http://www.eurekalert.org/pub_releases/2014-11/gumc-tna111614.php

Tau, not amyloid-beta, triggers neuronal death process in Alzheimer's

New research points to malfunctioning tau, not amyloid-beta (Abeta) plaque, as the seminal event that spurs neuron death in disorders such as Alzheimer's disease.

WASHINGTON - The lead Georgetown neuroscientist investigating the work explains the finding and the potential of an already approved drug in mediating the problem at the annual meeting of the Society for Neuroscience, Tuesday, Nov. 18, 8:15 a.m. in room WCC152A. The study, which dramatically alters the prevailing theory of Alzheimer's development, also explains why some people with plaque build-up in their brains don't have dementia. The work was describe earlier this month in the journal Molecular Neurodegeneration.

Neuronal death happens when tau, found inside neurons, fails to function. Tau's role is to provide a structure -- like a train track --inside brain neurons that allows the cells to clear accumulation of unwanted and toxic proteins.

"When tau is abnormal, these proteins, which include Abeta, accumulate inside the neurons," explains the study's senior investigator, Charbel E-H Moussa, MB, PhD, assistant professor of neuroscience at Georgetown University Medical Center. "The cells start to spit the proteins out, as best they can, into the extracellular space so that they cannot exert their toxic effects inside the cell. Because Abeta is 'sticky,' it clumps together into plaque," Moussa says.

He says his study suggests the remaining Abeta inside the neuron (that isn't pushed out) destroys the cells, not the plaques that build up outside. "When tau does not function, the cell cannot remove the garbage, which at that point includes Abeta as well as tangles of nonfunctioning tau, and the cell dies. The Abeta released from the dead neuron then sticks to the plaque that had been forming." Moussa's experiments in animal models also show less plaques accumulate outside the cell when tau is functioning; when tau was reintroduced into neurons that did not have it, plaques did not grow.

Malfunctioning tau can occur due to errant genes or through aging. As individuals grow older, some tau can malfunction while enough normal tau remains to help clear the garbage. In these cases, the neurons don't die, he says. "That explains the confusing clinical observations of older people who have plaque build-up, but no dementia," Moussa says. Moussa has long sought a way to force neurons to clean up their garbage. In this study, he shows that nilotinib, a drug approved to treat cancer, can aid in that process. Nilotinib helps the neuron clear garbage, but requires some functional tau, he says.

"This drug can work if there is a higher percentage of good to bad tau in the cell," Moussa says. "There are many diseases of dementia that have malfunctioning tau and no plaque accumulation, such as frontal temporal dementia linked to Parkinsonism," Moussa says. "The common culprit is tau, so a drug that helps tau do its job may help protect against progression of these diseases."

Co-authors include researchers from Capital Medical University in Beijing, China, and Merck Research Laboratories.

Funding for these studies was provided by Georgetown University grants and by Merck & Co. Moussa is an inventor on a Georgetown University patent application for use of nilotinib as a therapeutic approach in neurodegenerative diseases.

http://www.eurekalert.org/pub_releases/2014-11/msu-ofl111714.php

One firm's loss is another's gain

Good news for savvy businesses: Customers who walk through your doors unhappy with another firm's service can be won back with simple gestures of goodwill.

EAST LANSING, Mich. - Consider a dissatisfied airline passenger. A hotel can score loyalty points by providing the traveler a room upgrade or perhaps even a simple apology for the airline's failure, said Clay Voorhees, associate professor of marketing at Michigan State University.

In a study published online in the Journal of the Academy of Marketing Science, Voorhees and fellow researchers refute past findings that a bad service or retail experience taints a consumer for the entire day. The new paper is titled "One firm's loss is another's gain: capitalizing on other firms' service failures."

"We found that if you offer these goodwill gestures, you not only negate the negative feelings in the customer, you actually get a lift in attitude toward your firm," said Voorhees.

