another four hours, and performing some daily chores at home for another hour will burn more calories than jogging or running for 60 minutes."

He also points out that sedentary behaviour and environments that promote it are "seen as a sign of progress and economic power". For instance, poorer people are more likely to bike or walk than drive a car, and standing tickets to watch a football match or an opera will be cheaper than seated tickets. He concludes: "The unintended consequences of modern life promoting sedentary behaviours can be reversed. Health care providers, policy makers and people in general need to stand up for this. Literally."

[1] "Replacing sitting by standing or stepping: associations with cardio-metabolic risk biomarkers", by Genevieve N. Healy et al. European Heart Journal. doi:10.1093/eurheartj/ehv308

[2] The researchers used isotemporal substitution analyses to estimate the effects on health of replacing time spent in one activity with time in another. Isotemporal substitution analysis simultaneously models the specific activity being performed and the specific activity being displaced in an equal time-exchange manner, while keeping waking hours unchanged.

[3] mmol/L stands for millimoles per litre and is the standard way of measuring blood cholesterol and glucose.

[4] *"Standing for healthier lives - literally", by Francisco Lopez-Jimenez. European Heart Journal. doi:10.1093/eurheartj/ehv356*

[5] *Dr Healy is funded currently by the Australian Heart Foundation. The study was supported by the National Health and Medical Research Council of Australia and many more organisations (full details are given at the end of the paper).*

http://www.eurekalert.org/pub_releases/2015-07/uor-rft072615.php

Researchers find that Earth's magnetic shield is much older than previously thought

An older geomagnetic field suggests an early start to plate tectonics

Since 2010, the best estimate of the age of Earth's magnetic field has been 3.45 billion years. But now a researcher responsible for that finding has new data showing the magnetic field is far older.

John Tarduno, a geophysicist at the University of Rochester and a leading expert on Earth's magnetic field, and his team of researchers say they believe the Earth's magnetic field is at least four billion years old.

"A strong magnetic field provides a shield for the atmosphere," said Tarduno, "This is important for the preservation of habitable conditions on Earth."

The findings by Tarduno and his team have been published in the latest issue of the journal Science.

Earth's magnetic field protects the atmosphere from solar winds--streams of charged particles shooting from the Sun. The magnetic field helps prevent the solar winds from stripping away the atmosphere and water, which make life on the planet possible.

Earth's magnetic field is generated in its liquid iron core, and this "geodynamo" requires a regular release of heat from the planet to operate. Today, that heat release is aided by plate tectonics, which efficiently transfers heat from the deep interior of the planet to the surface. But, according to Tarduno, the time of origin of plate tectonics is hotly debated, with some scientists arguing that Earth lacked a magnetic field during its youth.

Given the importance of the magnetic field, scientists have been trying to determine when it first arose, which could, in turn, provide clues as to when plate tectonics got started and how the planet was able to remain habitable.

Fortunately for scientists, there are minerals--such as magnetite--that lock in the magnetic field record at the time the minerals cooled from their molten state. The oldest available minerals can tell scientists the direction and the intensity of the field at the earliest periods of Earth's history.

In order to get reliable measurements, it's crucial that the minerals obtained by scientists are pristine and never reached a sufficient heat level that would have allowed the old magnetic information within the minerals to reset to the magnetic field of the later time.

The directional information is stored in microscopic grains inside magnetite- a naturally occurring magnetic iron oxide. Within the smallest magnetite grains are regions that have their own individual magnetizations and work like a tape recorder. Just as in magnetic tape, information is recorded at a specific time and remains stored unless it is replaced under specific conditions.

Tarduno's new results are based on the record of magnetic field strength fixed within magnetite found within zircon crystals collected from the Jack Hills of Western Australia. The zircons were formed over more than a billion years and have come to rest in an ancient sedimentary deposit. By sampling zircons of different age, the history of the magnetic field can be determined.

The ancient zircons are tiny--about two-tenths of a millimeter--and measuring their magnetization is a technological challenge. Tarduno and his team used a unique superconducting quantum interference device, or SQUID magnetometer, at the University of Rochester that provides a sensitivity ten times greater than comparable instruments.

But in order for today's magnetic intensity readings of the magnetite to reveal the actual conditions of that era, the researchers needed to make sure the magnetite within the zircon remained pristine from the time of formation.

Of particular concern was a period some 2.6 billion years ago during which temperatures in the rocks of the Jack Hills reached 475?C. Under those conditions, it was possible that the magnetic information recorded in the zircons would have been erased and replaced by a new, younger recording of Earth's magnetic field.

"We know the zircons have not been moved relative to each other from the time they were deposited," said Tarduno. "As a result, if the magnetic information in the zircons had been erased and re-recorded, the magnetic directions would have all been identical."

Instead, Tarduno found that the minerals revealed varying magnetic directions, convincing him that the intensity measurements recorded in the samples were indeed as old as four billion years.

The intensity measurements reveal a great deal about the presence of a geodynamo at the Earth's core. Tarduno explains that solar winds could interact with the Earth's atmosphere to create a small magnetic field, even in the absence of a core dynamo. Under those circumstances, he calculates that the maximum strength of a magnetic field would be 0.6 ?T (micro-Teslas).

The values measured by Tarduno and his team were much greater than 0.6 ?T, indicating the presence of a geodynamo at the core of the planet, as well as suggesting the existence of the plate tectonics needed to release the built-up heat.

"There has been no consensus among scientists on when plate tectonics began," said Tarduno. "Our measurements, however, support some previous geochemical measurements on ancient zircons that suggest an age of 4.4 billion years."

The magnetic field was of special importance in that eon because solar winds were about 100 times stronger than today. In the absence of a magnetic field, Tarduno says the protons that make up the solar winds would have ionized and stripped light elements from the atmosphere, which, among other things, resulted in the loss of water.

Scientists believe that Mars had an active geodynamo when that planet was formed, but that it died off after four billion years. As a result, Tarduno says, the Red Planet had no magnetic field to protect the atmosphere, which may explain why its atmosphere is so thin.

"It may also be a major reason why Mars was unable to sustain life," said Tarduno.

http://www.eurekalert.org/pub_releases/2015-07/uoc--wuh072915.php

Waking up HIV

Two compounds show great potential to rouse latent virus

Highly active anti-retroviral therapy (HAART) has helped millions survive the human immunodeficiency virus (HIV). Unfortunately, HIV has a built-in survival mechanism, creating reservoirs of latent, inactive virus that are invisible to both HAART and the immune system.

But now, researchers at UC Davis have identified a compound that activates latent HIV, offering the tantalizing possibility that the virus can be flushed out of the silent reservoirs and fully cured. Even better, the compound (PEP005) is already approved by the FDA. The study was published in the journal PLOS Pathogens.

"We are excited to have identified an outstanding candidate for HIV reactivation and eradication that is already approved and is being used in patients," said lead author Satya Dandekar, who chairs the Department of Medical Microbiology and Immunology.

"This molecule has great potential to advance into translational and clinical studies."

While HAART has been quite successful - reducing HIV infection in newborns, restoring patients' immune systems and lowering viral loads to virtually undetectable levels - these therapies cannot cure the disease alone. Once treatment is discontinued, pools of latent virus reactivate, and the infection comes roaring back. As a result, patients must remain on treatment indefinitely, posing the risk of long-term toxicity.

"We've made great progress, but at the end of the day you still have more than 30 million people walking around with HIV," said Dandekar. "Without drugs, the virus can come back at the same threat level for patients. Eradicating HIV is extremely critical."

Eradication means activating latent virus and destroying it, a strategy called "shock and kill." Researchers around the world have been working on this approach, but finding the right compounds has been challenging. A successful molecule must precisely target proteins associated with HIV latency without overstimulating the immune system or wantonly activating protein master switches, such as NF-kappaB. Either outcome can generate severe side effects.

The UC Davis team may have succeeded with PEP005, the active ingredient in the FDA-approved anti-cancer drug PICATO, which increased HIV activation in patient blood samples and showed low toxicity.

