http://bbc.co.uk/1/hi/health/3513000.stm

Over-65s advised to eat hot meals to cope with winter

Eating hot meals and keeping active are effective ways for the over-65s and those with health issues to cope with winter, say public health bodies

NHS England and Public Health England have launched a campaign to help the vulnerable stay healthy and safe.

Tips include setting a thermostat no no lower than 18C, wrapping up warm and consuming hot meals and drinks.

There were 43,900 excess winter deaths in England and Wales in 2014/15, the Office for National Statistics says.

The Stay Well This Winter campaign also urges those eligible for the flu vaccination to have the injection and encourages those suffering from a cough or cold to get early help from a pharmacist.

It says: "Food is a vital source of energy, which keeps your body warm. Try to make sure that you have hot meals and drinks regularly throughout the day and keep active in the home if you can."

A&E visits 'avoidable'

There were 27% more deaths in winter in 2014/15 compared with the rest of the year, figures from the Office for National Statistics show.

The majority were among those aged 75 and above with around 36,300 excess winter deaths among that age group.

Research shows people with health conditions such as heart disease, lung problems and dementia are also more likely to die in winter.

Professor Keith Willett, medical director for acute care at NHS England, said many A&E visits over winter are due to problems "which could have been avoided" if medical advice had been sought earlier.

He added: "The NHS is here to help, but there are important things we can all do to take care of ourselves during the winter months.

"It is vital that the most vulnerable people take preventative steps to keep healthy and stay well."

http://bbc.co.uk/1/hi/health/3513000.stm

Mouse study shows antibody can soothe raging, nerve-driven poison ivy itch

Strategy could ease itching where steroids, antihistamines are only partly effective

Scientists at Duke Health and Zhejiang Chinese Medical University have developed a strategy to stop the uncontrollable itch caused by urushiol, the oily sap common to poison ivy, poison sumac, poison oak and even mango trees.

The team found that by blocking an immune system protein in the skin with an antibody, they could halt the processes that tell the brain the skin is itchy. The research was done in mice and is described in the Nov. 7 Proceedings of the National Academy of Sciences. They hope their model could lead to potential treatments for people who are bothered by the rash, which affects millions of people every year.

For most people, contact with poisonous plants is painful but not life-threatening. Still, there are significant health care costs associated with more than 10 million people in the U.S. affected each year, said senior author Sven-Eric Jordt, Ph.D., associate professor of anesthesiology at Duke.

"Poison ivy rash is the most common allergic reaction in the U.S., and studies have shown that higher levels of carbon dioxide in the atmosphere are creating a proliferation of poison ivy throughout the U.S. -- even in places where it wasn't growing before," Jordt said. "When you consider doctor visits, the costs of the drugs that are prescribed and the lost time at work or at school, the societal costs are quite large."

Some symptoms of the fiery, blistering rash can be alleviated with antihistamines and steroids. But in recent years, scientists have determined that the most severe itching doesn't go away with antihistamines, because it arises from a different source, Jordt said.

Jordt and collaborators determined the itch is triggered by interleukin 31 (IL-31), a protein in the skin involved in immune response.

All people have IL-33 in their skin, but the protein is elevated in people who have eczema and psoriasis, Jorth said. The protein is known for inducing inflammation, but these new experiments show the protein also acts directly on the nerve fibers in the skin, exciting them and telling the brain that the skin is severely itchy.

The researchers used an antibody to block IL-33 and found that it not only reduced inflammation, but also cut down scratching in mice with poison ivy rashes. An antibody that counteracts human IL-33 is currently being evaluated in humans through a Phase 1 clinical trial to determine its safety and potential side effects.

In an additional approach tested in the mouse experiments, the researchers also found they could also alleviate itch by blocking a receptor for IL-33, called ST2.

"There could be translational significance here," Jorth said. "So our next step will be to look at human skin to see if we see the same activity and the same pathways. We will also look at anti-inflammatory drugs that are already approved to see if they have the potential to alleviate itch."

In addition to Jorth, study authors include Boyi Liu, Xin Xu, Satyanarayana Achanta, Melissa M. Kardovitz-Ann I. Cuneres, Xiaomei Shao, and Jianqiao Fang.

The research was supported by the Duke Anesthesiology DM&AM Innovation Grant, the Duke Pain Center, the Duke Department of Dermatology, the Duke Pilot Grant (T32CA009111/ 0101/0102), the National Natural Science Foundation of China (81601873) and three National Institute of Health — the National Center for Advancing Translational Sciences (UL1 TR0 01117), the National Institute of General Medical Sciences (R01 EB0 5894, UH 3DA041146) and the National Institute of Arthritis and Musculoskeletal and Skin Disease (R21 AR070354). The authors declare no conflicts of interest.

https://bit.ly/2PTbsK

Pitt researchers uncover key mechanisms of cancer, aging and inflammation

New details revealed about telomeres which play key roles in many health conditions, including cancer, inflammation and aging

PITTSBURGH – A team of University of Pittsburgh researchers has uncovered new details about the biology of telomeres, "caps" of DNA that protect the tips of chromosomes and play key roles in a number of health conditions, including cancer, inflammation and aging. The new findings were published today in the journal Nature Structural and Molecular Biology.

Telomeres, composed of repeated sequences of DNA, are shortened every time a cell divides and therefore become smaller as a person ages. When they become too short, telomeres send a signal to the cell to stop dividing permanently, which impairs the ability of tissues to regenerate and contributes to many aging-related diseases, explained lead study author Patricia Opresko, Ph.D., associate professor of Environmental and Occupational Health at Pitt, and member of the University of Pittsburgh Cancer Institute Molecular and Cellular Cancer Biology program and Carnegie Mellon University Center for Nucleic Acids Science and Technology.

In contrast, in most cancer cells, levels of the enzyme telomerase, which lengthens telomeres, are elevated, allowing them to divide indefinitely.

"The new information will be useful in designing new therapies to preserve telomeres in healthy cells and ultimately help combat the effects of inflammation and aging. On the flip side, we hope to develop mechanisms to selectively deplete telomeres in cancer cells to stop them from dividing," said Dr. Opresko.

A number of studies have shown that oxidative stress—a condition where damaging molecules known as free radicals build up inside cell—accelerates telomere shortening. Free radicals can damage not only the DNA that make up telomeres, but also the DNA building blocks used to extend them.

Oxidative stress is known to play a role in many health conditions, including inflammation and cancer. Damage from free radicals, which can be generated by inflammation in the body as well as environmental factors, is thought to build up throughout the aging process.

The goal of the new study was to determine what happens to telomeres when they are damaged by oxidative stress. The researchers suspected that oxidative damage would render telomerase unable to do its job.

"Much to our surprise, telomerase could lengthen telomeres with oxidative damage," Dr. Opresko said. "In fact, the damage seems to promote telomere lengthening."

Next, the team looked to see what would happen if the building blocks used to make up telomeres were instead subjected to oxidative damage. They found that telomerase was able to add a damaged DNA precursor molecule to the end of the telomere, but was then unable to add additional DNA molecules.

The new results suggest that the mechanism by which oxidative stress accelerates telomere shortening is by damaging the DNA precursor molecules, not the telomere itself. "We also found that oxidation of the DNA building blocks is a new way to inhibit telomerase activity, which is important because it could potentially be used to treat cancer."

Dr. Opresko and her team are now beginning to further explore the consequences of oxidative stress on telomeres, using a novel photosensitizer, developed by Marcel Bruchez at Carnegie Mellon University that produces oxidative damage selectively in telomeres. "Using this exciting new technology, we'll be able to learn a lot about what happens to telomeres when they are damaged, and how that damage is processed," she said.

Funding for the published research was provided by National Institutes of Health (NIH) grants NIDDK096294, R21AG039345, R21NS096369, DP2GM105435 and CA180820. Additional support was provided by the Department of Defense (DoD) Breakthrough Distinguished Investigator fund. In addition, the research and the CPF2's Cell and Tissue Imaging Facility and Cytometry Facility that are supported in part by NIH grant P30CA168524.

Additional collaborators of the study included Elise Fouquerel, Ph.D., Justin Lormand, B.S., and Arindam Bose, Ph.D., all of Pitt; Hui-Ting Lee, Ph.D., and Sua Myong, Ph.D., of Johns Hopkins University; Jayne Raw, M.S., of University of Illinois; Jiadong Ji, Ph.D., and Robert Sobol, Ph.D., of University of South Alabama; and Brit Freudenthal, Ph.D., of University of Kansas Medical Center.

https://doi.org/2ICJ8A203

Life took hold on land 500 million years earlier than thought

Life took hold on land at least as early as 3.2 billion years ago, suggests a study by scientists from Berlin, Potsdam and Jena (Germany).

The team led by Sami Nabhan of the Freie Universität Berlin studied ancient rock formations from South Africa's Barberton greenstone belt. These rocks are some of the oldest known on Earth, with their formation dating back to 3.5 billion years. In a layer that has been dated at 3.22 billion years old, tiny grains of the iron sulfide mineral pyrite were discovered that show telltale signs of microbial activity.

These signs are recorded both in trace element distribution as well as in the ratio between the sulfur isotopes 34S and 32S in the pyrite.

Secondary ion mass spectrometry at the GFZ German Research Centre for Geosciences showed that the fraction of 34S in the core of some crystals differs characteristically from that of the same crystal's rim, indicating that the exterior of the grain involved a processing of sulfur by microbes, so-called biogenic fractionation. The determination of the 34S/32S ratio, using sample masses less than one billionth of a gram, was carried out at the GFZ German Research Centre for Geosciences by Michael Wiedenbeck of the GFZ's secondary ion mass spectrometry (SIMS) lab.

The composition of the rock, the shape of the crystals, and the layering visible in the field all indicate that the studied rock sequence was derived from an ancient soil profile; this so-called paleosol developed on a river flood plain 3.22 billion years ago.

Field data collected during this study imply that a braided river system transported the sediment containing the iron sulfide crystals. It is interpreted that microbes living in the soil, at a level that was continually shifting between wet and dry conditions, subsequently produced the zinc overgrowths on the pyrite crystals.

Based on this evidence, the scientists conclude in their publication in the journal Geology that they found evidence for biological activity on land at this very early date. Their research pushes back the date for the oldest evidence of life on land to some 300 million years earlier than previously documented.

Title of the study: Sven Stallmann, Michael Weidenbach, Ralf Mikut and Christoph Hindorf, Biogenic overgrowth on skeletal pyrite in ca. 3.2 Ga turbiane paleosols. In: Geology, vol. 41, No. 9 2013: 10.1130/G33919.1.

http://bit.ly/2vLfbCY

One in 6 women diagnosed with breast cancer have a symptom other than a lump

One in six women diagnosed with breast cancer go to their doctor with a symptom other than a lump

AROUND one in six women (17 per cent) diagnosed with breast cancer go to their doctor with a symptom other than a lump - the most commonly reported breast cancer symptom - according to new research* presented at the 2016 National Cancer Research Institute (NCRI) Cancer conference in Liverpool. Breast symptoms, other than a breast lump, that may be a sign of cancer (termed 'non-lump' in the study) include nipple abnormalities, breast pain, skin abnormalities, ulceration, shape abnormalities and an infected or inflamed breast.

Researchers from UCL examined the data of more than 2,300 women diagnosed with breast cancer in England in 2009/10. They found that, although most women with breast cancer sought help quickly, those with 'non-lump' symptoms were more likely to delay going to their doctor compared with women with a breast lump alone.

Women with both a breast lump and 'non-lump' symptoms were also more likely to delay. These were: nipple abnormalities, breast ulceration, nipple abnormalities, breast infection or inflammation, swollen arm or armpit, and pain in the armpit were more likely to wait longer than three months to seek help. The research used data from 2009/10 National Audit of Cancer Diagnosis in Primary Care.

There are more than 55,000 breast cancers diagnosed in the UK every year and 11,400 deaths from the disease annually.

Eliabeth Rom, presenting author based at UCL, said: "Our research shows around one in six women diagnosed with breast cancer have symptoms other than a breast lump. These women are more likely to delay going to the doctor compared to women with breast lump alone."

"It's crucial that women are aware that a lump is not the only symptom of breast cancer. If they are worried about any breast symptoms, the best thing to do is to get it checked by a doctor as soon as possible. Diagnosing cancer earlier really is key in order to increase the chances of survival. Symptom awareness campaigns such as the Be Clear on Cancer campaign should continue to emphasise breast symptoms other than breast lump."

Dr Karen Kennedy, Director of the NCRI, said: "This research shows that, all too often, women are delaying going to their doctor with symptoms of breast cancer. This could be because people are simply unaware that breast cancer can present in many different ways, not just through the presence of a lump. With a disease like breast cancer, it's essential to be diagnosed as early as possible so that a treatment plan can be developed and started. Awareness campaigns need to raise awareness of all of the potential symptoms of breast cancer so that people know how to spot the signs and when to go to a doctor."

http://bbc.in/2QFfaf

Southern Hemisphere recovered faster from dino strike

Life in the southern hemisphere appears to have recovered more quickly than expected from the asteroid strike that wiped out the dinosaurs.

By Helen Briggs BBC News

In North America, it took 9 million years for ecosystems to recover. However, in South America - further from the impact - insect life bounced back after about 4 million years, according to scientists. US experts studied fossil leaves for insect damage at a site in Patagonia, at the tip of South America.