To test the theory, the researchers conducted three experiments dealing with the airline, hotel and restaurant industries. More than 500 people participated in all. When the firm responsible for the bad service made a goodwill gesture, it actually had no effect on the customer's negative attitude, the study found. When a firm affiliated with the offending company made the attempt, the customer's attitude improved only slightly.

But when a completely unaffiliated company made the goodwill gesture after the negative experience, the customer's attitude toward that unaffiliated company improved significantly.

Voorhees said the findings underscore the importance of training frontline workers to react to customer complaints regarding other firms. Most companies don't provide this type of training to their frontline workers, who are often their lowest paid.

The study also suggests firms should investigate their entire service chain to identify possible weak spots. Insurance providers, for example, could potentially leverage breakdowns in the automobile-buying process.

Firms should also be careful about who they choose as affiliates. Partnering with companies prone to failure might not be worth the additional business volume, Voorhees said.

Voorhees' co-researchers are Alexis Allen from the University of Kentucky, Michael Brady from Florida State University and Stacey Robinson from East Carolina University.

http://phys.org/news/2014-11-right-to-carry-gun-laws-linked-violent.html

Right-to-carry gun laws linked to increase in violent crime, research shows

New Stanford research confirms that right-to-carry gun laws are linked to an increase in violent crime.

Right-to-carry or concealed-carry laws have generated much debate in the past two decades – do they make society safer or more dangerous?

While there is no federal law on concealed-carry permits, all 50 states have passed laws allowing citizens to carry certain concealed firearms in public, either without a permit or after obtaining a permit from local government or law enforcement.

Recently published scholarship updates the empirical evidence on this issue.

Stanford law Professor John J. Donohue III, Stanford law student Abhay Aneja and doctoral student Alexandria Zhang from Johns Hopkins University were the co-authors of the study.

"Trying to estimate the impact of right-to-carry laws has been a vexing task over the last two decades," said Donohue, the C. Wendell and Edith M. Carlsmith Professor of Law, in an interview.

He explained that prior research based on data through 1992 indicated that the laws decreased violent crime. But in 2004, he noted, the National Research Council issued a report that found that even extending this data through 2000 revealed no credible statistical evidence these particular laws reduced crime.

'Totality of the evidence'

Now, Donohue and his colleagues have shown that extending the data yet another decade (1999-2010) provides the most convincing evidence to date that right-to-carry laws are associated with an increase in violent crime. "The totality of the evidence based on educated judgments about the best statistical models suggests that right-to-carry laws are associated with substantially higher rates" of aggravated assault, rape, robbery and murder, said Donohue.

The strongest evidence was for aggravated assault, with data suggesting that right-to-carry (RTC) laws increase this crime by an estimated 8 percent – and this may actually be understated, according to the researchers.

"Our analysis of the year-by-year impact of RTC laws also suggests that RTC laws increase aggravated assaults," they wrote. The evidence is less strong on rape and robbery, Donohue noted. The data from 1979 to 2010 provide evidence that the laws are associated with an increase in rape and robbery.

The murder rate increased in the states with existing right-to-carry laws for the period 1999-2010 when the "confounding influence" of the crack cocaine epidemic is controlled for. The study found that homicides increased in eight states that adopted right-to-carry laws during 1999-2010.

Research obstacles, next step

"Different statistical models can yield different estimated effects, and our ability to ascertain the best model is imperfect," Donohue said, describing this as the most surprising aspect of the study.

He said that many scholars struggle with the issue of methodology in researching the effects of right-to-carry laws. But overall, his study benefits from the recent data. Donohue suggested it is worth exploring other methodological approaches as well. "Sensitive results and anomalies – such as the occasional estimates that right-to-carry laws lead to higher rates of property crime – have plagued this inquiry for over a decade," he said.