However, HIV is a complicated virus and, as clinicians have discovered with HAART, must be treated through multiple means. In addition to PEP005, the researchers tested other compounds capable of reactivating HIV through different pathways. This painstaking process identified another molecule, JQ1, which works synergistically with PEP005 to maximize HIV activation. PEP005 when combined with JQ1 increased HIV activation up to 15-fold.

While these results are promising, researchers are mindful that "shock" only works when it's followed by "kill."

"First, we need to identify the best combination of latency-activating agents," said Dandekar. "Then we must help patients clear these reactivated cells. Just reactivating the HIV from latency won't be enough."

Dandekar notes that many HIV patients receiving HAART regimens have robust immune responses, which will go a long way towards clearing the virus. She also believes HIV vaccines in development could give patients an extra edge. Even a vaccine that isn't 100 percent effective at preventing transmission could boost a patient's ability to destroy reactivated virus.

However, identifying PEP005 and JQ1 as potent HIV-activators is a key step in the right direction.

"It is really exciting is that the molecule in PICATO is already approved and being used by patients," said Dandekar. "In addition to being very effective in reactivating HIV, it also works beautifully with other latency reactivating agents, is less cytotoxic and doesn't cause a major immune response."

Other authors included Erica A. Mendes, Yuyang Tang, Anne Fenton, Gregory P. Melcher, James E. K. Hildreth, George R. Thompson at UC Davis; Philipp Kaiser and Joseph K. Wong at UC San Francisco; and Daniel P. Wong at Williams College.

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http://www.eurekalert.org/pub_releases/2015-07/ku-poo073015.php

Preventable onset of myocardial infarction through coadministration of 2 drugs

Investigating coadministration of statins with ezetimibe

This news release is available in Japanese.

Ischemic heart disease is the leading cause of death in the world and second in Japan behind cancer. It causes blood vessels to become clogged or narrowed through the buildup of cholesterol plaque along the inside of artery walls. This plaque buildup restricts blood flow to the heart and leads to heart damage or heart attack. The development of a drug treatment has long been anticipated.

The administration of a statin to lower "bad" cholesterol (LDL-C) values is the standard lipid-lowering treatment for heart disease patients. However, even with a statin therapy, more than half of all patients still develop heart disease. One explanation for this is that even though the treatment with statins inhibits cholesterol synthesis in the liver, it also promotes cholesterol absorption in the small intestine.

Researchers at Kumamoto University, Japan investigated the coadministration of statins with the cholesterol absorption inhibitor ezetimibe. The study, performed by the Department of Cardiovascular Medicine at Kumamoto University in collaboration with 17 other domestic facilities, culminated in a clinical trial of patients whose LDL cholesterol levels were 100 mg/dL or more (the normal range for healthy adults is 60 - 139 mg/dL) who were also undergoing percutaneous coronary intervention to widen their arteries with a balloon or stent. Patients were divided into a statin alone treatment group and a statin + ezetimibe coadministration treatment group. The target LDL cholesterol level was 70 mg/dL or less. Changes in the volume of atheroma, which contains lipids and other debris that tend to adhere to artery walls, were assessed after 9-12 months of treatment.

"At the end of the trial, we found a clear difference between the two groups of patients." said Dr. Kenichi Tsujita, who led the research. "The average LDL cholesterol level of the group of patients treated with statin alone went from 109.8 mg/dL at the beginning of the experiment down to 73.3 mg/dL at the end. On the other hand, the average LDL level of the group that received coadministration of statin and ezetimibe went down from 108.3 mg/dL to 63.2 mg/dL. In other words, the single drug treatment group had 29% lower cholesterol, whereas the cholesterol of the combined drug treatment group was lowered by approximately 40%."

Furthermore, the volume of atheroma was significantly different between both groups. Patients in the combination group had greater negative remodeling of blood vessels and showed outstanding plaque regression compared to the single treatment group.

"Significant plaque regression was especially seen in patients with acute coronary syndrome." said Professor Ogawa, head of Cardiovascular Medicine at Kumamoto University. "The research showed that the coadministration treatment efficiently and safely lowered LDL cholesterol levels, reduced the absorption of cholesterol in the body and produced greater regression of coronary artery plaque compared to statin only therapies. This treatment for high risk coronary artery disease patients is expected to be very useful clinically."

This study was published in the "Journal of the American College of Cardiology" online on July 28, 2015. http://content.onlinejacc.org/article.aspx?articleid=2411160&resultClick=1

http://www.eurekalert.org/pub_releases/2015-07/jhub-mrc073015.php

Mouth rinse could help predict recurrence of HPV-related oropharyngeal cancers

In small study, patients with HPV traces post-treatment were more likely to have cancer recurrence; finding could lead to new monitoring protocols

Johns Hopkins University Bloomberg School of Public Health

Oropharyngeal cancer patients who were found to have detectable traces of human papillomavirus type 16 (HPV16) in their saliva following cancer treatment are at an increased risk for recurrence, a study led by researchers at the Johns Hopkins Bloomberg School of Public Health has found.

The oropharynx is the area of the upper throat that includes the back of the tongue, the soft palate, the tonsils and the walls of the throat. Oropharyngeal cancer accounts for 2.8 percent of new cancers in the United States; it is often treated successfully with surgery.

In a small study, seven percent (five of 67) of oropharyngeal cancer patients who had HPV16 DNA in their oral rinse at the time of diagnosis were later found to still have traces of HPV16 DNA in their oral rinse following treatment. Of these, all developed a local recurrence of the cancer. The finding, believed to be the first of its kind, could lead to to new follow-up protocols for oropharyngeal cancer patients, the researchers say.

The study is published July 30 in the journal JAMA Oncology.

"It's a very small number so we have to be somewhat cautious," says Gypsyamber D'Souza, PhD, an associate professor in the Department of Epidemiology at the Johns Hopkins Bloomberg School of Public Health and a member of the Sidney Kimmel Comprehensive Cancer Center. "The fact that all of the patients with persistent HPV16 DNA in their rinses after treatment later had recurrence meant that this may have the potential to become an effective prognostic tool."

For their study, researchers tracked 124 patients who had been diagnosed with oropharyngeal cancer, collecting oral rinses from patients at the time of diagnosis and again following treatment, at nine, 12, 18 and 24 months after diagnosis. Patients were asked to rinse and gargle with Scope mouthwash. Of the 124 patients, slightly over half had oral HPV16 DNA in their oral rinse at the time of their cancer diagnosis. Most patients no longer had HPV DNA detectable in their oral rinse after completing treatment, but some did.

The researchers do not know if the presence of HPV16 DNA in the post-treatment rinse means that the treatment did not completely eradicate the cancer in the first place or if the cancer returned. Either way, the finding suggests that a simple oral rinse could be a powerful diagnostic tool for the reappearance of this type of oral cancer.

HPV is associated with several types of cancer, most notably cervical and oral cancers. Incidence of HPV-associated cancers is increasing in the United States and the virus is responsible for the majority of oropharyngeal cancer here. HPV-positive oropharyngeal cancer generally has a better prognosis than HPV-negative cancer, but like other cancers, it can recur, potentially in up to 25 percent of cases. HPV-related oropharyngeal cancer responds well to surgical treatment, but the success of surgical treatment decreases if the cancer is caught after it has spread to other parts of the body. The researchers hope that the detection of HPV DNA in oral rinses may enable earlier detection of recurrence and, therefore, better overall prognosis should the cancer recur.

Moreover, most of the recurrences observed in this study were localized oropharyngeal cancer and not cancers that spread to other regions of the body. "Those that had HPV DNA detected in their mouth after treatment had a much higher risk of local recurrence," says D'Souza.

Researchers say that in this study disease recurrence was diagnosed roughly seven months after the detection of HPV16 DNA in the oral rinse. Presence of HPV16 DNA in oral rinses may allow for the detection of cancer recurrence before any other clinical signs or symptoms, which enables earlier treatment options.