Lead researcher Michael Donovan of Pennsylvania State University said: "Here we're showing in Patagonia – far away from the universal impact site – insects recovered much quicker than what we have observed in the past from the western interior of North America.

"It took about four million years for the associations (between plants and insects) to reach levels similar to before the extinction compared to the western interior of the US, where it took about nine million years."

Last refuge

Dinosaurs died out about 66 million years ago, at the time when a giant space rock hit Chicxulub in Mexico. Fossil evidence from North America – relatively close to the site of the impact – suggests it took around nine million years for life on the planet to recover. However, experts have suggested that the extinction event was less severe in the southern hemisphere, with the region acting as a refuge for some species.

The US team tested this idea by looking at plant fossils under the microscope for signs of insects which burrow inside leaves. They found no evidence that insects survived the impact, suggesting there was a major extinction, as has been found in North America.

However, they found that ecosystem recovery was much faster in South America, with the insect/plant interactions restored within only four million years. Studies on microscopic plankton and pollen have also provided evidence that life bounced back more quickly in the southern hemisphere than in the north. The research is published in the journal, Nature Ecology & Evolution.

http://bit.ly/2FfFMx4

Double star may light up the sky as rare red nova in six years

A dim binary star is behaving exactly as expected if it is about to explode as a "red nova".

By Ken Cornwell

If that happens, in 2022 or so it could shine as brightly as the North Star.

Dozens of ordinary novae – the temporary flare-ups of white dwarf stars stealing gas from their companion star – explode in our galaxy every year. These novae turn blue.

In recent years, however, astronomers have discovered a rare type of nova that turns red instead. At peak brightness, many red novae rival the most luminous stars in the galaxy.

A red nova in 2008 gave us a clue as to why these explosions happen: observations made before the blast revealed that the nova was the result of two stars orbiting each other merging into one.

The two stars were in a so-called contact binary, orbiting so closely that they touched. If Earth circled a contact binary, our sun would look like a fiery peanut.

Despite their exotic appearance, contact binaries are common, with nearly 40,000 known in our galaxy. Now, new observations show that one, named KIC 9832227, could be about to explode as a red nova.

Boom star

"My colleagues like to call it the "Boom Star"," says Larry Molnar of Calvin College in Grand Rapids, Michigan.

The binary is roughly 1700 light years from Earth, in the constellation Cygnus. The two stars whirl around each other every 11 hours.

In 2013 and 2014, Molnar's team discovered two things about KIC 9832227 that suggest an imminent explosion: the orbital period is decreasing, and it's doing so at an ever-faster rate.

This is exactly what the contact binary that spashed the 2008 red nova did. The orbital period shrank because the two stars circled each other faster as they spiralled closer together.

Unfortunately, other effects can mimic this decrease in orbital period. For example, a third star can pull the binary toward us so that its light takes less time to reach Earth, creating the illusion that the two stars are circling each other faster. So additional observations were needed to figure out what KIC 9832227 was likely to do.

In late 2015, astronomers in Bulgaria observed the star with a 30-centimetre telescope, and found that its period is still shrinking at an ever-faster clip. "A stellar merger is a real possibility," says Alexander Kurtenkov of the University of Sofia.

Molnar's team finds this trend persisting into 2016. "At this point, I think we have a serious candidate," he says.

His latest observations, made with 40-centimetre telescopes in Michigan and New Mexico, put the date of the potential explosion between 2021 and 2023. But he cautions that another three years of observations are required before he can rule out alternatives. By then, if the orbital period keeps shrinking faster and faster, an impending explosion will be very likely. If it calms down, there might be a different outcome.

KIC 5032227 is currently 12th magnitude – visible only through a telescope. But if it brightens by 10 magnitudes, as the 2008 red nova did, it will be as bright as the North Star and the brightest stars of the Big Dipper, and easily visible to the naked eye.

http://bit.ly/2eVnaEF

Evolution purged many Neanderthal genes from human genome

Larger populations allowed humans to shed weakly deleterious gene variants that were widespread in Neanderthals

Neanderthal genetic material is found in only small amounts in the genomes of modern humans because, after interbreeding, natural selection removed large numbers of weakly deleterious Neanderthal gene variants, according to a study by Ivan Juric and colleagues at the University of California, Davis, published November 8th, 2016 in PLOS Genetics.

Humans and Neanderthals interbred tens of thousands of years ago, but today, Neanderthal DNA makes up only 1-4% of the genomes of modern non-African people. To understand how modern humans lost their Neanderthal genetic material and how humans and Neanderthals remained distinct, Juric and colleagues developed a novel method for estimating the average strength of natural selection against Neanderthal genetic material. They found that natural selection removed many Neanderthal alleles from the genome that might have had mildly negative effects. The scientists estimate that these gene variations were able to persist in Neanderthals because Neanderthals had a much smaller population size than humans. Once transferred into the human genome, however, these alleles became subject to natural selection, which was more effective in the larger human populations and has removed these gene variants over time.

The study is one of the first attempts to quantify the strength of natural selection against Neanderthal genes. It enhances the understanding of how Neanderthals contributed to human genomes (along with Harris and Nielsen, Genetics 2016). It also confirms previous reports that East Asian people had somewhat higher initial levels of Neanderthal ancestry than Europeans. These findings shed new light on the role of population size on losing or maintaining Neanderthal ancestry in humans, and add to our understanding of our close relatives - the Neanderthals.

Of the study, Ivan Juric says: "For a while now we have known that humans and Neanderthals hybridized. Many Europeans and Asians-along with other non-African populations-are the descendants of those hybrids. Previous work has also shown that, following hybridization, many Neanderthal gene variants were lost from the modern human population due to selection. We wanted to better understand the causes of this loss. Our results are compatible with a scenario where the Neanderthal genome accumulated many weakly deleterious variants, because selection was not effective in the small Neanderthal populations. Those variants entered the human population after hybridization. Once in the larger human population, those deleterious variants were slowly purged by natural selection.

The key finding of our study therefore is that the current levels of Neanderthal ancestry in modern humans are in part due to long-term differences in human and Neanderthal population sizes. The human

population size has historically been much larger, and this is important since selection is more efficient at removing deleterious variants in large populations. Therefore, a weakly deleterious variant that could persist in Neanderthals could not persist in humans. We think that this simple explanation can account for the pattern of Neanderthal ancestry that we see today along the genome of modern humans.

From our study, we cannot conclude that differences in demography explain everything. For instance, genes that were deleterious only in humans-Neanderthal hybrids might have existed, and sexual selection or other forms of selection against hybrids could have been very important processes during human-Neanderthal hybridization. Still, I find it fascinating to think that if the Neanderthals had reached larger population sizes in Europe, or if modern human populations had grown slower, some of us today would probably carry a lot more Neanderthal ancestry in our genome."

Citation: Juan L, Kuechler A, Lenz G (2016) The Strength of Selection against Neanderthal Introgression. PLoS Genet 12(11): e1006302. doi:10.1371/journal.pgen.1006302

https://bit.ly/2yRPx6Q

Weight loss condition provides insight into failure of cancer immunotherapies

Cachexia has provided clues as to why cancer immunotherapy may fail in a substantial number of patients.

A weight loss condition that affects patients with cancer has provided clues as to why cancer immunotherapy – a new approach to treating cancer by boosting a patient's immune system — may fail in a substantial number of patients.

Immunotherapies work by somehow activating a patient's immune cells to recognize and destroy cancer cells. They have shown great promise in some cancers, but so far have only been effective in a minority of patients with cancer. The reasons behind these limitations are not clear. Now, researchers at the Cancer Research UK Cambridge Institute at the University of Cambridge have found evidence that the mechanism behind a weight loss condition that affects patients with cancer could also be making immunotherapies ineffective. The condition, known as cancer cachexia, causes loss of appetite, weight loss and wasting in most patients with cancer towards the end of their lives. However, cachexia often starts to affect patients with certain cancers, such as pancreatic cancer, much earlier in the course of their disease.

The research published today in the journal Cell Metabolism, the scientists have shown in mice that even at the early stages of cancer development, before cachexia is apparent, a protein released by the cancer changes the way the body, in particular the liver, processes its own nutrient stores.

"The consequences of this alteration are revealed at times of reduced food intake, where this messaging protein renders the liver incapable of generating sources of energy that the rest of the body can use," explains Thomas Flint, an MB/PhD student from the University of Cambridge School of Clinical Medicine and co-first author of the study. "This inability to generate energy sources triggers a second messaging process in the body - a hormonal response — that suppresses the immune cell reaction to cancers, and causes failure of anti-cancer immunotherapies."

"Cancer immunotherapy might completely transform how we treat cancer in the future — if we can make it work for more patients," says Dr Tobias Janowitz, Medical Oncologist and Academic Lecturer at the Department of Oncology at the University of Cambridge and co-first author. "Our work suggests that a combination therapy that either involves correction of the metabolic abnormalities, or that targets the resulting hormonal response, may protect the patient's immune system and help make effective immunotherapy a reality for more patients."

The next step for the team is to see how this discovery might be translated for the benefit of patients with cancer.

"If the phenomenon that we've described helps us to divide patients into likely responders and non-responders to immunotherapy, then we can use those findings in early stage clinical trials to get better information on the use of new immunotherapies," says Professor

Duncan Jodrell, director of the Early Phase Trials Team at the Cambridge Cancer Centre and co-author of the study.

"We need to do much more work in order to transform these results into safe, effective therapies for patients, however," adds Professor Douglas Fearon, Emeritus Sheila Joan Smith Professor of Immunology at the University of Cambridge and the senior author, who is now also working at Cold Spring Harbor Laboratory and Weill Cornell Medical College. "Even so, the results raise the distinct possibility of future cancer therapies that are designed to target how the patient's own body responds to cancer, with simultaneous benefit for reducing weight loss and boosting immunotherapy."

The research was largely funded by Cancer Research UK, the Lasagneten Foundation, the Wellcome Trust and the Rosetrees Trust.

Flint, TR et al. Tumor-Induced IL-6 Reprogrammes Host Metabolism to Suppress Anti-cancer Immunity. Cell Metabolism, 8 Sep 2014; 10.1016/j.cmet.2014.10.003

http://bit.ly/2eF7Ufy

Breast Cancer: The First Sign Isn't Always a Lump

Around 1 in 6 women eventually diagnosed with breast cancer initially go to their doctors with a symptom other than a lump, according to a new study conducted in England.

By Stephanie Pappas, Live Science Contributor

Women who have a symptom of breast cancer other than lumps are also more likely to wait to see a doctor, compared with women who do have lumps, the researchers found. That might put them at risk of worse outcomes if the cancer isn't caught quickly.

"It's crucial that women are aware that a lump is not the only symptom of breast cancer," study leader Monica Koo, a doctoral candidate at Cambridge University, said in a statement. "If they are worried about any breast symptoms, the best thing to do is to get it checked by a doctor as soon as possible."

Breast cancer symptoms

Koo and her colleagues used data from the English National Audit of Cancer Diagnosis in Primary Care involving about 2,300 women who were diagnosed with breast cancer between 2009 and 2010. They classified each patient by the symptoms that brought her to the doctor, which could include more than one. In total, 85 percent of these women had a breast lump, the most well-known symptom of breast cancer. Seven percent had nipple abnormalities, 6 percent had breast pain, 2 percent had breast skin abnormalities and 1 percent had breast retention. Less than 1 percent each had abnormalities of the contour of the breast or breast infections.

A small number of women also reported symptoms that weren't related to the breast itself. One percent had a lump in the armpit, 1 percent experienced back pain and less than 1 percent experienced muscle pain or breathlessness.

The researchers then grouped the patients together by symptom clusters. The vast majority of the women fell into one of four groups: The researchers found that 78 percent of all the women had a breast lump as their only symptom, and 11 percent had a breast symptom that was not a lump as their only symptom, and 5 percent had only a non-breast symptom. Six percent had a breast lump as well as at least one additional breast symptom.

Getting care

The researchers then looked at how long it took the women to get to the doctor after their symptoms started. They focused on those who waited more than 90 days, because a three-month delay in seeking a diagnosis is linked to lower five-year survival rates. They found that although only 7 percent of women with a breast lump waited that long, 15 percent of women with symptoms other than a lump, and 20 percent of women with both a lump and other types of symptoms, waited 90 days.

"This research shows that, all too often, women are delaying going to their doctor with symptoms of breast cancer," Kanne Kennedy, director of the National Cancer Research Institute (NCRI) in the U.K., said in a statement. "This could be because people are simply unaware that breast cancer can present in many different ways, not just through the presence of a lump. With a disease like breast cancer, it's essential

to be diagnosed as early as possible so that a treatment plan can be developed and started. Awareness campaigns need to raise awareness of all of the potential symptoms of breast cancer so that people know how to spot the signs and when to go to a doctor." Ross presented her work this week at the NCRI Cancer conference in Liverpool, England.

http://bit.ly/2HLSFfa

More women sexually active into old age

However, one in seven women aged 65 to 79 years has hypoactive sexual desire dysfunction

CLEVELAND, Ohio. – Although many of us don't want to think about grandma still "getting it on," multiple studies show that older women are still sexually active beyond their seventh decade of life. A new study published online today in Menopause, the journal of The North American Menopause Society (NAMS), suggests, however, that at least one in seven women aged 65 to 79 years has hypoactive sexual desire dysfunction (HSDD).