More information: *Aneja, Abhay and Donohue, John J. and Zhang, Alexandria, "The Impact of Right to Carry Laws and the NRC Report: The Latest Lessons for the Empirical Evaluation of Law and Policy (September 4, 2014)." Stanford Law and Economics Olin Working Paper No. 461. Available at SSRN: ssrn.com/abstract=2443681 or dx.doi.org/10.2139/ssrn.2443681*

http://www.eurekalert.org/pub_releases/2014-11/uow-mbp111314.php

Major brain pathway rediscovered after century-old confusion, controversy

A couple of years ago a scientist looking at dozens of MRI scans of human brains noticed something surprising.

A large, fiber pathway that seemed to be part of the network of connections that process visual information showed up on the scans, but the researcher couldn't find it mentioned in any of the modern-day anatomy textbooks he had.

"It was this massive bundle of fibers, visible in every brain I examined," said Jason Yeatman, a research scientist at the University of Washington's Institute for Learning & Brain Sciences. "It seemed unlikely that I was the first to have noticed this structure; however, as far as I could tell, it was absent from the literature and from all major neuroanatomy textbooks."

With colleagues at Stanford University, where he was a graduate student at the time, Yeatman started some detective work to figure out the identity of that large, mysterious fiber bundle.

In the paper, to be published Nov. 17 by the Proceedings of the National Academy of Sciences, the team describes the history and controversy of the elusive brain pathway, explains how modern MRI techniques rediscovered it, and gives analytical tools researchers can use to identify the brain structure - now known as the vertical occipital fasciculus.

The "aha moment" in identifying the pathway came while Yeatman and Kevin Weiner, a Stanford postdoctoral researcher, were poring over the yellowed pages of 19th-century brain atlases in the basement of the Stanford Medical Library.

"Kevin found an atlas, written by Carl Wernicke near the turn of the (20th) century, that depicted the vertical occipital fasciculus," Yeatman said. "The last time that atlas had been checked out was 1912, meaning we were the first to view these images in the last century."

From there, Yeatman and Weiner, who share lead authorship on the paper, did more library research revealing these possibilities for why the pathway was forgotten:

- A scientific disagreement. In an 1881 neuroanatomy atlas, Wernicke, a well-known anatomist who in 1874 discovered "Wernicke's area," which is essential for language, wrote about a fiber pathway in a monkey brain he was examining. He called it "senkrechte Occiptalbündel" (translated as vertical occipital bundle). But its vertical orientation contradicted the belief of one of the most renowned neuroanatomists of the era, Theodor Meynert, who asserted that brain connections could only travel in between the front and the back of the brain, not up and down.

- Haphazard naming methods. The 1880s and 1890s were a fertile time in the neuroanatomy world, but scientists lacked a shared process for naming the brain structures they found. Looking at drawings of the brain from this time period, Yeatman and coauthors saw that the fiber pathway that they were looking for appeared in brain atlases but was called different things, including "Wernicke's perpendicular fasciculus," "perpendicular occipital fasciculus of Wernicke," and "stratum profundum convexitatis."

"When we started, it was just for our own knowledge and curiosity," said Weiner, who's also the director of public information at the Institute for Applied Neuroscience, a nonprofit based in Palo Alto, California.

"But, after a while, we realized that there was an important story to tell that contained a series of missing links that have been buried for so long within this puzzle of historical conversation among many who are considered the founders of the entire neuroscience field."

The researchers used a type of MRI measure called diffusion-weighted imaging to measure the size of the pathway and see where in the brain it went. Across brain scans taken from 37 subjects, they found that the vertical occipital fasciculus begins in the occipital lobe - the part of the brain's visual processing system located at the back of the head. From there, the fibers spread out like a sheet, connecting brain regions that are important for seeing objects with other brain regions that coordinate which objects to focus attention upon.

"We believe that signals carried by the VOF play a role in many perceptual processes, from recognizing a friend's face to rapidly reading a page of text," said Yeatman, who is now studying brain mechanisms involved in learning to read.

In the paper, the researchers also provide an algorithm that others can use on their own data to find the pathway and measure its properties.