"There was a lead time of several months between when we detected HPV16 DNA in the rinse and when they were diagnosed with recurrence," says D'Souza. "If we had known at the rinse time, it would have given a lead time for treatment."

D'Souza stresses that this type of testing is new. She also notes that this is a rare cancer, and that recurrence is even rarer still.

"It should be reassuring that most people who have been treated for HPV-related oropharyngeal cancers are cured and there is no HPV16 DNA detected in their mouths, but among those that did recur, this was an important potential predictor," she says.

"Prognostic Implication of Persistent Human Papillomavirus 16 DNA Detection in Oral Rinses for Human Papillomavirus-Related Oropharyngeal Carcinoma" was written by Eleni M Rettig, MD; Alicia Wentz, MA; Marshall R Posner, MD; Neil Gross, MD; Robert I Haddad, MD; Maura L Gillison, MD, PhD; Carole Fakhry, MD; Harry Quon, MD; Andrew G Sikora, MD PhD; William J Stott, CCRP; Jochen H Lorch, MD; Christine G Gourin, MD; Yingshi Guo, MS; Weihong Xiao, MD; Brett A Miles, DDS, MD; Jeremy D Richmon, MD; Peter E Anderson, MD; Krzysztof J Misiukiewicz, MD; Christine H Chung, MD; Jennifer E Gerber, MSc; Shirani D Rajan, MSPH; Gypsyamber D'Souza, PhD.

The research was supported by grants from the Johns Hopkins Richard Gelb Cancer Prevention Award (GD), the Oral Cancer Foundation (GD), the National Institute of Dental and Craniofacial Research (NIDCR) and the National Institutes of Health (NIH) Research Training in Otolaryngology grant 2T32DC000027-26 (EMR).

http://www.bbc.com/news/technology-33718311

Supercomputers: Obama orders world's fastest computer

The president has asked US scientists to build the fastest supercomputer

By Chris Baraniuk Technology reporter

President Obama has signed an executive order calling for the US to build the world's fastest computer by 2025. The supercomputer would be 20 times quicker than the current leading machine, which is in China. It would be capable of making one quintillion (a billion billion) calculations per second - a figure which is known as one exaflop.

A body called the National Strategic Computing Initiative (NSCI) will be set up to research and build the computer.

The US is seeking the new supercomputer, significantly faster than today's models, to perform complex simulations, aid scientific research and national security projects. It is hoped the machine would help to analyse weather data for more accurate forecasts or assist in cancer diagnoses by analysing X-ray images.

A blog post on the White House website also suggests it could allow NASA scientists to model turbulence, which might enable the design of more streamlined aircraft without the need for extensive wind tunnel testing. Such a computer would be called an exascale machine.

Bigger models

Richard Kenway at the University of Edinburgh says he thinks the plan is "spot on" in terms of strategy, bringing together both the ambition to develop new hardware and also improved analysis of big data. He explained the computer could aid the development of personalised medicines, tailored to specific individuals. "Today, drugs are designed for the average human and they work OK for some people but not others," he told the BBC. "The real challenge in precision medicine is to move from designing average drugs to designing drugs for the individual because you can know their genome and their lifestyle."

There could also be benefits in long-term climate modelling, according to Mark Parsons at the Edinburgh Parallel Computing Centre (EPCC).

Currently, climate scientists attempt to model how the Earth's climate will evolve in coming years, but the accuracy of these predictions is severely limited.

Today's fastest supercomputer, the Tianhe-2 in China's National Computer Centre, Guangzhou, performs at 33.86 petaflops (quadrillions of calculations per second), almost twice as fast as the second-quickest machine, which is American.

For Parsons, the latest US initiative is a clear attempt to challenge the dominance of the Chinese in this field. "The US has woken up to the fact that if it wants to remain in the race it will have to invest," he told the BBC.

£60m electricity bill

Both Kenway and Parsons point out that the challenges of building an exascale computer are not trivial and would require years of research and development.

Chief among the obstacles, according to Parsons, is the need to make computer components much more power efficient. Even then, the electricity demands would be gargantuan. "I'd say they're targeting around 60 megawatts, I can't imagine they'll get below that," he commented. "That's at least £60m a year just on your electricity bill."

Efforts to construct an exascale computer are not entirely new. Recently, IBM, the Netherlands Institute for Radio Astronomy (ASTRON) and the University of Groningen announced plans to build one to analyse data from the Square Kilometre Array (SKA) radio telescope project. SKA will be built in Australia and South Africa by the early 2020s.

http://nyti.ms/1eOwjqD

Rosetta's Philae Lander Discovers a Comet's Organic Molecules

Philae, the little lost lander that the European Space Agency dropped on a comet last November, is still lost.

By KENNETH CHANG JULY 30, 2015

Although it woke up last month and has made contact several times with the Rosetta orbiter, which is examining Comet 67P/Churyumov-Gerasimenko from miles away, Philae has not been heard from for more than two weeks, and the sporadic communications have not been long enough for the lander to resume its scientific work.

But even the 60 hours of observations it conducted immediately after landing have provided an intriguing wealth of data about the composition, structure and properties of the comet, described in detail in seven papers published Thursday by the journal Science.

Jean-Pierre Bibring, the lead scientist for the Philae lander, highlighted the discovery of large dark grains, a millimeter or more wide, which appeared to be made of complex light-absorbing organic molecules. "This is fundamental," Dr. Bibring said. "We didn't know that."

Organic, carbon-based molecules are thought to be the building blocks for life but can come together in nonbiological chemical reactions. These molecules, Dr. Bibring said, formed in the cold of space, possibly even before the grains clumped together to become the comet. The bouncing landing itself provided data that the scientists would not otherwise have obtained.

Philae, the size of a washing machine, landed on Comet 67P almost exactly where it had aimed. But a downward-pushing thruster failed to fire, and two harpoons meant to secure the lander to the surface did not deploy. Philae bounded back into space. It scraped the rim of a crater and bounced a second time off the surface before settling awkwardly in a smaller crater, in the shadow of a cliff without enough sunlight for its solar panels to recharge its battery.

At the targeted landing site, the feet of the lander sank about 10 inches into a soft granular material that absorbed much of its kinetic energy. Stephan Ulamec, the project manager for Philae, likened it to landing in a sandbox.

But at the final resting place, the comet proved unexpectedly hard - at least as hard as compacted snow - and the lander was unable to hammer in a sensor rod to measure temperatures below the surface.

"That was a surprise to us," said Tilman Spohn, the principal investigator for that instrument. "We didn't think it would be that hard." But the scientists also know that the material is highly porous. Dr. Spohn said the measured thermal properties were consistent with a hard ice layer covered by an inch or so of dust.

About 20 minutes after the first touchdown, in the middle of the first jump across the surface, two similar instruments took a sniff of the surroundings to identify molecules in the vicinity. One detected four organic compounds that had never been seen emanating from a comet. The other detected chains of formaldehyde molecules. Those are probably just hints of more complex organic molecules.

"It's clearly got a story to tell," said Ian P. Wright, the principal investigator for one of the instruments.

In appearance, Comet 67P has proved to be quite strange and varied, with a two-lobed shape resembling a rubber duck and a range of terrains. But a radar experiment, sending a signal from Philae to the Rosetta orbiter through the comet, revealed that the material, at least within the head portion, was fairly homogeneous beneath the surface.

The Rosetta spacecraft dropped the Philae lander onto Comet 67P/C-G last November. After seven months, the lander is now beginning to emerge from hibernation. If Philae comes to life again, the landing acrobatics may yet turn out to be more blessing than curse.

Had it landed as planned, Philae would have overheated and died by the spring. But instead it is in a cooler, more protected spot, and engineers expected it to wake up when more light reached its solar panels as Comet 67P came closer to the sun.

Philae indeed woke up June 13, but communication has been intermittent. Mission controllers were encouraged by 12 minutes of interrupted transmissions between Philae and the Rosetta orbiter on July 9, but it has been out of touch since.