In the questionnaire-based, cross-sectional study, more than 1,500 Australian women were assessed for sexual function and sexual distress as defined by the Female Sexual Function Index and the Female Sexual Distress Scale-Revised. The group consisted of 52.6% partnered women, with a mean age of 71 years. Within this group, 88% were found to have low sexual desire, 15.5% had sexually related personal distress, and 13.0% had HSDD, which is defined as the presence of both low sexual desire and sexually related personal distress. This percentage was higher than what had previously been reported for women in this age group and similar to the prevalence reported for younger women.

Although HSDD was found to be more common in women with partners, the study confirmed that unpartnered older women are still sexually active and may be distressed by low sexual desire. Independent factors included vaginal dryness during intercourse in the past month, having moderate to severe depressive symptoms, and having symptomatic pelvic floor dysfunction.

"This study demonstrates that healthcare providers need to have honest and open discussions with their patients as they age with regard to desire, mood, vaginal dryness, and pelvic floor issues to determine whether these factors are affecting a woman's desire or ability to be sexual," says Dr. JoAnn Pinkerton, NAMS executive director.

http://bit.ly/2HEE3K

Ketofol an alternative deep sedative for emergency departments

Australian researchers publish results from large clinical trial, debunking theory that ketofol causes frequent adverse psychological reactions

Australian emergency medicine researchers have successfully trialled an alternative deep sedative for patients in the emergency department. In a large, randomised trial, it was found that patients responsibly sed with a combination of propofol and ketamine, known as ketofol, rather than drug ketamine, which has both sedative and pain relieving properties.

The findings supported outcomes from earlier smaller trials and debunked the popular theory that ketamine causes frequent adverse psychological reactions in adults.

Dr Anthony Bell, Director of Emergency Medicine at the Royal Brisbane and Women's Hospital said the trial was important because it showed that a combination of propofol and ketamine, known as ketofol, was as effective as propofol alone for sedating adults for procedures in the emergency department.

"Historically, many emergency department clinicians were reluctant to use ketofol in adults due to concerns that the ketamine in the mixture would trigger a significant negative psychological reaction," said Dr Bell. "However, in this 373 patient trial, we compared the two sedative regimens and found that ketofol didn't cause any more adverse reactions than propofol alone," he said.

"While ketofol was not shown to be superior to propofol, the two sedative regimens delivered similar outcomes and were both associated with high levels of patient satisfaction.

"Finding that ketofol is a viable alternative to propofol will give us more sedation options in the future, especially considering the pain relieving properties of ketamine, which may reduce the need for other opiate type medications."

The trial was led by Dr Bell, Dr Greg Treston, Dr Ian Ferguson, and Associate Professor Anna Heidigan and ran across two Queensland hospitals — Queen Elizabeth II Jubilee Hospital and Bundaberg Hospital — as well as Liverpool Hospital in Sydney.

The researchers found that some patients receiving propofol were more likely to have slightly lower blood pressure (8%), with a small number of patients experiencing some form of hallucination (5% for ketofol compared with 2% for propofol). In the majority of cases, with both regimens, these were rated as pleasant by patients. Overall, the ketofol group had lower pain scores 30 minutes post-procedure.

The research was published in the November 2016 issue of the Annals of Emergency Medicine.

The research team was awarded an $135,000 Emergency Medicine Foundation (EMF) of Australia research grant, which was fully funded by Queensland Health.

http://bit.ly/2jpRXsa

Alzheimer's disease found to be a diabetic disorder of the brain

Promising treatment for Alzheimer's disease found through similar insulin signalling in the brain and the pancreas

Researchers at Tohoku University have found a promising treatment for Alzheimer's disease, by noticing a similarity in the way insulin signalling works in the brain and in the pancreas of diabetic patients.

"In the pancreas, the Kir6.2 channel blockade increases the insulin signaling, and insulin signaling decreases the blood glucose levels," says Dr. Shigeki Moriguchi. "In the brain, insulin signaling increases the acquisition of memory through CaM kinase II activation by Kir6.2 channel blockade." The research group, led by Dr. Moriguchi and Professor Kohji Fukunaga of the Graduate School of Pharmaceutical Sciences, thus concluded that Alzheimer's disease can be described as a diabetic disorder of the brain.

Memantine, a drug widely used to treat Alzheimer's disease, is a well known inhibitor of the N-methyl-D-aspartate (NMDA) receptors that prevent excessive glutamate transmission in the brain.

Researchers have now found that memantine also inhibits the ATP-sensitive potassium channel (Kir6.2 channel), improving insulin signal dysfunction in the brain.

Glutamatergic neuron
Memantine
Presynaptic membrane
Postsynaptic membrane
Glutamate
NMDA receptor
Ca2+
Ca2+
Kir6.2
Insulin receptor
Insulin
Insulin receptor
Insulin signal
Insulin signal
Insulin
Insulin receptor

The researchers now hope that results of their study and the parallels diners with diabetes, will lead to new treatments for Alzheimer's disease, using the inhibition of Kir6.2 channel.

http://bit.ly/2qNuEF

Why do seabirds eat plastic? The answer stinks

Marine plastic debris is an olfactory trap for seabirds

If it smells like food, and looks like food, it must be food, right?

Not in the case of ocean-faring birds that are sometimes found with bellies full of plastic. But very little research examines why birds make the mistake of eating plastic in the first place.

It turns out that marine plastic debris emits the scent of a sulfurous compound that some seabirds have relied upon for thousands of years to tell them where to find food, according to a study from the University of California, Davis. This olfactory cue essentially tricks the birds into confusing marine plastic with food.

The study, published Nov. 9 in the journal Science Advances, helps explain why plastic ingestion is more prevalent in some seabird species than in others. Tubenosed seabirds, such as petrels and albatross, have a keen sense of smell, which they use to hunt. They are also among the birds most severely affected by plastic consumption.

The Birds' Point of View

"It's important to consider the organism's point of view in questions like this," said lead author Matthew Savoca, who performed the study as a graduate student in the lab of UC Davis professor Gabrielle Nevitt and who is with the Graduate Group in Ecology. "Animals usually have a reason for the decisions they make. If we want to truly understand why animals are eating plastic in the ocean, we have to think about how animals find food."

The study could also open the door to new strategies that address the ocean's plastic problem, which plagues not only seabirds, but also fish, sea turtles and other marine life.

The Flavor Profile of Trash

To learn exactly what marine plastic debris smells like, the scientists put beads made of the three most common types of plastic debris – high-density polyethylene, low-density polyethylene, and polypropylene – into the ocean at Monterey Bay and Bodega Bay, off the California coast.

Taking care not to add to the marine plastic problem, the scientists placed the beads inside specially sewn mesh bags and tied them to an ocean buoy before collecting them about three weeks later.

They brought the retrieved plastic to a somewhat unusual resource for marine ecologists — the UC Davis Robert Mondavi Institute for Wine and Food Science, where researchers are more often found analyzing wine flavor chemistry than smelly trash. Using food-and-wine chemist Susan Ebeler's chemical analyzer, the team confirmed that, sure enough, the plastic reeked of the sulfur compound dimethyl sulfide, or DMS, a chemical cue released by algae, which coats floating plastic.

Co-author Nevitt, with the UC Davis Department of Neurobiology, Physiology and Behavior, had previously established that DMS is a scent that triggers tubenosed seabirds to forage. DMS is released when algae is eaten by animals like krill, one of the birds' favorite meals. So while the algae does not smell like food itself, it does smell like food being eaten, which is the birds' version of a dinner bell.

The study noted that seabirds that track the scent of DMS to find prey are nearly six times more likely to eat plastic than those that do not.

Overlooked Species Eating Plastic

"This study shows that species that don't receive lot of attention, like petrels and some species of shearwaters, are likely to be impacted by plastic ingestion," Nevitt said. "These species nest in underground burrows, which are hard to study, so they are often overlooked. Yet, based on their foraging strategy, this study shows they're actually consuming a lot of plastic and are particularly vulnerable to marine debris."

Matthew Savoca, a UC Davis graduate student of ecology, was also a study co-author.

The study was funded by a National Science Foundation Graduate Research Fellowship and the ASF Office of Polar Programs.

http://medicalxpress.com/news/2008-06-primates-regain-control-paralyzed-limb.html

Primates regain control of paralyzed limb

Neuroprosthetic interface bridges spinal cord injury

On June 23rd, 2015, a primate with spinal cord injury regained control of its paralyzed leg with the help of a neuroprosthetic system called the "brain-spine interface" that bypassed the lesion, restoring communication between the brain and the region of the spinal cord. The results are published today in Nature.

The interface decodes brain activity associated with walking movements and relays this information to the spinal cord – below the injury – through electrodes that stimulate the neural pathways that activate leg muscles during natural locomotion.

The neuroprosthetic interface was conceived at EPFL in Lausanne, Switzerland, and developed together with an international network of collaborators including Medtronic, Brown University and Fraunhofer ICT-IMM. It was tested in collaboration with the University of Bordeaux, Motac Neuroscience and the Lausanne University Hospital (CHUV).

"This is the first time that neurotechnology restores locomotion in primates," says EPFL neuroscientist Grégoire Courtine who led the collaboration. "But there are many challenges ahead and it may take several years before all the components of this intervention can be tested in people."

Decoding brain signals and activating leg muscles

The brain is a huge network of cells called neurons. Information is processed in the brain by transmitting spikes of electricity from one neuron to the next. This electrical spiking gives rise to brain signals that can actually be measured and interpreted.

The lumbar region of the spinal cord also contains complex networks of neurons that activate leg muscles to walk. Bundles of nerves coming from the brain carry the relevant information to the spinal cord about the intended activation of leg muscles.

For intact nervous systems, signals about walking come from a small region (about the size of a dime for primates) of the brain called the motor cortex. Signals from the motor cortex travel down the spinal cord, reach the neural networks located in the lumbar region, and these in turn activate muscles in the legs to produce walking movements.

Spinal cord lesions partly or completely prevent these signals from reaching the neurons that activate leg muscles, leading to paralysis. But the motor cortex can still produce spiking activity about walking, and the neural networks activating muscles in the paralyzed leg are still intact and can still generate leg movements.

How the brain-spine interface works

The brain-spine interface bridges the spinal cord injury, in real-time and wirelessly. The neuroprosthetic system decodes spiking activity from the brain's motor cortex and then relays this information to a system of electrodes located over the surface of the lumbar spinal cord, below the injury. Electrical stimulation of a few volts, delivered at precise locations in the spinal cord, modulates distinct networks of neurons that can activate specific muscles in the legs.

"To implement the brain-spine interface, we developed an implantable, wireless system that operates in real-time and enabled a primate to behave freely, without the constraint of tethered electronics," says Courtine. "We understood how to extract brain signals that encode flexion and extension movements of the leg with a mathematical algorithm. We then linked the decoded signals to the stimulation of specific hotspots in the spinal cord that induced the walking movement."

For partial lesions of the spinal cord, the scientists showed that the primate regained control of its paralyzed leg immediately upon activation of the brain-spine interface. The interface should also work for more severe lesions of the spinal cord, according to the scientists, likely with the aid of pharmacological agents. Note that for these

In spinal lesions, the primate is initially paralyzed and then spontaneously regains full mobility after about three months.

"The primate was able to walk immediately once the brain-spine interface was activated. No physiotherapy or training was necessary," says neuroscientist Erwan Bezard of Bordeaux University who oversaw the primate experiments.

Clinical Trials

"The link between the decoding of the brain and the stimulation of the spinal cord - to make this communication exist - is completely new," says neurosurgeon Jocelyne Bloch of the Lausanne University Hospital (CHUV) who leads the functional neurosurgery department at the Lausanne University Hospital and surgically implanted the brain and spinal cord implants.

She continues, "For the first time, I can imagine a completely paralyzed patient able to move their legs through this brain-spine interface."

In collaboration with EPFL, Bloch is currently leading a clinical feasibility study that evaluates the therapeutic potential of this spinal cord stimulation technology, without the brain implant, to improve walking in people with partial spinal cord injury affecting the lower limbs.

http://bit.ly/2ylcJ02

Mammalian bone gene may be repurposed to fuel cognition in humans

Gene regulating bone growth could also promote brain maturation

A gene that regulates bone growth and muscle metabolism in mammals may take on an additional role as a promoter of brain maturation, cognition and learning in human and nonhuman primates, according to a new study led by neurobiologists at Harvard Medical School.

Describing their findings in the Nov. 10 issue of Nature, researchers say their work provides a dramatic illustration of evolutionary economizing and creative gene retooling--mechanisms that contribute to the vast variability across species that share nearly identical set of genes yet differ profoundly in their physiology.

The research reveals that osteocrin--a gene found in the skeletal muscles of all mammals and well-known for its role in bone growth and muscle function--is completely turned off in rodent brains yet highly active in the brains of nonhuman primates and humans.

Notably, osteocrin was found predominantly in cells of the neocortex--the most evolved part of the primate brain, which regulates sensory perception, spatial reasoning and higher-level thinking and language in humans.

The gene's marked presence in an area of the brain responsible for higher-level function and thought, the researchers said, suggests a possible role in the development of cognition, a cardinal feature that distinguishes the brains of human and nonhuman primates from those of other mammals.

Brain development in humans and other primates is profoundly affected by sensory experience and social interactions. Scientists have long sought to unravel genes in the brain that are turned on and off by experiences to fuel the rise of brain functions unique to such complex species.