"To support reproducible research, our lab makes a strong effort to share software and data," said Brian Wandell, senior author of the paper and a psychology professor at Stanford. "We believe this is a powerful way to ensure that our findings can be both checked and used in labs around the world."

The researchers also hope that the algorithm will enable other researchers to study the pathway, possibly leading to a better understanding of its role in human cognition and in patient populations.

In addition to Yeatman, Weiner and Wandell, other co-authors are Franco Pestilli, Ariel Rokem and Aviv Mezer. This work was funded by grants to Wandell from the National Institutes of Health and the National Science Foundation.

http://www.eurekalert.org/pub_releases/2014-11/bu-rsw111414.php

Research suggests warmth, flowing water on early Mars were episodic

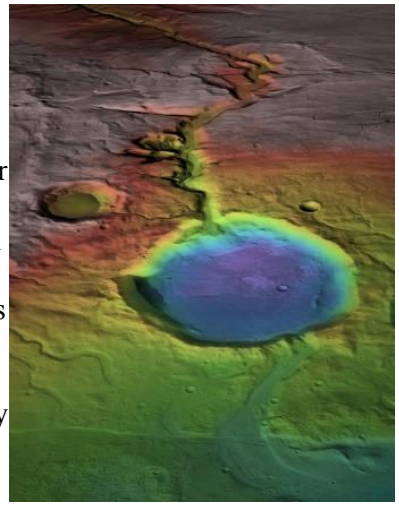
Ample evidence of ancient rivers, streams, and lakes make it clear that Mars was at some point warm enough for liquid water to flow on its surface.

PROVIDENCE, R.I. [Brown University] - While that may conjure up images of a tropical Martian paradise, new research published today in Nature Geoscience throws a bit of cold water on that notion.

The study, by scientists from Brown University and Israel's Weizmann Institute of Science, suggests that warmth and water flow on ancient Mars were probably episodic, related to brief periods of volcanic activity that spewed tons of greenhouse-inducing sulfur dioxide gas into the atmosphere. The work, which combines the effect of volcanism with the latest climate models of early Mars, suggests that periods of temperatures warm enough for water to flow likely lasted for only tens or hundreds of years at a time.

With all that's been learned about Mars in recent years, the mystery of the planet's ancient water has deepened in some respects. The latest generation of climate models for early Mars suggests an atmosphere too thin to heat the planet enough for water to flow. The sun was also much dimmer billions of years ago than it is today, further complicating the picture of a warmer early Mars.

"These new climate models that predict a cold and ice-covered world have been difficult to reconcile with the abundant evidence that water flowed across the surface to form streams and lakes," said James W. Head, professor of earth, environmental and planetary sciences at Brown University and co-author of the new paper with Weizmann's Itay Halevy. "This new analysis provides a mechanism for episodic periods of heating and melting of snow and ice that could have each lasted decades to centuries."



Halevy and Head explored the idea that heating may have been linked to periodic volcanism. Many of the geological features that suggest water flow date to around 3.7 billion years ago, a time when massive volcanoes are thought to have been active and huge lava outpourings occurred. On Earth, however, widespread volcanism often leads to cooling rather than warming. Sulfuric acid particles and thick ash reflect the sun's rays, and that can lower temperatures. But Head and Halevy thought the effects of sulfur in Mars' dusty atmosphere might have been different.

Although the surface is now cold and desiccated, in early Mars history water formed an open-basin lake, filling the crater, forming a delta, and breaching the lower rim as water flowed to lower elevations (blue). New research suggests that warmer temperatures and water flow on ancient Mars were likely related to periodic volcanism early in the planet's history **NASA/Mars Reconnaissance Orbiter Rendering by James Dickson, Brown University**

To find out, the researchers created a model of how sulfuric acid might react with the widespread dust in the Martian atmosphere. The work suggests that those sulfuric acid particles would have glommed onto dust particles, which would reduce their ability to reflect the sun's rays. Meanwhile sulfur dioxide gas would produce a modest greenhouse effect -- just enough to warm the Martian equatorial region so that water could flow.