Dr. Ulamec said the position of Philae may have shifted, its antenna perhaps blocked, and there appeared to be a problem with at least one of its transmitters. Otherwise the lander seemed to be in good condition.

Meanwhile, the Rosetta orbiter has continued its observations of the comet, examining, for example, how the outpourings of gas interact with the wind of high-speed particles from the sun. At present, it has moved to take a closer look at the southern hemisphere of the comet, which is emerging from shadow into sunlight. The current position, where the comet itself blocks any radio transmissions, makes it impossible to hear anything from Philae even if Philae were broadcasting, but next week Rosetta will return north and listen again.

“I’m an optimist,” Dr. Ulamec said. “I think we should have contact again.”

In two weeks, on Aug. 13, Comet 67P will make its closest approach to the sun, some 115 million miles away, halfway between the orbits of Earth and Mars.

As to where Philae is precisely, nobody knows for sure.

Triangulation of the radar signals has narrowed the position to an area about 110 feet by 70 feet. Rosetta’s cameras have spotted glints of light that could be Philae. The location might not be confirmed until late next year, when Comet 67P has moved away from the sun, and Rosetta can descend for a closer look.

By then, Philae will certainly be dead, but that information could retroactively refine the measurements the scientists already possess.

http://s.nikkei.com/1hqdVip

Japanese longevity reaches record

Japanese women have the highest average life span in the world.

TOKYO -- The average life expectancy of the Japanese population hit a new record in 2014, with women attaining the ripe old age of 86.83 years and men living 80.5 years, according to a government report released Thursday.

"Declining death rates from cancer, heart disease, pneumonia and stroke were contributing factors," said the Japanese health ministry, which conducted the study. Because of advances in medical technology and growing health consciousness, the ministry believes it is possible for the average life expectancy to grow even further.

The Japanese have been extending their longevity almost the entire postwar era. Japanese women had an average life expectancy of 80 years in 1984 while men attained that milestone in 2013. Women added 0.22 year since 2013, and men gained 0.29 year. The gap between women and men continued to shrink from 2003's peak of 6.97 years.

Divided by gender, Japanese women are first in the world for longevity, barely beating the 86.75 years lived by Hong Kong women. Japanese men are tied for third with Switzerland and Singapore, bested only by Hong Kong's 81.17 and Iceland's 80.8. Iceland's age is based on 2013 numbers.

Japan's health ministry estimates 87.3% of women and 74.1% of men born in 2014 will live to be at least 75. For those who will live to see their 90th year, the percentage drops to 48.3% for women and 24.2% for men.

Among those born in 2014, 47.8% of women and 52.2% of men will die of cancer, heart disease or stroke. However, it is still estimated that women born last year will extend their life spans by 6.02 years while men will live 7.28 more years for those who do not succumb to those ailments.

Aside from the absolute life span, the health ministry also publishes average "healthy life expectancies" indicating the number of years a person lives without being handicapped by health conditions. For women it is 74.21 years while it is 71.19 years for men.

http://www.eurekalert.org/pub_releases/2015-07/uom-dab073015.php

Discovery about brain protein causes rethink on development of Alzheimer's disease

Copper hypothesis questioned

Researchers at the University of Melbourne have discovered that a protein involved in the progression of Alzheimer's disease also has properties that could be helpful for human health. The discovery helps researchers better understand the complicated brain chemistry behind the development of Alzheimer's disease, a condition that affects hundreds of thousands of Australians.

An international team of researchers, led by Dr Simon Drew at the University of Melbourne and Prof Wojciech Bal at the Polish Academy of Sciences, has revealed that a shorter form of a protein called beta amyloid, may act as a sponge that safely binds a metal that can damage brain tissue when it's in excess.

Researchers have been intensely interested in the role of beta-amyloid in the development of Alzheimer's disease. This is because clumps of the protein are formed in brains of people with the illness.

In the late 1990s, high levels of copper were discovered within these clumps. Copper is essential to health, but too much can produce harmful free radicals.

Many scientists began to suspect that this copper might be contributing to the disease. They found that beta-amyloid can bind to copper indiscriminately and allow it to produce these damaging free radicals. Closer analysis of beta amyloid protein has revealed different sizes. A good proportion of beta amyloid is missing the first three links at the start of the protein's chain-like structure.

"This short form has been overlooked by most researchers since the composition of beta amyloid was first identified 30 years ago," Dr Simon Drew explains.

"We know that the shorter form of beta amyloid is present in the diseased brain, but we now know that it is abundant in healthy brains as well.

"The small change in length makes a huge difference to its copper binding properties. We found that the short form of the protein is capable of binding copper at least 1000 times stronger than the longer forms. It also wraps around the metal in a way that prevents it from producing free radicals.

"Given these properties and its relative abundance, we can speculate this type of beta amyloid is protective. It's very different from the current view of how beta amyloid interacts with biological copper."

So far, therapies aimed at lowering the production of beta amyloid have shown only a modest ability to slow cognitive decline and the number of people affected by the Alzheimer's disease continues to grow.

Dr Drew and the team from Poland are now working to develop a method for identifying the copper-bound form of the short beta amyloid in the body.

This will enable them to screen how much copper it holds in the brain, whether it safely escorts the copper from one place to another, and how this may change in ageing and disease. "If a beneficial role in copper balance can be established, it's still possible to have too much of a good thing," Dr Drew said.

"As the amount of beta amyloid in the brain increases during Alzheimer's disease, the shorter form can also clump together and this may interfere with its normal function. Higher levels of the short form may further enable it to soak up copper from other places where it is needed. It could be a Jekyll and Hyde scenario."

Dr Drew's research was published in Angewandte Chemie.

http://www.eurekalert.org/pub_releases/2015-07/uoe-bhu072915.php

Butterflies heat up the field of solar research

The humble butterfly could hold the key to unlocking new techniques to make solar energy cheaper and more efficient, pioneering new research has shown.

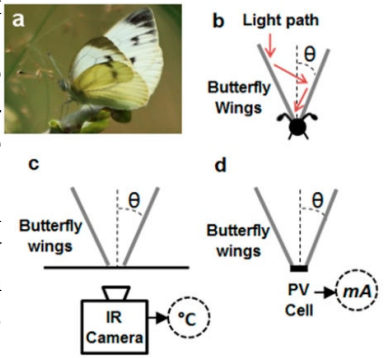
A team of experts from the University of Exeter has examined new techniques for generating photovoltaic (PV) energy - or ways in which to convert light into power. They showed that by mimicking the v-shaped posture adopted by Cabbage White butterflies to heat up their flight muscles before take-off, the amount of power produced by solar panels can increase by almost 50 per cent.

Crucially, by replicating this 'wing-like' structure, the power-to-weight ratio of the overall solar energy structure is increased 17-fold, making it vastly more efficient.

The research by the team from both the Environment and Sustainability Institute (ESI) and the Centre for Ecology and Conservation, based at the University of Exeter's Penryn Campus in Cornwall, is published in the leading scientific journal, Scientific Reports.

Professor Tapas Mallick, lead author of the research said: "Biomimicry in engineering is not new. However, this truly multidisciplinary research shows pathways to develop low cost solar power that have not been done before."

The Cabbage White butterflies are known to take flight before other butterflies on cloudy days - which limit how quickly the insects can use the energy from the sun to heat their flight muscles. This ability is thought to be due to the v-shaped posturing, known as reflectance basking, they adopt on such days to maximise the concentration of solar energy onto their thorax, which allows for flight.



(a), Photograph of large white (taken by Richard ffrench-Constant) with wings in 'V-shape' basking posture. (b), Schematic diagram of theoretical light concentration towards thorax via reflection from wings of butterfly. (c), Method for measuring wing angle effect on 'body' temperature (°C). (d), Method for measuring wing angle effect on current output (mA) from solar cell in place of 'body'.