The HMS team's findings--part of an ongoing quest to elucidate the mechanisms that underlie human brain development, function and disease--reveal that osteocrin is precisely one such gene, activated by sensory stimulation. Furthermore, the team added, this is the first illustration of evolutionary gene repurposing in the brain.

"We have uncovered what we believe is a critical clue into the evolution of the human brain, one that gives us a glimpse into the genetic mechanisms that may account for differences in cognition between mice and humans," said senior investigator Michael Greenberg, the Nathan Marsh Pusey Professor of Neurobiology and chair of the HMS Department of Neurobiology.

For their experiments, the team analyzed RNA levels--the molecular footprints of gene activity--in the brain cells of mice, rats and humans.

Although many of the same genes were activated in both mouse and human brain cells, the scientists observed, a small subset of genes was activated solely in human brain cells. Much to the scientists' surprise, the bona gene osmocrin was most highly expressed in the human brain yet completely shut off in the brain cells of mice.

Going a step further, the scientists placed brain cells from all three species in lab dishes and chemically re-created conditions that mimic sensory stimulation. Chemical stimulation activated osmocrin selectively in excitatory neurons, so called for their role in stimulating rather than dampening nerve signaling. But researchers noted something even more intriguing: The activity of the gene was most intense in neurons of the neocortex, the topmost layer of cells covering the brain and responsible for higher-level cognition, such as long-term memory, thought and language. At the same time, osmocrin was noticeably absent from other parts of the brain responsible for noncognitive functions such as spatial navigation, balance, breathing, heart rate and temperature control.

When researchers compared osmocrin levels to levels of another brain protein, BDNF, well known for its role in neuronal growth and repair, they noticed another striking difference. While BDNF was present throughout the brain, osmocrin was restricted to the neocortex and, to a lesser extent, the amygdala, an area of the brain thought to play a role in memory formation, decision making and emotional responses. Osmocrin was also markedly expressed in cells of the temporal lobe, which houses functions such as learning, memory and audio-visual processing—and the occipital lobe, which houses the visual cortex, the area of the brain responsible for the processing of visual information.

Further analysis revealed that in the primate brain, sensory stimulation appears to switch on osmocrin through a previously unknown DNA enhancer. Enhancers—snippets of DNA that act as the genome's regulators—are the "handles" that turn on some genes while shutting off others. In doing so, enhancers can profoundly alter genetic expression, fueling dramatic differences between organisms with nearly identical DNA. The rodent versions of osmocrin lacked the stimulation-activated DNA enhancer, the analysis showed.

In yet another critical observation, researchers found the osmocrin enhancer was, in turn, switched on by a protein called MEF2. Mutations in MEF2 are a well-established cause of intellectual disability and neurodevelopmental disorders, so the link between the two begs further study, the researchers said, as it may portend a role for osmocrin in such developmental anomalies.

"Humans share many genes with rodents and as much as 90 percent of their DNA in some parts of the genome," says co-first author Gabriella Brealing, a neurobiologist at HMS. "In this case we see how turning up the expression of the same gene in a different location may precipitate dramatic differences in the function of brain cells."

Further analysis revealed that osmocrin's activation curbed the growth of neuronal dendrites—branchlike projections responsible for transmitting signals from one brain cell to the next.

"Restricting dendritic growth is a precision-enhancing mechanism, essential to ensuring that neuronal wires don't get crossed and compromise signal transmission from one cell to the next," says study first co-author Bulent Ataman, a neurobiologist at HMS.

This observation, Ataman added, suggests that osmocrin's activity may help enhance nerve cell agility and proper signal transmission to ensure robust communication across neurons.

To confirm that the activity-induced gene expression observed in nerve cells in the lab also occurred in the functioning, intact brain, researchers temporarily blocked vision in one eye of a macaque, a common technique to study activity-triggered brain plasticity and visually-induced gene activation in the visual cortex. This proof-of-concept experiment, they surmised, would reveal whether osmocrin is, indeed, awakened by visual stimulation and shut off by its absence. A day later, the researchers observed that osmocrin expression was markedly higher in cells from the visually intact parts of the macaque brain, compared with cells in vision-deprived areas.

The findings illustrate a foundational principle in neurobiology—abnormal visual experiences can interfere with the development and function of brain cells in the visual cortex, a phenomenon first described more than 50 years ago by David Hubel and Torsten Wiesel, two of the founding members of the Department of Neurobiology at HMS. The two shared the 1981 Nobel Prize in Physiology or Medicine for their discoveries on visual information processing in the brain.

"Nature and nurture interact to wire up the brain, and abnormal vision can alter that wiring," said Margaret Livingstone, the Takeda Professor of Neurobiology at HMS. "Our observations reveal the molecular basis for what Hubel and Wiesel observed more than half a century ago."

Researchers say their findings have sparked more questions, including exactly how osmocrin interacts with neurons, precisely what factors regulate its expression and, most importantly, how it can alter brain physiology in disease and health.

The research was funded by the National Institutes of Health grants RC2MH089912 and P50MH094271 with additional support from the Ellen R. and Melvin J. Gordon Center for Integrative Neuroscience, the Simons Foundation, the Vision Research Foundation and by the NIH's Ruth L. Kirschstein National Research Service Award 5F32NS086278.

Co-investigators included David Hornes, Monty Sing, *Mollie Baker-Salisbury, Fu-Lyan Yap, Albert Hsieh, Rex Liu, Alex Ko, Jennifer Spangler, Chris Zhu-Jones, Nikhil Sharma, Linda Hu, Jeremy Nathan, Nitin Griffith, Jennifer Sanchez, Ashley Ortiz, Chris Stevens, Mother Adli, Marina Chekrene, Trond Sessen, Christopher Walsh, and Vladimir Berezovski.*

http://bit.ly/2eYkcfP

Expensive new cancer drugs have little effect on survival of many cancers

New cancer drugs approved in the past 10 years may have little effect on survival in adults with cancer

Despite considerable investment and innovation, new cancer drugs approved in the past 10 years may have little effect on survival in adults with cancer, raising a number of concerns, argues an expert in The BMJ today.

Peter Wise, a former consultant at Charing Cross Hospital in London, says spending an annual six figure sum to prolong life by a few weeks or months "may be inappropriate" for many patients. In 2015, global sales of cancer drugs were around $110bn (£85bn; €98bn).

He calls for stricter drug approval criteria and improved consent processes "to achieve ethical treatment and reduce cancer costs."

Cancer survival has improved in recent decades, he explains. In the US, for example, five year relative survival in adults with solid cancer increased from 49% in to 68% over 40 years.

But how much of the improvement in cancer survival can we attribute to new drugs, he asks? Other factors are more likely to have been responsible. Many new drugs approved in the last decade prolonged life by just one to two months.

"The approval of drugs with such small survival benefits raises ethical questions, including whether recipients are aware of the drugs' limited benefits, whether the high cost/benefit ratios are justified, and whether trials are providing the right information," writes Wise, whose major interests lie in the ethical elements of medical research and care.

He draws attention to limitations of cancer drug trials, such as the use of surrogate endpoints that allow earlier approval of new drugs, but are not always true indicators of survival benefit. And he warns that the marginal responses in clinical trials may not even apply to the majority of patients treated outside trials.

He is hopeful that the recent integration of the Cancer Drugs Fund into the National Institute of Health and Care Excellence (NICE) in England might make it possible to monitor the "real-world benefit" of these drugs.

He also raises concern over the US Food and Drug Administration (FDA)'s accelerated and "breakthrough" category which, he says, compounds the risk of premature approval on limited evidence.

"The low bar of approval for these expensive drugs ignores the ethical principle of fairness and equity," he writes. "By promoting marginally better treatment of poorly *responsive* cancers it diverts valuable

research that might be better employed for other health needs, within and outside cancer care." A lack of fully informed consent for cancer treatment is also a concern, often leading to misinformed patients with unrealistic expectations, he adds.

"Good cancer care demands empowerment of patients with accurate, impartial information followed by genuinely informed consent in both the clinical trial and therapeutic settings," he writes. "Ethical impediments to sound practice need to be addressed and corrected."

"Above all, the threshold for approval of new and existing cancer drugs needs to be raised – using more meaningful disease specific criteria of risk-benefit and cost-benefit," he concludes.

Analysis: Cancer drugs and survival http://www.bmj.com/cgi/content/full/337/7672/a2502

http://bit.ly/2ZYRFc

It's not a bird! It's not a plane! It's the fastest flying mammal, says UT study

When most people think of animals moving at high speed, they envision cheetahs or swiftly diving raptors. They can now add the Brazilian free-tailed bat – a tiny nocturnal mammal – to the list.

KNOXVILLE – A new study from the University of Tennessee, Knoxville, shows that the Brazilian free-tailed bat can achieve flight speeds faster than those previously documented for any bat or bird. They are achieving short bursts of ground speeds of up to 100 miles per hour.

The research was published today in the journal Royal Society Open Science. The study's lead author is Gary McCracken, UT professor of ecology and evolutionary biology and one of the world's leading experts on bats. Other collaborators include researchers from the Max Planck Institute for Ornithology and the University of Konstanz, both in Germany, and from Boston University and Brown University.

Much of the current literature has indicated that birds fly faster and bats are slower and more maneuverable. The new study demonstrates otherwise.

The researchers conducted the study in southwestern Texas using a novel airplane tracking method. They caught seven Brazilian free-tailed bats, each weighing 11 to 12 grams, as they emerged from the entrance of the Fain Cave at night. They then attached 0.5-gram radio transmitters to their backs using surgical glue.

Until now, the fastest bird records were collected during short flight segments using tracking radar and high-speed video. McCracken and collaborators used an airplane to follow the complete flight track of the bats. They followed one bat per night.

The researchers' models indicate that tail winds did not assist the bats' flight speed. They observed that the bats did exactly what airplanes and birds do, depending on wind conditions.

"When they have a headwind, they fly faster. When they have a tailwind, they slow up," he said. "This is exactly what has been demonstrated in other flight machines, from airplanes to birds."

The study results suggest a reevaluation of the performance abilities and capabilities of bats, McCracken said, noting that their flight performance has been underappreciated.

http://bit.ly/2g1JIfs1

Licorice compound interferes with sex hormones in ovary, study finds

Isa diargen steroid sex hormone production in the ovary

CHAMPAIGN, ILL. – A study of mouse reproductive tissues finds that exposure to isoliquiritigenin, a compound found in licorice, disrupts normal sex hormone production in the ovary, researchers report. This is the first study to examine the effects of this chemical on the ovary.

Exposure to high levels of the compound, which the researchers call "iso," lowered the expression of key genes involved in hormone production, the researchers found. In particular, expression of a gene for aromatase, an enzyme that converts testosterone to estrogen, dropped by 50 percent or more.

The findings are reported in the journal Reproductive Toxicology.

Though preliminary – more research must be done to determine iso's effects in living animals – the discovery is concerning, said University

of Illinois comparative bioscientcen pmfemcnr Jsdl Flnss, who ltel the study with ossnrcher Sharada Mubalingam.

"In general, when you start to have lcwsr hormone levels, you crsdl start to have problems with reproduction," Flnss sasd.

"Jsdl bst nasc estrogen is also ltnportsnt for healthy brsins, healthy bonss, a healthy cardiovscsdar system, if the lewls are depleted for tcn lcng, you cssdl hsve problems with those systems. We hsvent sbnwn that to be the cssc. That's jsst a psssdbility. "I wssdl say, thcsgh, that a 50-plss percent dtrcp in aromatsse in hsmsns wcssdl be a sertcsn psssdlem for fertility and for cdher things," Flnss sasd.

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Whsdc lcrscrce rcsscsdt sscsd psssdfsed fcrcss cf sscs sscs sssscd in hscbsdsd sssdpsssdscssdscssdsc

Yoshihiro Sato, MD, of Minoo Hospital in Tagawa, Japan, admitted to scientific misconduct. Sato accepted full responsibility, admitting fabrication of the fraudulent Neurology papers, which reported on the effects of therapies to reduce hip fractures both after stroke and in Parkinson's disease patients. Sato stated that none of the coauthors participated in any misconduct and were named as authors on an honorary basis only. Sato requested retraction of the three studies.

For the analysis of the 33 trials, 26 of which Sato was lead author, Holland's team conducted a rigorous review and found reported results that differed markedly from what could be expected statistically; further, the results were remarkably positive.

The characteristics of the groups of people chosen to participate in the trials were much more similar than would have happened by chance. The trials reported large reductions in hip fractures, no matter what treatment was used, that were much greater than those reported in similar trials from other research groups. Overall in the 33 trials, the people receiving the therapy were 78 percent less likely to break a hip than the control group, while several meta-analyses of other trials found either no benefit of the treatments or a benefit of less than 40 percent.

Holland's team also found multiple examples of inconsistencies between and within trials, errors in reported data, misleading text, duplicated data and text as well as uncertainties about ethical oversight. "The researchers were remarkably productive, conducting 33 randomized controlled trials within 15 years, the outcomes of each being remarkably positive," said Holland. "Our analysis suggests that the results of at least some of these trials are not reliable. In addition, results from these trials were not consistent with results found in similar studies by other researchers."

"This statistical analysis demonstrates probable scientific misconduct on a large scale," said Robert A. Gross, MD, PhD, of Rochester, N.Y., Editor-in-Chief of Neurology and Fellow of the American Academy of Neurology, who wrote a corresponding editorial. "Fraud in an individual paper may be difficult to detect. One cannot conclude that any one study in the analysis is, or is not, fraudulent. As part of our due process, we have notified other editors of journals that published papers by Sato and colleagues, communicated with Sato's institution, and published retractions of the three papers and a letter published in Neurology."