Head has been doing fieldwork for years in Antarctica and thinks the climate on early Mars may have been very similar to that of the cold, desert-like McMurdo Dry Valleys. "The average yearly temperature in the Antarctic Dry Valleys is way below freezing, but peak summer daytime temperatures can exceed the melting point of water, forming transient streams, which then refreeze," Head said. "In a similar manner, we find that volcanism can bring the temperature on early Mars above the melting point for decades to centuries, causing episodic periods of stream and lake formation."

But as that early active volcanism on Mars ceased, so did the possibility of warmer temperatures and flowing water. Head said the research may offer new clues about where the fossilized remnants of life might be found on Mars, if it ever existed. "Life in Antarctica, in the form of algal mats, is very resistant to extremely cold and dry conditions and simply waits for the episodic infusion of water to 'bloom' and develop," he said. "Thus, the ancient and currently dry and barren river and lake floors on Mars may harbor the remnants of similar primitive life, if it ever occurred on Mars."

http://www.eurekalert.org/pub_releases/2014-11/tjnj-eoo111414.php

Effect of once-daily, low-dose aspirin on CV death and other outcomes

Investigating whether once-daily, low-dose aspirin would reduce the total number of cardiovascular events

Yasuo Ikeda, M.D., of Waseda University, Tokyo, Japan, and colleagues examined whether once-daily, low-dose aspirin would reduce the total number of cardiovascular (CV) events (death from CV causes, nonfatal heart attack or stroke) compared with no aspirin in Japanese patients 60 years or older with hypertension, diabetes, or poor cholesterol or triglyceride levels. The study appears in JAMA and is being released to coincide with its presentation at the American Heart Association's Scientific Sessions 2014.

The World Health Organization estimates that annual global mortality due to cardiovascular diseases (including heart attack and stroke) will approach 25 million by 2030. A recent study of trends in cardiovascular disease in Japan indicated that there has been, from 1960 to 2000, a steep increase in the prevalence of glucose intolerance, hypercholesterolemia, and obesity, probably due to the adoption of Western diets and lifestyles. By 2030, it is estimated that 32 percent of the Japanese population will be 65 years or older. Prevention of atherosclerotic cardiovascular diseases is an important public health priority in Japan due to an aging population, according to background information in the article.

This study included 14,464 patients (60 to 85 years of age) with hypertension, dyslipidemia (poor cholesterol or triglyceride levels), or diabetes mellitus who were randomized to aspirin (100 mg/d) or no aspirin in addition to ongoing medications. The patients were recruited by primary care physicians at 1,007 clinics in Japan. The study was terminated early by the data monitoring committee after a median follow-up of 5.02 years based on likely futility.

The researchers found that there was no statistically significant difference between the two groups in time to the primary end point (a composite of death from cardiovascular causes, nonfatal stroke, and nonfatal heart attack). At 5 years after randomization, the cumulative primary event rate was similar in participants in the aspirin group (2.77 percent) and those in the no aspirin group (2.96 percent). Aspirin significantly reduced incidence of nonfatal heart attack and transient ischemic attack, and significantly increased the risk of extracranial hemorrhage requiring transfusion or hospitalization.

The authors write that despite inconsistent evidence for the benefit of aspirin in primary prevention of cardiovascular events, the benefits in secondary prevention are well documented, including in Japanese patients. "There is also a growing body of evidence to suggest benefits for aspirin in the prevention of colorectal and other cancers, and the prevention of cancer recurrence, including in the Japanese population. Reduction in the incidence of colorectal cancer may influence the overall benefit-risk profile of aspirin. Further analyses of [this] study data are planned, including analysis of deaths associated with cancers, to allow more precise identification of the patients for whom aspirin treatment may be most beneficial."