Furthermore, specific sub-structures of the butterflies' wings allow the light from the sun to be reflected most efficiently, ensuring that the flight muscles are warmed to an optimal temperature as quickly as possible. The team of scientists therefore investigated how to replicate the wings to develop a new, lightweight reflective material that could be used in solar energy production.

The team found that the optimal angle by which the butterfly should hold its wings to increase temperature to its body was around 17 degrees, which increased the temperature by 7.3 degrees Centigrade compared to when held flat. They also showed that by replicating the simple mono-layer of scale cells found in the butterfly wings in solar energy producers, the could vastly improve the power-to-weight rations of future solar concentrators, making them significantly lighter and so more efficient.

Professor Richard ffrench-Constant, who conducts world-leading research into butterfly mimicry at the University of Exeter, said: "This proves that the lowly Cabbage White is not just a pest of your cabbages but actually an insect that is an

expert at harvesting solar energy." The paper, White butterflies as solar photovoltaic concentrators, by Katie Shanks, Dr Senthilarasu Sundaram, Professor Richard ffrench-Constant and Professor Tapas Mallick from the University of Exeter, is available online.

http://www.bbc.com/news/health-33733711

Ebola vaccine is 'potential game-changer'

A vaccine against the deadly Ebola virus has led to 100% protection and could transform the way Ebola is tackled, preliminary results suggest.

By James Gallagher Health editor, BBC News website

There were no proven drugs or vaccines against the virus at the start of the largest outbreak of Ebola in history, which began in Guinea in December 2013. The World Health Organization (WHO) said the findings, being published in the Lancet, could be a "game-changer". Experts said the results were "remarkable".

This trial centred on the VSV-EBOV vaccine, which was started by the Public Health Agency of Canada and then developed by the pharmaceutical company Merck. It combined a fragment of the Ebola virus with another safer virus in order to train the immune system to beat Ebola. A unique clinical trial took place in Guinea. When a patient was discovered, their friends, neighbours and family were vaccinated to create a "protective ring" of immunity.

Analysis

This could be the breakthrough the world has been waiting for. There is caution as the results are still preliminary, with more data coming in. But officials at the WHO believe the effectiveness of the vaccine will end up being between 75% and 100%. Had such a vaccine been available 18 months ago then thousands of lives could have been saved.

There are still other vaccines being trialled - notably from GSK and Johnson & Johnson - although as the number of cases continues to fall it is becoming increasingly difficult to prove how effective they are. Ebola will inevitably come again. The hope now is that the legacy of this unprecedented outbreak will be a vaccine that means a tragedy of this scale can never be repeated.

One hundred patients were identified in the trial between April and July and then close contacts were either vaccinated immediately, or three weeks later.

In the 2,014 *(??)* close contacts who were vaccinated immediately there were no subsequent cases of Ebola. In those vaccinated later there were 16 cases, according to the results published in the Lancet medical journal.

'Promising'

The WHO says it is so far 100% effective, although that figure may change as more data is collected. Close contacts of Ebola patients in Guinea will now be vaccinated immediately. And since the vaccine has been shown to be safe, that process will also be extended to include children.

Médecins sans Frontières (MSF) is involved with this research, and is part of a parallel trial for frontline healthcare workers. Medical director Bertrand Draguez said the Lancet results should spur instant action.

"With such high efficacy, all affected countries should immediately start and multiply ring vaccinations to break chains of transmission and vaccinate all frontline workers to protect them."

Marie-Paule Kieny, an assistant director general at the WHO told BBC News: "It is certainly promising. We have seen that where rings have been vaccinated, the transmission has stopped. "Prior to vaccination there were cases, cases, cases. The vaccine arrives and 10 days later the cases are flat. "It could be a game-changer because previously there was nothing, despite the disease being identified 40 years ago. "When there is a new outbreak this vaccine will be put to use to stop the outbreak as soon as possible to not have the terrible disaster we have now."

More than 11,000 people have died from Ebola and nearly 28,000 have been infected. The sheer scale of the 2014-15 outbreak led to an unprecedented push on vaccines - and a decade's work has been condensed into around 10 months.

The number of cases has fallen - and in the week up to July 26th 2015 there were just four cases in Guinea and three in Sierra Leone.

Prof John Edmunds, from the London School of Hygiene & Tropical Medicine, helped design the trial: "The development has been at an absolutely unprecedented speed. "This is very good news, these are very significant results, the epidemic is not over and this shows we have another potential weapon. "The trial is still continuing, these are interim results which need confirming, but there's now light at the end of the tunnel."

Dr Jeremy Farrar, the director of the Wellcome Trust medical charity, said this was a "remarkable result" and was the product of international collaboration.

He added: "Our hope is that this vaccine will now help bring this epidemic to an end and be available for the inevitable future Ebola epidemics."

http://bit.ly/1IUN6fQ

Hackers Have Figured Out How to Control Smart Rifles

With the right code, hackers can control guns from afar

After a year of research, a pair of security researchers have figured out how to hack a $13,000 smart rifle. By exploiting security weaknesses in a computer-powered sniper rifle, Runa Sandvik and Michael Auger devised a method that can not only prevent a gun from firing or hitting a target, but can throw the user’s aim off enough to hit an entirely different bullseye.

"You can make it lie constantly to the user so they'll always miss their shot," Sandvik tells Andy Greenberg for *Wired*.

The pair spent a year dissecting and examining a "self-aiming" rifle made by TrackingPoint, a company specializing in computer-assisted guns. The rifle itself is fairly traditional, but the scope sitting atop it is actually a small Wi-Fi enabled computer that can make an amateur marksman hit a bullseye every time. The technology in the rifle allows the shooter to automatically account for wind, temperature and the bullet's weight. It will even delay firing after the trigger is pulled until the barrel is lined up for a perfect shot, Greenberg writes.

By hacking into the gun's computer through the Wi-Fi, Sandvik and Auger can remotely alter all of these variables without the user's knowledge, change the gun's actual target, or delete the onboard files and render the aiming system useless. It could even be possible to infect the smart rifle with malware to alter the gun's aiming mechanism long after the hacker has gone.

Smart guns and rifles have been a controversial topic in recent years for gun owners and safety advocates alike. Back in 2013, California both approved the sale of the first smart gun in the U.S. and passed a law requiring all new or imported guns be smart guns. New Jersey passed a law in 2002 that declared within 30 months of the first "personalized" handgun sold in the state, all guns sold must be smart guns – which has effectively kept them off the shelves. Meanwhile, the Defense Advanced Research Projects Agency (DARPA) is continuing to develop self-steering bullets for the U.S. military.

TrackingPoint founder John McHale says that the company will work with Sandvik and Auger to develop a patch for the rifle's software, Greenberg writes. But while he's appreciative of their research, he says the ultimate responsibility for a gun's safety comes down to the shooter, not the software.

http://www.eurekalert.org/pub_releases/2015-08/osu-ndb073115.php

New design brings world's first solar battery to performance milestone

Sunlight makes the new 'aqueous solar flow' battery 20 percent more efficient than today's lithium-iodine batteries

Pam Frost Gorder

COLUMBUS, Ohio--After debuting the world's first solar air battery last fall, researchers at The Ohio State University have now reached a new milestone.

In the Journal of the American Chemical Society, they report that their patent-pending design--which combines a solar cell and a battery into a single device--now achieves a 20 percent energy savings over traditional lithium-iodine batteries. The 20 percent comes from sunlight, which is captured by a unique solar panel on top of the battery, explained Yiying Wu, professor of chemistry and biochemistry at Ohio State.

The solar panel is now a solid sheet, rather than a mesh as in the previous design. Another key difference comes from the use of a water-based electrolyte inside the battery. Because water circulates inside it, the new design belongs to an emerging class of batteries called aqueous flow batteries.

"The truly important innovation here is that we've successfully demonstrated aqueous flow inside our solar battery," Wu said.