The analysis was supported by the Health Research Council of New Zealand and the Health and Social Care Directorate of the Scottish Government.

http://bit.ly/2IFqHw

High-intensity statins linked to better survival rates of cardiovascular patients

A large national study has confirmed the value of high-intensity statin treatments for people with cardiovascular disease, according to researchers at the Stanford University School of Medicine.

Over the duration of a year, the researchers found that patients taking high-intensity statins had an increased chance of survival over those on moderate-intensity statins. The study will be published online Nov. 9 in JAMA Cardiology.

Statins, a class of drugs that lowers cholesterol levels in the blood, are commonly prescribed for preventing the acceleration of cardiovascular disease caused by the buildup of plaque in the arteries, which can lead to heart attacks and stroke.

Health-care providers have long debated the benefits of prescribing high-intensity statins to their patients with cardiovascular disease. Patients, in turn, have been hesitant to take them because of equivocal messages from their doctors and internet searches of patient and doctor perspectives.

"Previously, there was definitely a certain amount of fear on the patient's part because most people don't like taking medication," said Paul Heidenreich, MD, professor of cardiovascular medicine and the study's senior author. Some studies have shown an increased risk of side effects, such as diabetes or muscle damage, associated with higher-intensity statins.

Conflicting recommendations

In 2013, the American College of Cardiology and American Heart Association jointly recommended high-intensity statin therapy for patients with atherosclerotic cardiovascular disease who were no older than 75. The ACC/AHA guidelines differed, *however*, from guidelines established in 2014 by the Veterans Affairs Health Care System, which recommended only moderate-intensity statins, noting the lack of conclusive evidence that higher-intensity statins are more beneficial than those of moderate intensity.

In their study, Heidenreich and his team found evidence to support the ACC/AHA guidelines. They determined that high-intensity statins do in fact increase rates of survival, not only in younger and middle-aged patients with cardiovascular disease, but also in a patient population not well-studied: adults over 75.

"The greatest strength of this study is that we used a very large, well-defined clinical cohort," said Fatima Rodriguez, MD, a cardiology fellow at Stanford and the study's lead author. "The results show that high-intensity statins confer a survival advantage for patients with cardiovascular disease, including older adults."

Large sample size reduces possibility of chance

The researchers studied the medical records of 509,766 patients across the country receiving care from the Veterans Affairs Health Care System. "This is a very large patient population rich in cardiovascular disease," said Rodriguez. "In addition to defining this large, national patient population, we also had access to their detailed clinical data, including comorbidities and cholesterol values."

The primary purpose was to look at overall patient death rates from 2013 to 2014, the researchers said. They included patients with coronary artery disease, cerebrovascular disease and peripheral artery disease. "These are basically the three main areas affected by plaque buildup — the heart, the brain and the large arteries of the *rest* of the body," Heidenreich said.

Patients were taking high-intensity, moderate-intensity or low-intensity statins in many different but commonly prescribed forms, such as rosuvastatin and atorvastatin. The researchers also followed one group that wasn't taking any statins. Patients had different severities of cardiovascular disease, making some more likely to be prescribed higher-intensity statins than others. So the researchers assigned each patient a score for the propensity to receive high-intensity statins and adjusted the results of the study accordingly.

The results showed a 9 percent increased chance of survival for patients taking high-intensity statins compared to those receiving moderate-intensity treatments. "We found basically the same risk reductions reviewed by the Veterans Affairs guidelines, but they didn't think the benefit was significant because the sample size was small," Heidenreich said. "We have so many more patients, we can be confident that it wasn't due to chance."

Examining specific patient groups

The study considered data from patients over 75 — a group little studied in clinical trials. It found that patients between the ages of 75 and 85 taking high-intensity statins had a survival-rate benefit comparable to that of younger patients: a 9 percent higher chance of survival compared to those on moderate-intensity statins.

"Our results suggest that clinical trial data from heart studies for those younger than 75 could also be applied to this older population," Heidenreich said.

Finally, they studied the effect of different doses within the high-intensity statin group. Patients treated with the maximum dose of statins were 10 percent more likely to survive than patients on submaximal doses. "This suggests to practitioners that instead of starting a patient on a *low dose*, just to go ahead and put them on the maximum dose they can tolerate," Rodriguez said.

A limitation of the study was that the researchers were unable to determine whether patients died of cardiovascular disease or another cause.

Settling the debate

The next step, researchers said, is to find out why some patients who should be on high-intensity statins are not. They hope doctors will take their study's results into consideration when prescribing statins. "There are a lot of guidelines and recommendations out there, so I think we also have to make the system better," Rodriguez said. "Maybe hospitals can employ a clinical reminder to doctors, a message that pops up on the doctor's screen that asks why a cardiovascular patient isn't on a high-intensity statin."

The researchers also hope to follow up on longer-term data from these patient populations. "Not only do we hope to continue studying this population, but we also hope to study patients without prior cardiovascular disease but who are at high risk for it," said Rodriguez. Finally, they hope these results will help to settle the debate on which guidelines doctors should use when prescribing statins to patients. Heidenreich said, "We think this should give clinicians, physicians and nurse practitioners more comfort in following the American College of Cardiology and American Heart Association guidelines and putting people with prior cardiovascular disease on a high-intensity statin."

The work is an example of Stanford Medicine's focus on prevention health, the goal of which is to prevent people from ever developing cardiovascular disease, diagnosis and treat disease in its early stages. Stanford cardiologist Euan Ashley, MD, clinical professor of medicine and Joshua Knowles, MD, PhD, assistant professor of medicine, Stanford's Department of Medicine supported this study.

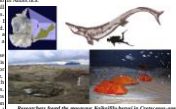
http://bit.ly/2jb4PFu

Newfound Ancient 'Sea Monster' Is Largest Yet from Antarctica

About 66 million years ago, an ancient sea monster the height of a five-story office building once gnashed its sharp teeth as it swam around the dark waters of Antarctica, a new study finds.

By Laura Geggel, Senior Writer | November 9, 2016 12:20pm ET

The newfound beast, known as a mosasaur – a Cretaceous-age aquatic reptile that sped through the ancient seas using its paddle-like limbs and long tail – is only the second fossilized mosasaur skull ever found in Antarctica.



Researchers found the mosasaur Kaikaifilusherveri in Cretaceous-age rocks on Seymour Island in Antarctica (upper left). Kaikaifilu was quite large, See the human for scale (upper right). Paleontologists struggled through Antarctica's extreme climate when they excavated the specimen (lower right). The muddy site where they excavated the specimen (lower left).

Mors, R.A. et al. Cretaceous Research, 2016

The mosasaur specimen is different enough from other known species that it qualifies for its own genus and species. Researchers named it Kaikaifilu hervei after "Kai-Kai filú," an almighty giant reptile that roam the sea in legends from the Mapuche culture from southern Chile and Argentina. The species name honors Francisco Hervé, a world-renowned Chilean geologist and Antarctic explorer, the researchers said.

Scientists with the Chilean Paleontological Expedition discovered the mosasaur skull on Seymour Island in January 2011. The team had run into bad weather, and only during the last few days in the field, while

they were snacking around in knee-deep mud, did they discover the enormous fossil, the researchers said.

Based on the skull's anatomy and size (4 feet, or 1.2 metres, long), the reptile's entire body stretched about 33 feet (10 m), making it the largest marine predator in the region, the researchers said.

North versus South

It's not uncommon to find mosasaur remains in North America, especially in the seaway that once divided the East from the West in North America. But with the exception of New Zealand, it's relatively rare to find the giant creatures in the Southern Hemisphere, said Rodrigo Otero, a paleontologist at the University of Chile and the lead researcher on the study.

Still, K. hervei was a close relative of — and similar in size to — the North American mosasaur known as Tylosaurus, which lived about 20 million years earlier. K. hervei was also a close relative of another Antarctic mosasaur (Tetrahmosasrus antarcticus), which was smaller, with a skull about 2.3 feet (0.7 m) in length, and lived about 5 million years before K. hervei did, the researchers said.

What's more, other researchers have found an array of other isolated mosasaur teeth in the rocks of Antarctica. Mosasaurs have multiple types of teeth (a condition called heterodonty), meaning that differently shaped teeth might belong to the same mosasaur species. Thus, researchers will need to be careful not to overestimate the number of species as they review the discovered teeth, the researchers said.

Warm Antarctic

Although Antarctica is now a frigid continent, it was warmer during the dinosaur age, the researchers said. A slew of animals swam in the region's waters, giving K. hervei a smorgasbord of contemporaries to dine on, they said.

For instance, the plesiosaurs - mostly long-necked marine reptiles that ate plankton via filter feeding - likely would have been prime targets for K. hervei, the researchers said.

Prior to this research, the known mosasaur remains from Antarctica provided no evidence for the presence of very large predators like Kaikaifilu, in an environment where plesiosaurs were especially abundant," Otero said in a statement. "The new find complements one expected ecological element of the Antarctic ecosystem during the latest Cretaceous." The study was published online Nov. 4 in the journal Cretaceous Research.

http://bit.ly/2kTfFR

Vitamin D Deficiency Widely Overestimated, Doctors Warn

Too much vitamin D can lead to high levels of calcium in the blood, which can cause nausea, constipation, kidney stones, an abnormal heart rhythm and other problems.

Too much vitamin D can lead to high levels of calcium in the blood, which can cause nausea, constipation, kidney stones, an abnormal heart rhythm and other problems.

Doctors are warning about vitamin D again, and it's not the "we need more" news you might expect. Instead, they say there's too much needless testing and too many people taking too many pills for a problem that few people truly have.

The nutrient is crucial for strong bones and may play a role in other health conditions, though that is far less certain. Misunderstandings about the recommended amount of vitamin D have led to misinterpretation of blood tests and many people thinking they need more than they really do, some experts who helped set the levels write in Thursday's New England Journal of Medicine.

Correctly interpreted, less than 6 percent of Americans ages 1 to 70 are deficient and only 13 percent are in danger of not getting enough. That's concerning, "but these levels of deficiency do not constitute a pandemic," the authors write. Yet people may think there is one.

'Sunshine vitamin'

Blood tests for vitamin D levels — not advised unless a problem like bone loss is suspected — are soaring. Under Medicare, there was an

83-fold increase from 2000 to 2010, to 8.7 million seen last year, at $40 apiece. It's Medicare's fifth most common test, just after cholesterol levels and ahead of blood sugar, urinary tract infections and prostate cancer screening.

"I'm not sure when it got popular to check everybody for vitamin D deficiency," but patients often ask for it, especially baby boomers, said Dr. Kenny Lin, a Georgetown University family physician and preventive medicine expert. Vitamin D pill use also grew, from 5 percent of Americans in 1999 to 19 percent in 2012.

That may be due to many reports suggesting harm from too little of "the sunshine vitamin," called that because our skin makes vitamin D from sun exposure. It's tough to get enough in winter or from dietary sources like milk and oily fish, though many foods and drinks are fortified with vitamin D and labels soon will have to carry that information.

Need for any vitamin varies

Too much vitamin D can lead to high levels of calcium in the blood, which can cause nausea, constipation, kidney stones, an abnormal heart rhythm and other problems.

"We're not saying that moderate-dose supplements are risky, but more is not necessarily better," said Dr. JoAnn Manson of Brigham and Women's Hospital in Boston. She and several other advisers to the Institute of Medicine, which set the RDA, or recommended dietary allowance, wrote the journal article.

People vary, biologically, in how much of any vitamin they need. The institute estimated this by comparing various intake and blood levels with measures of bone health. They estimated that, on average, people need about 400 international units of vitamin D per day, and 600 for people over 70.

To be safe and ensure that everyone gets enough, they set the RDA at the high end of the spectrum of the population's needs — 600 to 800 units, depending on age. So by definition, nearly everyone's true requirement is below that.

Big D-3 study in works

Many people and their doctors regard the RDA and its corresponding blood levels as a threshold that everyone needs to be above, the authors write. As a result, people often are told they are inadequate or deficient in D when, in fact, they're not.

"If you're chasing a lab number, that will lead to many people getting higher amounts of vitamin D than they need," and labs vary a lot in the quality of testing, Manson said. The bottom line: Get 600 to 800 units a day from food or supplements and skip the blood test unless you have special risk factors, Manson said.

A big study she is helping lead is testing whether higher levels lower the risk of cancer, heart disease, stroke, memory loss, depression, diabetes, bone loss or other problems. Nearly 26,000 people have been taking 2,000 units of D-3 (the more active form of vitamin D, also known as cholecalciferol) or dummy pills every day for five years. Results are expected in early 2018.

http://bit.ly/2Ge1Ty

Oldest Beer Brewed from Shipwreck's 220-Year-Old Yeast Microbes

An international team of scientists has recreated a 220-year-old beer recipe, using live yeast recovered from a bottle found in an 18th-century shipwreck in Australia.

By Tom Metcalfe, Live Science Contributor

Yeast microbes from the world's oldest bottle of beer - a 220-year-old bottle found in one of Australia's earliest shipwrecks - are being used to create a new, modern beer with the characteristic taste of the 18th-century brew.