J. Michael Gaziano, M.D., M.P.H., of the Veterans Affairs Boston Healthcare System, Brigham and Women's Hospital, Harvard Medical School, Boston, and Associate Editor, JAMA, and Philip Greenland, M.D., of the Northwestern University Feinberg School of Medicine, Chicago, and Senior Editor, JAMA, write in an accompanying editorial that the findings from this study adds to the body of evidence that helps refine the answer to the question of when aspirin should be used to prevent vascular events.

"Decision making involves an assessment of individual risk-to-benefit that should be discussed between clinician and patient. However, at present the choice of aspirin remains clear in several situations. Aspirin is indicated for patients at high short-term risk due to an acute vascular event and those undergoing certain vascular procedures; patients with any evidence of vascular disease should be given daily aspirin. On the other hand, patients at very low risk of vascular events should not take aspirin for prevention of vascular events, even at low dose."

"However, some individuals who do not have overt vascular disease will have risk levels that approach those of patients with CVD (such as patients with multiple risk factors). It remains likely that there is some level of risk of CVD events that would result in a positive trade-off of benefit and risk for the use of aspirin, but the precise level of risk is uncertain."

doi:10.1001/jama.2014.15690 doi:10.1001/jama.2014.16047

http://www.eurekalert.org/pub_releases/2014-11/uoia-gfr111414.php

Growth factor regenerates damaged nerves without sprouting new blood vessels

Growth factor can regenerate damaged peripheral nerves without causing the growth of new blood vessels

Researchers at the University of Illinois at Chicago College of Medicine have found that a growth factor can regenerate damaged peripheral nerves without causing the growth of new blood vessels -- making it a unique candidate to treat nerve damage in areas of the body where the proliferation of blood vessels would be a drawback.

"One example would be in the cornea, which has a requirement for dense innervation but where the formation of new blood vessels would block vision," said Dr. Mark Rosenblatt, professor and head of ophthalmology and visual sciences at UIC and corresponding author on the study, published in the Proceedings of the National Academy of Sciences.

Peripheral nerves -- those outside the brain, spinal cord and optic nerve -- have the capacity to regenerate when damaged. The process is guided by numerous signaling mechanisms, including a family of growth factors called VEGFs, or vascular endothelial growth factors, which are involved in the development of blood vessels as well as nerves. Understanding exactly how they work could lead to the development of drugs that enhance the body's ability to repair damaged nerves.

VEGF-A is a factor that Rosenblatt and several others have studied extensively. It helps repair damaged nerves, but also induces angiogenesis - the formation of new blood vessels. Rosenblatt and colleagues wanted to better understand the role of a related growth factor, VEGF-B, in neuroregeneration and angiogenesis. They investigated its effects in the corneas of mice. They found that mice lacking VEGF-B had a significantly impaired ability to repair damage to the corneal nerve. But if VEGF-B was delivered to the corneas of these mice, nerve regeneration improved. The new nerves restored normal sensation to the eye, and proper secretion of chemical signals to maintain the health of the cornea.

Importantly, the researchers also found that treatment with VEGF-B did not induce formation of new blood vessels, or have any effect on undamaged nerves. In experiments with normal mice able to produce VEGF-B, Rosenblatt saw that levels of the growth factor rose significantly around corneal nerves after they were damaged. "The selective effects of VEGF-B on injured nerves -- and its lack of angiogenic activity -- suggest that its main function may be neuroregeneration," Rosenblatt said.

The findings, he said, warrant further investigation of VEGF-B as a potential therapy to treat corneal nerve damage, which can be caused by dry eye, contact lenses, viruses or eye surgery, in addition to trauma. As a treatment, VEGF-B may prove superior to nerve growth factor, which has been used to treat certain eye diseases but can cause significant eye pain or the growth of new blood vessels.

Co-authors on the study are Victor Guaiquil, Zan Pan, Natalia Karagianni, Shima Fukuoka and Gemstonn Alegre of Weill Cornell Medical College in New York.