As such, it is the first aqueous flow battery with solar capability. Or, as Wu and his team have dubbed it, the first "aqueous solar flow battery." "It's also totally compatible with current battery technology, very easy to integrate with existing technology, environmentally friendly and easy to maintain," he added.

Researchers around the world are working to develop aqueous flow batteries because they could theoretically provide affordable power grid-level energy storage someday. The solar flow battery could thus bridge a gap between today's energy grid and sources of renewable energy.

"This solar flow battery design can potentially be applied for grid-scale solar energy conversion and storage, as well as producing 'electrolyte fuels' that might be used to power future electric vehicles," said Mingzhe Yu, lead author of the paper and a doctoral student at Ohio State.

Previously, Yu designed the solar panel out of titanium mesh, so that air could pass through to the battery. But the new aqueous flow battery doesn't need air to function, so the solar panel is now a solid sheet.

The solar panel is called a dye-sensitized solar cell, because the researchers use a red dye to tune the wavelength of light it captures and converts to electrons. Those electrons then supplement the voltage stored in the lithium-anode portion of the solar battery.

Something has to carry electrons from the solar cell into the battery, however, and that's where the electrolyte comes in. A liquid electrolyte is typically part salt, part solvent; previously, the researchers used the salt lithium perchlorate mixed with the organic solvent dimethyl sulfoxide. Now they are using lithium iodide as the salt, and water as the solvent. (Water is an inorganic solvent, and an eco-friendly one. And lithium iodide offers a high-energy storage capacity with low cost.)

In tests, the researchers compared the solar flow battery's performance to that of a typical lithium-iodine battery. They charged and discharged the batteries 25 times. Each time, both batteries discharged around 3.3 volts.

The difference was that the solar flow battery could produce the same output with less charging. The typical battery had to be charged to 3.6 volts to discharge 3.3

volts. The solar flow battery was charged to only 2.9 volts, because the solar panel made up the difference. That's an energy savings of nearly 20 percent.

The project is still ongoing, and the solar flow design will undoubtedly evolve again as the researchers try to make the battery more efficient.

Doctoral student and study co-author Billy McCulloch said that there are many different directions the research could take. "We hope to motivate the research community to further develop this technology into a practical renewable energy solution," he added.

The team's ultimate goal is to boost the solar cell's contribution to the battery past its current 20 percent--maybe even to 100 percent. "That's our next step," Wu said, "to really achieve a fully solar-chargeable battery."

Other coauthors on the paper included doctoral students Damian R. Beauchamp, Zhongjie Huang and Xiaodi Ren.This research was funded by the Department of Energy.

http://www.eurekalert.org/pub_releases/2015-08/cru-ccc073115.php

Childhood cancer cells drain immune system's batteries

Neuroblastoma cells produce a molecule that breaks down arginine, a building block of proteins and an essential energy source for immune cells

Cancer cells in neuroblastoma contain a molecule that breaks down a key energy source for the body's immune cells, leaving them too physically drained to fight the disease, according to new research published in the journal Cancer Research today (Saturday).

Cancer Research UK-funded scientists have discovered that the cells in neuroblastoma - a rare type of childhood cancer that affects nerve cells - produce a molecule that breaks down arginine, one of the building blocks of proteins and an essential energy source for immune cells.

This molecule - called 'arginase' - creates a huge dip in the level of arginine found in the area around the tumour. As soon as the body's immune cells get close to the cancer, the sudden lack of their favourite energy source makes them lethargic and ineffective.

Neuroblastoma cells have a molecule on their surface that marks them out as different from healthy cells. This had led to hopes that the immune system might be trained to recognise and destroy them. But this new research may explain why early attempts to harness the immune system in this way have so far been unsuccessful.

Dr Francis Mussai, study author at the University of Birmingham, said: "We've known for a while that harnessing the power of the immune system could be an effective way to treat neuroblastoma. But we didn't know why the immune cells were having such difficulty recognising and destroying the tumour.

"Armed with this new knowledge about the role of arginine, we may be able to activate the immune system to attack cancer cells."

Dr Carmela De Santo, co-study author at the University of Birmingham, said: "Now the challenge is to develop new drugs which stop neuroblastoma from using arginine, and may make immune therapy more effective."

Around 90 cases of neuroblastoma are diagnosed each year in the UK, mostly in children under five years old.

Eleanor Barrie, senior science information manager at Cancer Research UK, said: "These findings could have huge implications for treating neuroblastoma. Better understanding the role of arginine could help us to boost the body's immune cells and we hope this could lead to more effective treatments.

"We recently launched Cancer Research UK Kids and Teens as part of our commitment to bringing forward the day when no young lives are lost to cancer. Our target is to find more cures and kinder treatments for children with the disease so that, in the future, every child with cancer can go on to live a long and healthy life."

Mussai, F., et al, Neuroblastoma arginase activity creates an immunosuppressive microenvironment that impairs autologous and engineered immunity. Cancer Research, 2015. DOI: 10.1158/0008-5472.CAN-14-3443

http://www.eurekalert.org/pub_releases/2015-08/uoc--cmf073015.php

Common medications for dementia could cause harmful weight loss

UCSF researchers recommend clinicians account for risk when prescribing to older adults

Medications commonly used to treat dementia could result in harmful weight loss, according to UC San Francisco researchers, and clinicians need to account for this risk when prescribing these drugs to older adults, they said.

Their study appears online and in the August issue of the Journal of the American Geriatrics Society.

"This is very relevant to patient care because unintentional weight loss in older adults is associated with many adverse outcomes, including increased rates of institutionalization and mortality, a decline in functional status, and poorer quality of life," said lead author Meera Sheffrin, MD, geriatrics fellow in the UCSF School of Medicine at the UCSF-affiliated San Francisco VA Medical Center. "Our study provides evidence in a large, real-world population that cholinesterase inhibitors may contribute to clinically significant weight loss in a substantial proportion of older adults with dementia."

Alzheimer's disease and other dementias are prevalent, affecting one in six people over age 80. The main drug treatments, a class of medications called cholinesterase inhibitors (i.e., donepezil, galantamine, rivastigmine), are marginally beneficial for most patients and may have serious side effects such as gastrointestinal symptoms.

Weight loss also is a significant problem in dementia patients and linked to increased mortality. Data from randomized controlled trials suggests this weight loss may be an under-recognized side effect of cholinesterase inhibitors, but evidence is limited and conflicting.

In this study, Sheffrin and her colleagues used national VA data from 2007-2010 to evaluate patients age 65 or older diagnosed with dementia who received a new prescription for a cholinesterase inhibitor or other new chronic medication. The primary outcome was timed to a 10-pound weight loss over a 12-month period, as this represents a degree of loss that would be noticed by a clinician and perhaps prompt further action in considering the causes and potential treatments.

A total of 1,188 patients started on cholinesterase inhibitors were matched to 2,189 patients started on other medications. At 12 months, 78 percent were still on the inhibitors, compared to 66 percent for other medications. About 29.3 percent of patients on the inhibitors experienced significant weight loss, compared to 22.8 percent of non-users.

These results demonstrated that patients started on the medications had a higher risk of clinically significant weight loss over a 12-month period compared to matched controls, Sheffrin said. Specifically, one out of every 21 patients treated experienced at least a 10-pound weight loss.

Further research is needed to validate these findings and address study limitations, including if there is a specific subgroup in which starting cholinesterase inhibitors had a higher risk of weight loss, as this study may have been underpowered to find those differences. The sample also included mainly older male veterans, so the generalizability of the findings to women is uncertain, the researchers said.

"Clinicians should take into account the risk of weight loss when weighing the risks and benefits of prescribing cholinesterase inhibitors in patients with dementia," the authors write. "In addition, clinicians should monitor for weight loss if these medications are prescribed and consider discontinuing cholinesterase inhibitors if significant weight loss occurs."