The yeast was grown from the contents of a bottle of beer recovered from the wreck of the Sydney Cove, a British trading ship that got caught in a storm near the island of Tasmania, off Australia's south coast, in 1797 while on its way from Calcutta to the prison colony at Port Jackson, now Sydney.

The crew of the Sydney Cove survived by grounding the sinking ship on a tiny island off southern Tasmania, now called Preservation Island, which is part of the inspiration for the name of the recreated beer: Preservation Ale.

The researchers used the yeast to brew a mild-tasting beer using a traditional recipe from the time, and they say it has a distinct flavor.

"It's got quite a sweet taste — some people have described it as almost a cider or fresh taste — which has come from the yeast," said project leader David Thurrowgood, a conservator and chemist at the Queen Victoria Museum at Launceston in Tasmania.

The researchers also uncovered a historical account of a celebrated English beer from the time that was known for its sweet, cider-like flavor, similar to the beer brewed from the reanimated yeast.

"That was quite a surprise, but having found that reference, and to have that particular taste come out in the beer ... it showed that the beer did actually have a distinctive taste at the time that we're only rediscovering now," Thurrowgood told Live Science.

But beer aficionados will have to wait a while longer to sample the 220-year-old taste. Thurrowgood said several brewing companies are keen to market Preservation Ale, but so far, the entire stock consists of a few bottles brewed for his research.

Beer on board

The yeast microbes used to brew the recreated beer were grown from samples taken from one of 26 beer bottles found in the hold of the Sydney Cove wreck during excavations by marine archaeologists in the 1980s.

A single unopened bottle from the wreck now enjoys pride of place at the Queen Victoria Museum as the world's oldest bottle of beer — the oneest contender being a 133-year-old bottle of lager in the Carlsberg Museum in Denmark, Thurrowgood said.

DNA tests show the shipwrecked yeast microbes are related to yeast species used in so-called Trappist ales brewed in monasteries in Europe, and Thurrowgood thinks the bottles contained a premium beer exported from England for military officers at Port Jackson.

Bottles of wine, brandy and gin were also found aboard the Sydney Cove, as well as several casks of cheaper beer for mass consumption, the researchers said.

The scientists from Australia, Belgium, France and Germany have revived five distinct species of yeast microbes from the shipwrecked beer bottle, as well as several species of bacteria, which will provide rare information about the microorganisms in human diets from a time before the Industrial Revolution in Europe.

"People talk about autoimmune diseases and other issues [relating to] the fact that we have quite a clean diet today, whereas in the past we had a diet full of microbes," Thurrowgood said. "This is one of the few chances we've got to actually test those microbes, and actually see what they were."

Shipwreck survivors

Thurrowgood and his colleagues plan further studies of the bottles of wine recovered from the shipwreck, which may also hold microorganisms that can be revived after 220 years.

"We can definitely see dead cells within the wine bottles, and we're much less likely to find live materials in there, but you never know until you've done the work," he said.

The future of Preservation Ale, meanwhile, is at the center of commercial discussions that the researchers hope will develop into revenue to preserve the museum's important collection of artifacts from the Sydney Cove wreck and the survivors' camp sites on Preservation Island, the researchers said.

Thurrowgood added that the possibilities include establishing a mini brewery at the historic museum buildings in Launceston, or creating a homebrew beer based on the 18th-century yeast strain.

After the Sydney Cove ran aground in February 1797, a party of survivors set out in an open boat to reach the colony at Port Jackson.

The voyage took them across Bass Strait, between Tasmania and the Australian mainland, where they were searched again on the coast.

The voyagers then faced an overland trek of more than 370 miles (600 kilometers) through unknown territory peopled by both friendly and hostile aboriginal tribes. Of the 17 individuals who set out, just three made it to Port Jackson in May 1797, according to an official letter by the colony's governor, John Hunter.

"They were the first Europeans to do that trek, so in terms of early colonial history, it was an enormous trip and tale of survival — I don't know how they did it," Tharoyogod said.

https://bit.ly/2Ffu8c5

Scientists reveal how a common virus triggers blood cancer

Epstein-Barr virus takes control of two genes involved in cancer development

Scientists at the University of Sussex, trying to uncover how the common Epstein-Barr virus causes blood cancer in adults and children, have discovered how the virus takes control of two genes involved in cancer development so it can switch them on or off.

The research team led by Professor Michelle West, set out to determine how the virus controls two genes; MYC, a gene known to drive cancer development when it is altered or switched on at high level and BCL2L11, a gene which normally triggers cell death to prevent cancer, but can be turned off by the virus.

With thanks to funding from the charity Bloodwise, the scientists discovered that the virus controls the MYC and BCL2L11 genes by hijacking 'enhancer' DNA regions which are situated far away from the genes. These enhancers act as 'control centres' and are able to contact and control genes from long distances by the looping out of the intervening stretches of DNA.

Professor West's team, who are based in the University's School of Life Sciences, found that Epstein-Barr virus turns on the MYC gene by increasing contacts between a specific set of enhancers and the genes. The scientists believe this may explain how the virus causes the changes to the MYC gene that are found in Burkitt's lymphoma.

The team also discovered new enhancers which control the BCL2L11 gene. In this case, they found that Epstein-Barr virus stops these control centres from contacting the gene. Encouragingly the team have discovered that this blocking effect can be reversed by using a specific drug – paving the way for new treatments.

Professor Michelle West, from the University of Sussex, said: "This is a key step towards uncovering how this common virus which, affects thousands of people every year, causes blood cancer.

"It is now important to carry out further studies to determine how the Epstein-Barr virus controls other genes that are associated with lymphoma. This will tell us more about how the virus drives lymphoma development and will help to identify new ways of targeting Epstein-Barr virus-infected cancer cells with specific drugs."

Dr Alasdair Rankin, Research Director at Bloodwise, said: "We have known for many years that the Epstein-Barr virus causes various types of lymphoma, but we were never sure of the exact mechanisms. These new findings have shed light on how the virus is able to drive blood cancer development by altering the behaviour of genes that control cancer growth.

"By mapping out the complex genetic interactions that help lymphoma cells grow and survive, this research can guide the design of new treatments to target the disease. It may also help to identify those drugs currently used to treat other diseases that could be effective in treating these types of lymphoma."

Professor West has been granted £460,000 by the blood cancer charity Bloodwise for two research projects that are helping to uncover how a number of common types of leukaemia and lymphoma arise in children and adults and to find new ways to treat them. The research team are studying how the common Epstein-Barr virus can sometimes trigger the development of blood cancers including Burkitt and Hodgkin lymphomas.

The study entitled: "NFIC activation and IFIG-C1 silencing by a tumor virus through the large-scale reverse[]genetics of enhancer-promoter hubs" has been published in eLIFE Journal and can be found here http://dx.doi.org/10.7554/eLife.02718

http://dx.doi.org/10.7554/eLife.02718

Red squirrels in the British Isles are infected with leprosy bacteria

Microbiologists at EPFL and the University of Edinburgh have discovered that red squirrels in Britain and Ireland carry the two bacterial species that cause leprosy in humans.

Once rampant in medieval Europe, leprosy dramatically declined by the end of the Middle Ages for reasons that are still unclear. About a century ago, leprosy in Europe virtually disappeared, at least among humans. Examining diseased red squirrels from England, Ireland, and Scotland, scientists at EPFL and the University of Edinburgh have now discovered that the same bacteria that cause leprosy in humans also infect red squirrels. The work is published in Science.

Leprosy is an infectious disease that mainly affects the skin, peripheral nerves, the upper respiratory tract, and the eyes. It is caused by the bacteria Mycobacterium leprae and the recently discovered Mycobacterium lepromatosis. One of the most ancient diseases, leprosy has had an enormous social impact across multiple cultures throughout history. Largely controlled today thanks to antibiotics, there are still over 200,000 new cases of leprosy reported each year worldwide.

A little-known fact is that leprosy also affects animals, such as armadillos, which have reportedly caused a few cases of animal-to-human (or "zoonotic") infections. Drawing from this evidence, the EPFL and University of Edinburgh researchers, led by Stewart Cole, Edinburgh carried out DNA tests on 110 red squirrels from England, Scotland, and Ireland. Some of these animals showed clinical symptoms of leprosy, while others did not; nonetheless, most were found to be infected with leprosy bacteria.

Surprisingly, red squirrels from Brownsea Island, off the south coast of England, were infected with a strain of M. leprae that is closely related to one found in a skeleton of a leprosy victim that was buried in Winchester 750 years ago, just 70 km from Brownsea Island.

On the other hand, red squirrels from Scotland and Ireland and the Isle of Wight (South England) were found to be infected with the other leprosy bacterium, M. lepromatosis. This species causes leprosy in humans in Mexico, and further analysis showed that the two strains from Mexico and Europe diverged from a common ancestor around 27,000 years ago.

"It was completely unexpected to see that centuries after its elimination from humans in the UK M. leprae causes disease in red squirrels," says Stewart Cole. "This has never been observed before."

The study shows how a pathogen can remain undetected in the environment even hundreds of years after it has been cleared from the human population. "The discovery of leprosy in red squirrels is worrying from a conservation perspective but shouldn't raise concerns for people in the UK," says Anna Meredith. "We need to understand how and why the disease is acquired and transmitted among red squirrels so that we can better manage the disease in this iconic species."

But is there a danger to humans? "There is no reason for panic," says Andrej Benjak, one of the paper's lead authors. "Autochthonous leprosy has not been detected in the UK in decades, though we cannot exclude the possibility of rare, unreported or misdiagnosed cases that originated within the UK." He suggests increasing efforts to monitor the disease, as part of the WHO's global Leprosy Surveillance Programme, where there is still room for improvement.

"The next logical step after this study is to check the red squirrel population outside the British Isles, and that includes Switzerland," says Benjak. "Even if there is leprosy in red squirrels in continental Europe, the risk of transmission to people is generally low because of their limited contact with humans, and hunting red squirrels is forbidden in most European countries."

This work was supported by grants to S.J.C. from the Fondation Bazet-Fréderam, the Swiss National Science Foundation and the Thomas O'Hanlon Memorial Award to Vincent Gladstone.

Gustavo Arenal, Jorge del-Pozo, Andrej Benjak, Karen Stevenson, Victor R. Simpson, Philippe Busso, Joyce McLaughlin, Chloé Loiseau, Carlo Lanzas, Jenny Schoenberg, Darren J. Shaw, Jérémie Pizon, Lucia Vera-Cabrera, Jesús S. Velasco-Pelón, Pengal McDermott, Stephen V. Gordon, Stewart T. Cole, Nicolas Gicquel, Sebastien Gagneux, Jerome Rigouts, Stephen H. Gordon, Stewart T. Cole

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http://bbc.in/2fDuzU4

'First flu' affects lifetime risk

A person's chances of falling ill from a new strain of flu are at least partly determined by the first strain they ever encountered, a study suggests.

By Caroline Robinson Health editor, BBC News website

Research in Science journal looked at the 18 strains of influenza A and the hemagglutinin proteins on its surface. They say there are only two types of this protein and people are protected from the one their body meets first, but at risk from the other one. A UK expert said that could explain different patterns in flu pandemics.

'Lollipop flavours'

The researchers, from University of Arizona in Tucson and the University of California, Los Angeles, suggest their findings could explain why some flu outbreaks cause more deaths and serious illnesses in younger people.

The first time a person's immune system encounters a flu virus, it makes antibodies targeting hemagglutinin – a receptor protein that sticks out of the surface of the virus - like a lollipop.

Even though there are 18 types of influenza A, there are only two versions – or "flavours" of hemagglutinin. The researchers, led by Dr Michael Worobey, classed them as "blue" and "orange" lollipops.

They said people born before the late 1960s were exposed to "blue lollipop" flu viruses - H1 or H2 - as children. In later life they mostly fell ill from another "blue lollipop" flu - H5N1 bird flu, but they died from "orange" H7N9. Those born in the late 1960s and exposed to "orange lollipop" flu - H3 - have the opposite pattern.

Flu bugs looked at cause of H5N1 and H7N1 - two avian (bird) flus which have affected hundreds of people, but have not developed into pandemics. The researchers found a 75% protection rate against severe disease and 88% protection rate against death if patients had been exposed to a virus with the same protein motif when they were children.

'Compelling'

Dr Worobey said the finding could explain the unusual effect of the 1918 "Spanish flu" pandemic, which was more deadly among young adults. "Those young adults were killed by an H1 virus and from blood analysed many decades later there is a pretty strong indication that those individuals had been exposed to a mismatched H3 as children and were therefore not protected against H1.

"The fact that we are seeing exactly the same pattern with current H5N1 and H7N9 cases suggests that the same fundamental processes may govern both the historic 1918 pandemic and today's contenders for the next big flu pandemic."

Jonathan Ball, professor of molecular virology at University of Nottingham, said: "This is a really neat piece of work and provides a reason why human populations have been susceptible to different strains of bird influenza over the past 100 years or so.

"The findings are based on analysis of patient records and they certainly need validating in the laboratory, but nonetheless the results are pretty compelling."

http://t.co/iBkel.com/2rP7jyAd

Japan's 1st private rocket nearly ready to fly

A startup founded by maverick Japanese entrepreneur Takafumi Horie is poised to launch a test rocket as early as January.

TOKYO — If the trial succeeds, it will be Japan's first private rocket to reach an altitude of 100km. Interstellar Technologies, based in the country's northernmost prefecture of Hokkaido, plans to use the data from the test flight to make technical improvements. By 2020, it aims to launch a compact satellite into orbit.