This research was supported by grants R01EY018594 and K08EY015829 from the National Eye Institute of the National Institutes of Health and by a Research to Prevent Blindness Career Development Award.

http://www.eurekalert.org/pub_releases/2014-11/s-ftt111714.php

Family ties that bind: Having the right surname sets you up for life

'Laws of inheritance' govern social status across generations

If your surname reveals that you descended from the "in" crowd in the England of 1066--the Norman Conquerors--then even now you are more likely than the average Brit to be upper class. To a surprising degree, the social status of your ancestors many generations in the past still exerts an influence on your life chances, say Gregory Clark of the University of California, Davis, in the US and Neil Cummins of the London School of Economics in the UK. They used the Oxbridge attendance of people with rare English surnames (last names) to track social mobility from 1170 to 2012. In an article in Springer's journal Human Nature, they show that social mobility in England has always been slow and today is not much greater than it was in pre-industrial times.

Social status is generally seen as a ranking of families across such aspects of status as education, income, wealth, occupation, and health. Clark and Cummings used various databases to calculate the social trajectory of families with rare English surnames over the past 28 generations. For this purpose, they analyzed the surnames of students who attended Oxford and Cambridge universities between 1170 and 2012, rich property owners between 1236 and 1299, as well as the national probate registry since 1858. Rare surnames such as Atthill, Bunduck, Balfour, Bramston, Cheslyn, and Conyngham were included in the study.

Clark and Cummins found that social status is consistently passed down among families over multiple generations--in fact, it is even more strongly inherited than height. This correlation is unchanged over centuries, with social mobility in England in 2012 being little greater than in pre-industrial times.

Their analysis further shows that the rate of social mobility in any society can be estimated from the knowledge of just two facts: the distribution over time of surnames in the society and the distribution of surnames among an elite or underclass.

"The relative constancy of the intergenerational correlation of underlying social status across very different social environments in England from 1800 to 2012 suggests that it stems from the nature of inheritance of characteristics within families," says Clark. "Strong forces of familial culture, social connections, and genetics must connect the generations."

"Even more remarkable is the lack of a sign of any decline in status persistence across major institutional changes, such as the Industrial Revolution of the eighteenth century, the spread of universal schooling in the late nineteenth century, or the rise of the social democratic state in the twentieth century," adds Cummins. "Status persistence measured by education status is just as strong now as in the pre-industrial era."

Reference: Clark, G. & Cummins, N. (2014). Surnames and Social Mobility in England, 1170-2012, Human Nature. DOI 10.1007/s12110-014-9219-y

http://www.eurekalert.org/pub_releases/2014-11/m-cko111714.php

Chlamydia knock out the body's own cancer defence

By breaking down the cancer-suppressing protein p53, Chlamydia prevent programmed cell death and thereby favor the process of cancer development

Infections due to the sexually transmitted bacterium Chlamydia trachomatis often remain unnoticed. The pathogen is not only a common cause of female infertility; it is also suspected of increasing the risk of abdominal cancer. A research team at the Max Planck Institute for Infection Biology in Berlin has now observed the breakdown of an important endogenous protective factor in the course of chlamydial infection. By activating the destruction of p53 protein, the bacterium blocks a key protective mechanism of infected cells, the initiation of programmed cell death. This protective function of p53 is also impaired in many forms of cancer. The new insights underpin the suspected relationship between chlamydial infection and the occurrence of certain types of cancers.

Hundreds of mutations occur every day in almost every cell in our body. The protein p53 is then activated in order to limit these changes in the genome: either the cell repairs the damaged DNA or, if that is not possible, it triggers the cellular suicide program. In this way, cells are normally protected against the development of cancer.

As the Berlin-based team at the Max Planck Institute for Infection Biology reported last year, chlamydial infections lead to a drastic increase in the mutation rate. Activation of the suicide program would be fatal for Chlamydia, however, as the bacteria are only able to multiply inside their host cells from which they draw

their nutrients. To protect themselves, Chlamydia therefore block activation of the cellular suicide program.