Other UCSF contributors to the Journal of the American Geriatrics Society study were senior author Mike Steinman, MD, associate professor, and Yinghui Miao, MD, MPH, statistician, of geriatrics; and W. John Boscardin, PhD, professor of epidemiology and biostatistics. Funding was provided by the National Institute on Aging and the American Federation on Aging Research.

http://www.eurekalert.org/pub_releases/2015-08/muhc-css073115.php

Canadian study sheds surprising light on the causes of cerebral palsy

Wider use of genetic testing in children with CP should be considered

TORONTO/MONTREAL - Cerebral palsy (CP) is the most common cause of physical disability in children. It has historically been considered to be caused by factors such as birth asphyxia, stroke and infections in the developing brain of babies. In a new game-changing Canadian study, a research team from The Hospital for Sick Children (SickKids) and the Research Institute of the McGill University Health Centre (RI-MUHC) has uncovered strong evidence for genetic causes of cerebral palsy that turns experts' understanding of the condition on its head.

The study, published online August 3 in Nature Communications could have major implications on the future of counselling, prevention and treatment of children with cerebral palsy.

"Our research suggests that there is a much stronger genetic component to cerebral palsy than previously suspected," says the lead study author Dr. Maryam Oskoui, Paediatric neurologist at The Montreal Children's Hospital (MCH) of the MUHC, co-director of the Canadian Cerebral Palsy Registry and an Assistant Professor in the Department of Paediatrics and Department of Neurology and Neurosurgery at McGill University. "How these genetic factors interplay with other established risk factors remains to be fully understood. For example, two newborns exposed to the same environmental stressors will often have very different outcomes. Our research suggests that our genes impart resilience, or conversely a susceptibility to injury."

Children with cerebral palsy have difficulties in their motor development early on, and often have epilepsy and learning, speech, hearing and visual impairments. Two out of every thousand births are affected by cerebral palsy with a very diverse profile; some children are mildly affected while others are unable to walk on their own or communicate. Genetic testing is not routinely done or recommended, and genetic causes are searched for only in rare occasions when other causes cannot be found.

The research team performed genetic testing on 115 children with cerebral palsy and their parents from the Canadian Cerebral Palsy Registry, many of which had other identified risk factors. They found that 10 per cent of these children have copy number variations (CNVs) affecting genes deemed clinically relevant. In the general population such CNVs are found in less than one per cent of people. CNVs are structural alterations to the DNA of a genome that can be present as deletions, additions, or as reorganized parts of the gene that can result in disease.

"When I showed the results to our clinical geneticists, initially they were floored," says Dr. Stephen Scherer, Principal Investigator of the study and Director of The Centre for Applied Genomics (TCAG) at SickKids. "In light of the findings, we suggest that genomic analyses be integrated into the standard of practice for diagnostic assessment of cerebral palsy."

The study also demonstrates that there are many different genes involved in cerebral palsy. "It's a lot like autism, in that many different CNVs affecting different genes are involved which could possibly explain why the clinical presentations of both these conditions are so diverse," says Scherer, who is also Director of the University of Toronto McLaughlin Centre. "Interestingly, the frequency of de novo, or new, CNVs identified in these patients with cerebral palsy is even more significant than some of the major CNV autism research from the last 10 years. We've opened many doors for new research into cerebral palsy."

"Finding an underlying cause for a child's disability is an important undertaking in management," says Dr. Michael Shevell, co-director of the Canadian Cerebral Palsy Registry and Chair of the Department of Paediatrics at the MCH-MUHC. "Parents want to know why their child has particular challenges. Finding a precise reason opens up multiple vistas related to understanding, specific treatment, prevention and rehabilitation. This study will provide the impetus to make genetic testing a standard part of the comprehensive assessment of the child with cerebral palsy."

This study was supported by NeuroDevNet Networks Centre of Excellence, the Canadian Institutes of Health Research (CIHR), Genome Canada, the University of Toronto McLaughlin Centre, and SickKids Foundation. The Canadian Cerebral Palsy Registry has been funded by the Réseau de recherche sur le développement, la santé et le bien-être de l'enfant (RSDE) des Fonds de Recherche en Santé du Québec (FRSQ) and NeuroDevNet. Dr. Maryam Oskoui is a FRQS Chercheur-Boursier Clinicien Junior 1.

http://www.bbc.com/news/health-33757929

Everyone 'should take vitamin D pills'

Everyone should consider taking vitamin D supplements to counter the lack of sunshine in the UK, government experts are proposing.

The draft Scientific Advisory Committee on Nutrition guidelines suggest, from the age of one, 10 microgram pills be taken to ensure people get enough. The plans are now being consulted on until 23 September.

Current advice is only at risk groups - including pregnant women, under fives and over 65s - should take supplements. But as there is no easy way of assessing who is getting enough vitamin D, SACN has proposed a blanket recommendation for everyone because of the benefits it would bring.

The risk of getting too much vitamin D is considered to be extremely low.

Winter months

It comes after the National Institute for Health and Care Excellence (NICE), which advises the NHS on treatments, has already suggested vitamin D should be given more widely to counter a hidden epidemics of deficiency. Official estimates suggest one in five adults and one in six children in England may have low levels.

People get most of their vitamin D from the action of sunlight on their skin. But the amount in food is small, unlike many other vitamins.

The low level of sunlight during winter months means people in the UK are at risk. The NICE guidelines called for more free supplements and for supermarkets to sell low-cost tablets. Deficiency can result in rickets and brittle bones.

http://www.eurekalert.org/pub_releases/2015-08/acoe-bt3080215.php

Brain teaser: 3-D printed 'tissue' to help combat disease

The difficult challenge of creating bench-top brain tissue to learn how the brain functions

The brain is amazingly complex, with around 86 billion nerve cells. The challenge for researchers to create bench-top brain tissue from which they can learn about how the brain functions, is an extremely difficult one. Researchers at the ARC Centre of Excellence for Electromaterials Science (ACES) have taken a step closer to meeting this challenge, by developing a 3D printed layered structure incorporating neural cells, that mimics the structure of brain tissue.

The value of bench-top brain tissue is huge. Pharmaceutical companies spend millions of dollars testing therapeutic drugs on animals, only to discover in human trials that the drug has an altogether different level of effectiveness. We're not sure why, but the human brain differs distinctly from that of an animal.

A bench-top brain that accurately reflects actual brain tissue would be significant for researching not only the effect of drugs, but brain disorders like schizophrenia, and degenerative brain disease.

ACES Director and research author Professor Gordon Wallace said that the breakthrough is significant progress in the quest to create a bench-top brain that will enable important insights into brain function, in addition to providing an experimental test bed for new drugs and electroceuticals.

"We are still a long way from printing a brain but the ability to arrange cells so as they form neuronal networks is a significant step forward," Professor Wallace said.

To create their six-layered structure, researchers developed a custom bio-ink containing naturally occurring carbohydrate materials. The custom materials have properties that allow accurate cell dispersion throughout the structure, whilst providing a rare level of protection to the cells.

The bio-ink is then optimised for 3D-printing, and developed for use in a standard cell culturing facility without the need for expensive bio-printing equipment.

The result is a layered structure like brain tissue, in which cells are accurately placed and remain in their designated layer. "This study highlights the importance of integrating advances in 3D printing, with those in materials science, to realise a biological outcome," Professor Wallace said. "This paves the way for the use of more sophisticated printers to create structures with much finer resolution."

The research, funded through Professor Gordon Wallace's Australian Laureate Fellowship, is published in Biomaterials journal.

http://www.eurekalert.org/pub_releases/2015-08/afot-tau073015.php

Tel Aviv University researcher discovers trigger of deadly melanoma

Study pinpoints cause of melanoma transformation within the epidermis

Skin cancer is the most common of all cancers, and melanoma, which accounts for 2% of skin cancer cases, is responsible for nearly all skin cancer deaths. Melanoma rates in the US have been rising rapidly over the last 30 years, and although scientists have managed to identify key risk factors, melanoma's modus operandi has eluded the world of medical research.