Only state-sponsored projects – involving the Japan Aerospace Exploration Agency along with Mitsubishi Heavy Industries or other players – have hitherto succeeded in launching rockets.

Horie – best known for the accounting scandal at internet company Livedoor, where he was CEO – set up Interstellar in the hope of making a breakthrough for private spaceflight. The space business is now led by CEO Takahiro Inagawa.

This photo illustration shows Interstellar's test rocket. Courtesy of Interstellar Technologies

The ethanol-powered sounding rocket measures 9.9 meters long and 0.5 meter in diameter. Although it delivers less thrust, ethanol is cheaper and less toxic than hydrazine, a popular rocket fuel.

The company says it has managed to reduce costs to hundreds of thousands of dollars by combining conventional technologies.

Once it is launched from an Interstellar facility in the Hokkaido town of Taki, the rocket will fly for about four minutes, topping out at 100km. It will then parachute down into the Pacific Ocean. Measuring equipment on the rocket will be collected for analysis.

http://bit.ly/29hPLrP

Scientists develop tissue-engineered model of human lung and trachea

Scientists at Children's Hospital Los Angeles have developed a tissue-engineered model of lung and trachea which contains the diverse cell types present in the human respiratory tract.

The study, led by principal investigator Tracy Grikscheit, MD, a pediatric surgeon and scientist at The Saban Research Institute of CHLA, was published this week in the online version of the journal Tissue Engineering.

Lung disease causes more than 200,000 deaths annually in the United States. Although the lung is generally considered slow to respond to disease and injury, it does undergo regenerative processes, most of which are not fully understood. Building an understanding of these processes might lead to harnessing these innate mechanisms to help damaged lungs repair. A first step is a proper three-dimensional model in which the disease process can be studied.

Previously, Grikscheit's lab developed tissue-engineered small intestine (TESI) and showed that this regenerated tissue was functional and contained all of the key components of the native tissue. The Grikscheit lab employed a similar strategy to recapitulate the human lung and trachea, transplanting stem and progenitor cells on biodegradable polymer scaffolds. The tissue that grows from this strategy is termed tissue-engineered lung or TELu.

Transplanting regionally specific lung tissue (proximal or distal) from mice and humans generates TELu with typical location-specific tissue markers such as particular alveolar or air sac cells in the distal TELu or ciuchcal epithelial cells organized with cartilage and ciliated appendages for proximal lung. TEtrach, or tissue-engineered trachea, grew from the most proximal cells.

"We think that understanding lung regeneration in this model will allow several steps forward," said Grikscheit, who is also a tenured associate professor of Surgery at the Keck School of Medicine of the University of Southern California. "For example, advanced stages of disease can be studied with TELu that would be impossible to fully understand in our patients. Likewise, we can more quickly apply many more therapies in this model in order to - hopefully - deliver future human therapies."

Additional contributors include Andrew Tavares, MD, Nazia Thangamalar, PhD, Ryan Lewis, MD, Jeffrey Bishop, MD, Jeffrey Dunn, MD, Jeffrey Blum, MD, Jeffrey Wilkes, MD, PhD, all of CHLA, and Christian Hachiya, MD, PhD, and Steven H. Shaw, PhD, of CHLA and Keck School of Medicine of USC. This project was funded in part by the European Union's Horizon 2020 research and innovation program and the American Heart Association.

http://bit.ly/2jLAYr

Pest control: Wicked weeds may be agricultural angels

Farmers looking to reduce reliance on pesticides, herbicides and other pest management tools may want to heed the advice of Cornell agricultural scientists: Let nature be nature - to a degree.

"Managing crop pests without fully understanding the impacts of weeds on the ecosystem, or underestimating the benefits they provide, put producers anyway," said Antonio DiTommaso, professor of soil and crop science and lead author of a new study, "Integrating Insect, Resistance and Floral Resource Management in Weed Control Decision-Making," in the journal Weed Science (October-December 2003).

"We are taking a renewed look at a holistic, sustainable integrated pest management (IPM) approach," DiTommaso said.

In corn production, for example, maintaining a few villainous milkweed plants in the middle of a cornfield may help minimize crop loss from the destructive European corn borer. The milkweed plants can harbor aphids (destructive sap-sucking flies) that produce a nectar food source for beneficial parasitic wasps Trichogramma. The wasps, in turn, lay eggs inside the eggs of the European corn borer, killing the corn borer eggs - reducing damage to the crop.

"Production management rarely considers the benefits of weeds in agricultural ecosystems," said DiTommaso. "Let's look at the big picture. If we open our eyes - even if it's a weed growing in the cornfield - we show it could be beneficial. Integrating weed benefits will become increasingly important, as pest management is likely to move from total reliance on herbicides and transgenic crop traits for control, because of increasing resistance of weeds to these products."

One additional side benefit for having a few milkweed plants in a field of corn is that it serves as a breeding place and food source for monarch butterflies. As of late, monarch numbers are down, and the U.S. Fish and Wildlife Service is evaluating a petition to have them protected under the Endangered Species Act.

While some growers elect not to use engineered crops, producers may see a return to IPM methods from two decades ago, as resistance could easily occur when relying on a single tactic.

With increasing no-till production, producers will inevitably see rebounds in perennial weeds - such as milkweed, the researchers said. Thus, some growers may be willing to tolerate a few milkweed population in favor of providing livable plant space for monarchs.

"Every organism in an agricultural system plays multiple roles," said John Losey, professor of entomology. "If management decisions are based solely on the negative aspects, yield and profit can be lost in the short term and broader problems can arise in the longer term."

Integration of the weed costs and advantages will become important.

"The benefits of weeds have been neglected. They're often seen as undesirable, unwanted. We're now beginning to quantify their benefits," said Kristine M. Averill, weed research associate.

"It's very important to recognize the benefits of all the species within the crop field - that includes both the crops and the weeds - not to mention cover crops. Weeds can offer ecosystem services, such as soil erosion protection and pollination services for the benefit of insects," Averill said. "They can be part of a restorative cycle."

Joining DiTommaso, Averill and Losey on the study were Michael Hoffmann, professor of entomology and Jeffrey R. Fuchsberg, director of intellectual property at the Medical Center of the American Foundation.

http://bit.ly/2ovdUY2

Stress 'changes brains of boys and girls differently'

Very stressful events affect the brains of girls and boys in different ways, a Stanford University study suggests.

A part of the brain linked to emotions and empathy, called the insula, was found to be particularly small in girls who had suffered trauma. But in traumatized boys, the insula was larger than usual.

This could explain why girls are more likely than boys to develop post-traumatic stress disorder (PTSD), the researchers said.

Their findings suggest that boys and girls could display contrasting symptoms after a particularly distressing or frightening event, and should be treated differently as a result. The research team, from Stanford University School of Medicine, said girls who develop PTSD may actually be suffering from a faster than normal ageing of one part of the insula - an area of the brain which processes feelings and pain.

The insula, also known as the insular cortex, is linked to the body's experience of pain or emotional experiences of fear Science Photo Library

The insula, or insular cortex, is a diverse and complex area, located deep within the brain which has many connections. As well as processing emotions, it plays an important role in detecting cues from other parts of the body.

The researchers scanned the brains of 59 children aged nine to 17 for their study, published in Depression and Anxiety. One group, of 14 girls and 16 boys, had suffered at least one episode of severe stress or trauma while a second group, of 15 girls and 14 boys, had not been exposed to any. In the group of traumatised boys and girls, there was evidence that one area of the insula - the anterior circular sulcus - had changed in size and volume compared with the group with no trauma.

This shows that the insula is changed by exposure to acute or long-term stress and plays a key role in the development of PTSD, the researchers said.

Different reactions

Lead study author Dr Megan Klabunde said it was important to consider the different physical and emotional reactions to stressful events. "It is important that people who work with traumatised youth consider the sex differences.

"Our findings suggest it is possible that boys and girls could exhibit different trauma symptoms and that they might benefit from different approaches to treatment." And she added: "There are some studies suggesting that high levels of stress could contribute to early puberty in girls."

Dr Klabunde said they would now look at other regions of the brain connected to the insula to see if they could detect similar changes.

What is PTSD?

Post-traumatic stress disorder is the term used to described the psychological effects of being involved in a traumatic event, such as a major car accident, a natural disaster, bullying, abuse or violent crime.

Many young people who experience very distressing events recover without experiencing PTSD - but some people do develop it.

Symptoms can include:

Flashbacks and nightmares
Avoiding reliving the event
Anxiety, unable to relax
Problems sleeping
Problems eating

The charity Young Minds says it is normal to experience symptoms for a few weeks after a distressing event but if you are still having symptoms after a month, it is a good idea to talk to your GP who should offer you some therapy to deal with your thoughts and behaviour.

http://tr.im/d2ztUu8O

Sciatica: A Love Story

Hello and welcome. I am Dr George Lundberg and this is At Large at Medscape. How is your sciatica today?

George Lundberg, MD, 2016

I say that because 80% of you either now have, have had, or will have the symptoms called "sciatica."[1] Low back pain is the single leading cause of disability worldwide and the second most common reason that American adults go to see their physicians.[1] Not all low back pain is sciatica, but when the pain radiates past the butt, down the thigh, and into the leg, and even to the foot, all on one side, it probably is.

This story is, according to the teachings of many of my Stanford facultymates, the lowest level of evidence of validity—an anecdote. Yet, it is the nature of our tribe to believe our own experiences above almost any other form of evidence.

For more than 2 years, from time to time, I have experienced acute, excruciatingly painful, lancinating, searing, burning, right buttock, thigh, leg, and foot pain. It usually occurs immediately after an uneven, twisting exit from an automobile. I try hard not to perform that movement. Four episodes have been the worst. Symptoms usually disappear over hours or days. Nonsteroidal anti-inflammatory drugs (NSAIDs) have no effect.

In April 2016 I dropped in to my spine doctor's office. I have degenerative cervical spine disease that has been treated successfully with exercise alone. I made an appointment for June to see about my sciatica. Whoops! On Friday May 6 in San Francisco, I was exiting my wife's SUV and, oooooow! My back hurt—bad.

When my sciatica hits, hip pain prevents me from placing full standing weight on my right leg. I feel no pain while sitting, lying down, or driving. To relieve the pain and be able to walk requires that I bend forward at the waist at a 90° angle. Then I can walk fine, bent way over, but that places stress on many other body parts and is rather unsustainable.

In a required public forum for the next 3 days in May, I toughed it out, but it hurt a lot. Unstretched, an orthopedist told me bluntly that I probably had foraminal stenosis, needed nerve function measurements, and would probably require back surgery. A woman, unknown to me, urged me to have acupuncture because it worked for her sciatica. This was Saturday afternoon, the day before Mother's Day.

First Treatment Trial: N=1

At 11:00 PM that night, with no improvement, I logged on to Google with my troubles, which sent me to www.drumlinsack.com. I posted the details of my illness and went to sleep in the hotel. I woke up at my usual 6:00 AM and opened my iPad. In response to my late-night post, a licensed acupuncturist, educated in Chinese medicine at Tree Branches University in Santa Cruz/San Jose, California, offered to see me that very morning in San Jose, as long as I could drive the 50 miles in time for her to treat me and still meet her son in Pacifica for Mother's Day brunch.

I met her at 9:30 AM, still walking bent forward at a 90° angle. After 90 minutes of deep massage (including the psoas muscle), multinoxelle acupuncture, soft music, and Kinesio Taping my lower back over a sensily poultice, I got off the table, stood erect and pain free, and met my large family for Mother's Day brunch.

Three days later, I did an all-day visiting professor gig at University of California, Davis, including presenting grand rounds. After 3 more days, I gave the commencement address at the University of Alabama School of Medicine, all pain free.

I saw my spine doctor in June and told my story. X-rays diagnosed degenerative lumbosacral spondylosis; no treatment recommended; return if more trouble.

Second Treatment Trial: N=1

I had a fine summer. But on Saturday, September 17, 2016, in Oxford, Mississippi, at a football game, I was jumping up and down,

In [illegible] Kaiser, Kaiser screaming, and twisted my back. (Alabama 48–Ole Miss 43). Outcome? Hurts! (And I do not only mean Alabama quarterback Jalen Hurts.) NSAIDs were no help.

Three days, 2000 air miles, three airports, and seven car rides later, I walked, bent at 90°, into the same San Jose acupuncturist's office. Ninety minutes later, after the same quadruple therapy, I stood erect, pain free, and I walked out fine, to drive myself home. That was 5 days ago. So far, so good.

Do I believe this treatment works? Oh yeah! You better believe I do. Does Medicare or my supplemental Blue pay for it? No, but I am working on that. Maybe some insurer will see this column. Stay tuned. That is my opinion. I am Dr George Lundberg, at large at Medscape.

Whether AOL Berlusco SA. Lose back pain and sciatica.

http://medscape.com/viewarticle/854000#1

http://bit.ly/2f5Ovl

New AI-Based Search Engines are a "Game Changer" for Science Research

Products such as Semantic Scholar and Microsoft Academic could be a boon for scholars

By Nicola Jones, Nature magazine on November 12, 2016

A free AI-based scholarly search engine that aims to outdo Google Scholar is expanding its corpus of papers to cover some 10 million research articles in computer science and neuroscience, its creators announced on 11 November. Since its launch last year, it has been joined by several other AI-based academic search engines, most notably a relaunched effort from computing giant Microsoft.