A new Tel Aviv University study published in Molecular Cell sheds light on the precise trigger that causes melanoma cancer cells to transform from non-invasive cells to invasive killer agents, pinpointing the precise place in the process where "traveling" cancer turns lethal.

The research was led by Dr. Carmit Levy of the Department of Human Genetics and Biochemistry at TAU's Sackler School of Medicine and conducted by a team of researchers from TAU, the Technion Institute of Technology, the Sheba Medical Center, the Institut Gustave Roussy and The Hebrew University of Jerusalem.

If melanoma is caught in time, it can be removed and the patient's life saved. But once melanoma invades the bloodstream, turning metastatic, an aggressive treatment must be applied. When and how the transformation into aggressive invasion took place was until now a mystery.

Understanding the skin

"To understand melanoma, I had to obtain a deep understanding about the structure and function of normal skin," said Dr. Levy, "Melanoma is a cancer that originates in the epidermis, and in its aggressive form it will invade the dermis, a lower layer, where it eventually invades the bloodstream or lymph vessels, causing metastasis in other organs of the body. But before invading the dermis, melanoma cells surprisingly extend upward, then switch directions to invade.

"It occurred to me that there had to be a trigger in the microenvironment of the skin that made the melanoma cells 'invasive,'" Dr. Levy continued. "Using the evolutionary logic of the tumor, why spend the energy going up when you can just use your energy to go down and become malignant?"

After collecting samples of normal skin cells and melanoma cells from patients at hospitals around Israel, the researchers mixed normal and cancerous cells and performed gene analysis expression to study the traveling cancer's behavior. They found that, completely independently of any mutation acquisition, the microenvironment alone drove melanoma metastasis.

"Normal skin cells are not supposed to 'travel,'" said Dr. Levy. "We found that when melanoma is situated at the top layer, a trigger sends it down to the dermis and then further down to invade blood vessels. If we could stop it at the top layer, block it from invading the bloodstream, we could stop the progression of the cancer."

A new way of saving lives

The researchers found that the direct contact of melanoma cells with the remote epidermal layer triggered an invasion via the activation of "Notch signaling," which turns on a set of genes that promotes changes in melanoma cells, rendering them invasive.

According to the study, when a molecule expressed on a cell membrane -- a spike on the surface of a cell, called a ligand -- comes into contact with a melanoma cell, it triggers the transformation of melanoma into an invasive, lethal agent.

"When I saw the results, I jumped out of the room and shouted, 'We got it!'" Dr. Levy said. "Now that we know the triggers of melanoma transformation and the kind of signalling that leads to that transformation, we know what to block. The trick was to solve the mystery, and we did. There are many drugs in existence that can block the Notch signalling responsible for that transformation. Maybe, in the future, people will be able to rub some substance on their skin as a prevention measure."

Dr. Levy is continuing to explore the research with the end goal of providing medical professionals with another tool of analysis of different stages of melanoma. "Melanoma is a cancer with a very long gestation period," said Dr. Levy. "If you can provide a simple kit with precise answers, you can catch it at the beginning stage and hopefully save lives."

http://www.eurekalert.org/pub_releases/2015-08/hcfa-chq080315.php

Cassiopeia's hidden gem: The closest rocky, transiting planet

Only 21 light-years away is the nearest transiting rocky planet

Skygazers at northern latitudes are familiar with the W-shaped star pattern of Cassiopeia the Queen. This circumpolar constellation is visible year-round near the North Star. Tucked next to one leg of the W lies a modest 5th-magnitude star named HD 219134 that has been hiding a secret.

Astronomers have now teased out that secret: a planet in a 3-day orbit that transits, or crosses in front of its star. At a distance of just 21 light-years, it is by far the closest transiting planet to Earth, which makes it ideal for follow-up studies. Moreover, it is the nearest rocky planet confirmed outside our solar system. Its host star is visible to the unaided eye from dark skies, meaning anyone with a good star map can see this record-breaking system.

"Most of the known planets are hundreds of light-years away. This one is practically a next-door neighbor," said astronomer Lars A. Buchhave of the Harvard-Smithsonian Center for Astrophysics (CfA).

"Its proximity makes HD 219134 ideal for future studies. The James Webb Space Telescope and future large ground-based observatories are sure to point at it and examine it in detail," said lead author Ati Motalebi of the Geneva Observatory.

The newfound world, designated HD 219134b, was discovered using the HARPS-North instrument on the 3.6-meter Telescopio Nazionale Galileo in the Canary Islands. The CfA is a major partner with the Geneva Observatory on the HARPS-North Collaboration, which includes several other European partners.

HARPS-North detects planets using the radial velocity method, which allows astronomers to measure a planet's mass. HD 219134b weighs 4.5 times the mass of Earth, making it a super-Earth.

With such a close orbit, researchers realized that there was good possibility the planet would transit its star. In April of this year they targeted the system with NASA's Spitzer Space Telescope. At the appropriate time, the star dimmed slightly as the planet crossed the star's face. Measuring the depth of the transit gave the planet's size, which is 1.6 times Earth. As a result, the team can calculate the planet's density, which works out to about 6 g/cm3. This shows that HD 219134b is a rocky world.

But wait, there's more! The team detected three additional planets in the system using radial velocity data. A planet weighing at least 2.7 times Earth orbits the star once every 6.8 days. A Neptune-like planet with 9 times the mass of Earth circles in a 47-day orbit. And much further out, a hefty fourth world 62 times Earth's mass orbits at a distance of 2.1 astronomical units (200 million miles) with a "year" of 1,190 days. Any of these planets might also transit the star, so the team plans to search for additional transits in the months ahead.

HD 219134 is an orange Type K star somewhat cooler, smaller and less massive than our Sun. Its key measurements have been pinned down very precisely, which thus allows a more precise determination of the properties of its accompanying planets.

This discovery came from the HARPS-North Rocky Planet Search, a dedicated survey examining about 50 nearby stars for signs of small planets. The team targeted nearby stars because those stars are brighter, which makes follow-up studies easier. In particular, additional observations might allow the detection and analysis of planetary atmospheres.

HD 219134 was one of the closest stars in the sample, so it was particularly lucky to find that it hosts a transiting planet. This system now holds the record for the nearest transiting exoplanet. As such, it likely will be a favorite for researchers for years to come.

http://bit.ly/1IUONtG

The Science Behind Dogs' Goofy Greetings

Why do dogs go nuts when their owners get home? The answers lie in their DNA and brains

By Helen Thompson

After returning home from a trip, work or even a short walk to take out the garbage, dog owners are routinely greeted with copious amounts of drool and tail wagging. But why? As George Dvorsky explains over at *io9*, the answer is in dogs' brains — and even their DNA.

Some of dogs' enthusiasm comes down to their wolf ancestry. Wolves often greet each other with face licking—a way of affirming social bonds and checking out what your buddy caught on a hunt. That said, wolves are more skeptical of new things, so dog greetings are much more exaggerated. Some argue that the most social wolves would have been the ones domesticated by humans 10,000 to 15,000 years ago, explains Dvorsky.

There's another component to goofy dog greetings: the brain. Dogs can distinguish human smells from those of canines and recognize familiar odors, writes Dvorsky in a deep dive that's well worth checking out. Brain imaging studies also suggest that the sight of an owner switches on pathways in the brain associated with reward. (The same thing happens when humans see friends.)

All of those explanations could certainly be behind the plethora of YouTube videos of dogs going nuts at the sight of owners returning home:

There's also some recent evidence that dogs and humans share a unique bond. When they gaze into each other's eyes, their brains secrete the hormone oxytocin. It's linked to social bonding in several species, but most notably between human mothers and babies. Even when raised by humans, wolves do not experience the same oxytocin rush.

Obviously, all dogs are different, and greetings definitely vary. Dogs who aren't used to being separated from their owner may be more enthusiastic when that long-lost owner returns (even if it's only been a few minutes). Either way, it's clear that dogs can get as much enjoyment out of seeing their human as their human gets out of seeing them.