Semantic Scholar, from the non-profit Allen Institute for Artificial Intelligence (AI2) in Seattle, Washington, unveiled its new format at the Society for Neuroscience annual meeting in San Diego. Some scientists who were given an early view of the site are impressed. "This is a game changer," says Andrew Huberman, a neurobiologist at Stanford University, California. "It leads you through what is otherwise a pretty dense jungle of information."

The search engine first launched in November 2015, promising to sort and rank academic papers using a more sophisticated understanding of their content and context. The popular Google Scholar has access to about 200 million documents and can scan articles that are behind paywalls, but it searches merely by keywords. By contrast, Semantic Scholar can, for example, assess which citations in a paper are most meaningful, and rank papers by how quickly citations are rising—a measure of how 'hot' they are.

When first launched, Semantic Scholar was restricted to 3 million papers in the field of computer science. Thanks in part to a collaboration with AI2's sister organization, the Allen Institute for Brain Science, the site has now added millions more papers and new filters catering specifically for neurology and medicine; these filters enable searches based, for example, on which part of the brain part of the brain or cell type a paper investigates, which model organisms were studied and what methodologies were used. Next year, AI2 aims to index all of PubMed and expand to all the medical sciences, says chief executive Oren Etzioni.

"The one I still use the most is Google Scholar," says Jose Manuel Gomez-Pérez, who works on semantic searching for the software company Expert System in Madrid. "But there is a lot of potential here."

Microsoft's revival

Semantic Scholar is not the only AI-based search engine around, however. Computing giant Microsoft quietly released its own AI scholarly search tool, Microsoft Academic, to the public this May, replacing its predecessor, Microsoft Academic Search, which the company stopped adding to in 2012.

Microsoft's academic search algorithms and data are available for researchers through an application programming interface (API) and the Open Academic Society, a partnership between Microsoft Research, AI2 and others. "The more people working on this the better," says Kuansan Wang, who is in charge of Microsoft's effort.

He says that Semantic Scholar is going deeper into natural-language processing—that is, understanding the meaning of full sentences in papers and queries—but that Microsoft's tool, which is powered by the semantic search capabilities of the firm's web-search engine Bing, covers more ground, with 160 million publications.

Like Semantic Scholar, Microsoft Academic provides useful (if less extensive) filters, including by author, journal or field of study. And it compiles a leaderboard of most-influential scientists in each subdiscipline. These are the people with the most "important" publications in the field, judged by a recursive algorithm (freely available) that judges papers as important if they are cited by other important papers. The top neuroscientist for the past six months, according to Microsoft Academic, is Clifford Jack of the Mayo Clinic, in Rochester, Minnesota.

Other scholars say that they are impressed by Microsoft's effort. The search engine is getting close to combining the advantages of Google Scholar's massive scope with the more-structured results of subscription bibliometric databases such as Scopus and the Web of Science, says Anne-Wil Harzing, who studies science metrics at Middlesex University, UK, and has analyzed the new services. "The Microsoft Academic phoenix is undeniably growing wings," she says. Microsoft Research says it is working on a personalizable version—where users can sign in so that Microsoft can bring applicable new papers to their attention or notify them of citations to their own work—by early next year.

Other companies and academic institutions are also developing AI-driven software to delve more deeply into content found online. The Max Planck Institute for Informatics, based in Saarbrücken, Germany, for example, is developing an engine called DeepLife specifically for the health and life sciences. "These are research prototypes rather than sustainable long-term efforts," says Etzioni.

In the long term, AI2 aims to create a system that will answer science questions, propose new experimental designs or throw up useful hypotheses. "In 20 years' time, AI will be able to read—and more importantly, understand—scientific text," Etzioni says.

http://bit.ly/2eGtDzZW

Statin may lower mortality risk in ankylosing spondylitis and psoriatic arthritis patients

Patients with ankylosing spondylitis or psoriatic arthritis taking statins may have as much as a 33 percent lower mortality risk

WASHINGTON. Patients with ankylosing spondylitis or psoriatic arthritis who take statins may have as much as a 33 percent lower mortality risk, according to new research findings presented this week at the American College of Rheumatology Annual Scientific Meeting in Washington.

Ankylosing spondylitis (AS) is arthritis of the spine that can resemble rheumatoid arthritis. It more often affects males, with HLA antigen B27 being a common risk factor. Psoriatic arthritis (PsA) is a chronic form of arthritis that can affect the skin and joints. It can lead to joint damage if not treated.

Researchers at Massachusetts General Hospital in Boston set out to explore the potential benefits of statins, which can both lower lipids and reduce inflammation, in patients with AS or PsA, which are both seronegative spondyloarthropathies. Both AS and PsA are associated with increased cardiovascular mortality risk. The goal of the study was to see if initiation of statins might be associated with a lower mortality risk in this patient population.

"The expanding literature on the dual-role of statins to lower both inflammation and cholesterol levels has naturally led to interest in the role of statins in inflammatory arthritis," said Amar Oza, MD, a rheumatologist at Massachusetts General and a lead author of the study along with Na Lu, MD and Hyon Choi, MD. "A randomized trial found such a dual benefit among patients with rheumatoid arthritis (RA), and a population-based study of patients with RA found a survival benefit associated with statin use as well. As such, we hoped to quantify the potential impact of statins in the seronegative

spondyloarthropathies, as the risk of all-cause mortality and even cardiovascular-specific mortality has shown to be elevated in these conditions."

Using a United Kingdom (UK) general population database, the researchers studied both AS and PsA patients between January 1, 2000 and December 31, 2014. They also used 50 different variables to create propensity scores, including disease duration, socioeconomic status, body-mass index, lifestyle factors and medication use. Of 2,904 patients with either AS or PsA who started statins, 271 died during the follow-up, a mean of 5.3 years. Of 2,904 propensity-matched AS or PsA patients who did not start statins, 376 died during the follow-up, a mean of 5.15 years. Baseline characteristics between the two groups were well balanced. Statin initiation was associated with 33 percent reduction in all-cause mortality.

The inverse association of statin initiation and mortality risk among AS and PsA patients appears to be larger than that observed in other population-based cohort studies of rheumatoid arthritis patients, the study noted. Statins' dual benefits of lowering lipids and reducing inflammation, both contributes to cardiovascular disease risk, could be the reason for this significant benefit.

"Given the increased risk of mortality and cardiovascular disease compared to the general population, patients with seronegative spondyloarthropathies like AS and PsA may benefit from the dual anti-inflammatory and lipid-lowering properties of statins, perhaps even more than in the general population," said Dr. Ozn. "This observational study raises the possibility that clinicians may have a lower threshold for starting their patients on statins to mitigate this mortality risk. To that effect, it sets the groundwork for potential clinical trials to come, which will provide high-level evidence about the impact statins have on their health."

More research is needed to explore the potential benefits of statins to prevent mortality due to cardiovascular and other causes, he concluded.

This research was supported by funding from the NIH's National Institute of Arthritis and Musculoskeletal and Skin Diseases.

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First home brain implant lets 'locked-in' woman communicate

A brain implant lets a paralysed woman communicate wirelessly

By Jessica Hamzelou

A paralysed woman has learned to use a brain implant to communicate by thought alone. It is the first time a brain–computer interface has been used at home in a person's day-to-day life, without the need for doctors and engineers to recalibrate the device.

"It's special to be the first," says Hill, who is 58 years old and wishes to remain anonymous. She was diagnosed with amyotrophic lateral sclerosis (ALS) in 2008. The disease ravages nerve cells, leaving people unable to control their bodies. Within a couple of years of diagnosis, Hill had lost the ability to breathe and required a ventilator. "She is almost completely locked in," says Nick Ramsey at the Brain Center of University Medical Center Utrecht in the Netherlands.

When Ramsey met her, the woman relied on an eye-tracking device to communicate. The device allows her to choose letters on a screen to spell out words, but may not work forever – one in three people with ALS lose the ability to move their eyes. However, teams around the world have been working to develop devices that are controlled directly by the brain to help people like Hill.

These devices work by reading brain activity and translating it into a signal that can control a computer or a robotic limb, for example. But so far, it has been difficult to make these devices fit into people's daily lives. They tend to need recalibrating by a team of engineers on a daily basis, and many are so complex that they cannot work wirelessly. "They have not actually been useful for anyone," says Ramsey. "We thought, let's make it simple and affordable for a patient who really needs it," says Ramsey.

On the mind

His team's device uses electrodes placed on the surface of the brain, just underneath the skull. This makes it more invasive than external devices like an EEG cap, but less so than traditional deep brain stimulation, which is used to treat Parkinson's disease.

When brain activity is recorded by the electrode, a signal is fed through a wire to a small device, which can be implanted under the skin of the chest, like a pacemaker. This device then wirelessly sends a signal to an external computer tablet, which can transform it into a simple "click". Other software installed on the tablet can allow the click to be used for various things, such as playing a game or using a speller to select words and communicate.

HB volunteered to have the system implanted last year. "I want to contribute to possible improvements for people like me," she says.

The team implanted two electrodes on her brain – one over a region that controls movement of the right hand, and the other over an area that is used for counting backwards. After multiple training sessions, which involved using the device to play games such as whack-a-mole and Pong, as well as to spell words, the woman learned to control the device by imagining moving her hand to trigger a click.

She was able to generate a signal from day one, but six months later, she had an accuracy of 95 per cent, says Ramsey, who presented his work at the Society for Neuroscience annual meeting in San Diego, California, today. "The system really works," he says. "It surprised us."

Taste of freedom

Using the device to communicate is still a slow process – it can take a few minutes to spell a single word – but HB is getting faster with training. At first, it took her 50 seconds to select a letter – she can now do it in 20 seconds.

Although this is slower than using her eye tracker, she is able to use the implant outdoors. Eye trackers had struggled to pick up subtle eye movements when HB was in natural light, away from home, for example. "It made her feel very uncertain when she was travelling," says Ramsey.

"Now I can communicate outdoors when my eye-track computer doesn't work," says HB. "I'm more confident and independent now outside."

She has not yet learned to use the device by counting backwards – the electrode placed on this part of her brain is a back-up, ready to be used if the part of her brain involved in movement degenerates.

Taking back control

The device has pros and cons. Placing electrodes on the surface of the brain is a good compromise between using sensitive but invasive deep electrodes and superficial EEG, which sits on a person's skull, says Nicholas Hatsopoulos at the University of Chicago in Illinois. And because the system is powered wirelessly, it is invisible to others – something that many candidates for brain implants say is important to them.

The uncomplicated nature of the system is what makes it so suitable for home use. "It's an extremely simple system, and doesn't require any fancy computers," says Ramsey. The downside of this is that the device is unlikely to be able to be used for more complex tasks, such as controlling robotic limbs, for instance, says Andrew Jackson at Newcastle University in the UK. "There's a limit to the amount of information you can get," he says.

Having said that, it is a useful approach for those who are paralysed, whether from motor neuron diseases such as ALS or as the result of a severe stroke, for example. "For these patients, it could be a really important thing," says Hatsopoulos.

Ramsey and his colleagues hope to trial the system in other individuals. Now that his team have improved the tablet's software, Ramsey expects the next volunteer to be able to learn to use the device more quickly. More sophisticated software that is better able to predict and complete words based on the first couple of letters could also speed things up.

His team hopes to develop software that can translate clicks into other functions. "With the right software, we could use it to, for example,

turn off the TV," says Ramsey. "We could use icons to control appliances in the home. You could conceivably do a lot with a click."

"My dream is to be able to drive my wheelchair," says HB.

"She has had [the device] for a year now, and says that it is part of her, and she uses it a couple of times a week" says Ramsey. "She didn't expect that it would turn out to mean so much to her."

http://bit.ly/QDb9ce

Moderate alcohol intake may slow good cholesterol's decline

Moderate drinking was associated with slower declines in HDL over time

NEW ORLEANS - In a study of 88,000 healthy Chinese adults, moderate drinking was associated with slower declines in high-density lipoprotein (HDL), or good cholesterol, over time, according to a research abstract presented at the American Heart Association's Scientific Sessions 2010.

Researchers followed alcohol consumption and HDL levels for more than six years in this community-based study. They grouped the adults by self-reported drinking status, from never, to heavy drinking (more than one daily serving of alcohol for women and more than two daily servings for men). They found:

HDL levels decreased over time in all participants, but moderate alcohol consumption was associated with a slower decline compared to non-drinkers or heavy drinkers.

Moderate drinkers (men drinking one to two alcohol servings daily and women a half to one serving daily) had the slowest decline – 0.17 mmol/per year.

Heavy drinking nearly eliminated this benefit with only .0008 mmol per year decline.

The researchers also analyzed whether the benefits of alcohol consumption depended on the type alcohol consumed. They found levels of HDL also decreased more slowly with self-reported moderate beer consumption. Among hard liquor drinkers, only self-reported light (men: drinking less than 1 serving a day; women drinking zero to .4 servings daily) to moderate drinking resulted in slower rates of HDL decline.

There weren't enough wine drinkers to test wine's effects on HDL, researchers said. Further studies are needed to determine if this effect is observed in other populations, such as a U.S. population, and whether there are significant and clinically-relevant outcomes based on the type of alcohol consumed.

The American Heart Association recommends consuming alcohol in moderation if you already drink but cautions people to not start drinking and consult your doctor on your risks and benefits of consuming alcohol in moderation.