With the help of colleagues from the Max Delbrück Center for Molecular Medicine and from Australia, the Max Planck team has now shown that Chlamydia ensure the survival of host cells by breaking down p53. They do so by activating a breakdown pathway that is already present in cells. The pathogens thereby gain enough time to successfully reproduce inside the cells. However, this has potentially fatal consequences for the host organism: destruction of p53, the central "guardian of the genome", increases the risk of mutant cells surviving and developing into cancer cells.

Degradation of p53 is also observed in infections with human papillomavirus, the cause of cervical cancer. Chlamydia may play a role in this disease as well. However, they penetrate much deeper into the genital tract and can cause inflammation of the fallopian tubes, where they often reside unnoticed for a long time. Ovarian cancer, one of the deadliest cancers in women, is now also believed to originate within the fallopian tubes.

"The impact of Chlamydia on p53 is an important part in the complex puzzle of cancer development. The more substantiated the relationship between infection and cancer becomes, the more important it will be to promote the development of effective vaccines and antibiotics to prevent cancer," says Thomas F. Meyer, Director at the Max Planck Institute in Berlin.

González E, Rother M, Kerr MC, Al-Zeer M, Abu-Lubad M, Kessler M, Brinkmann V, Loewer A & Meyer TF Chlamydia infection depends on a functional MDM2-p53 axis
Nature Communications 2014, 13 November 2014

http://bit.ly/1qyMHqT

Eat More Seaweed (It's Good for You)

Foraging for fresh seaweeds gives you option and the best taste according to this Brittany seaweed eater

By Marissa Fessenden

To make this three-seaweed quiche, you will need the bright green sea lettuce (*Ulva lactuca)*, a reddish algae from the genus *Porphrya* and some fresh dulse (*Palmaria palmata*). Combine with crème fraîche, butter, cheese, eggs, sauteed onions, perhaps some carrots and zucchini. Mound it altogether in a pastry. Delicious, as long as your seaweed is fresh - perhaps picked that very morning at low tide.

As seaweed forager Cristelle Maine demonstrates in the video above, via The Kid Should See This, anyone who lives near the sea might enjoy the wide variety of edible plants that grow on the edge of the land. "It is not like with mushrooms," she says (according to the subtitles - Maine speaks French). "There's no seaweed that is naturally toxic." She describes the different tastes of the red, green and brown seaweeds that she collects and cooks into a seaweed tart.

If you frequent sushi restaurants, with their nori and succulent seaweed salads, noshing on greens from the sea may not seem odd. Seaweed has found its way to the plates of many shore-dwelling cultures, probably because it is abundant but also low in calories and rich in vitamins and minerals. And, in fact, seaweed is farmed and foraged around the world.

Its place in Western cuisine hasn't been explored as much, though - perhaps because it's "associated with poverty," the BBC suggests. In Wales, for example laverbread - "bara lawr" in Welsh - includes a paste of cooked seaweed. In Ireland, dried dulse makes a snack. So, if you live near the ocean, perhaps now is the time to add some new plants to your diet.

http://bit.ly/1uWb9T0

Largest study of gay brothers homes in on 'gay genes'

A genetic analysis of 409 pairs of gay brothers, including sets of twins, has provided the strongest evidence yet that gay people are born gay.

15:48 17 November 2014 by Andy Coghlan

The study clearly links sexual orientation in men with two regions of the human genome that have been implicated before, one on the X chromosome and one on chromosome 8.

The finding is an important contribution to mounting evidence that being gay is biologically determined rather than a lifestyle choice. In some countries, such as Uganda, being gay is still criminalised, and some religious groups believe that gay people can be "treated" to make them straight.

"It erodes the notion that sexual orientation is a choice," says study leader Alan Sanders of the NorthShore Research Institute in Evanston, Illinois.

The region on the X chromosome picked out by the study, called Xq28, was originally identified in 1993 by Dean Hamer of the US National Institutes of Health in Bethesda, Maryland, but attempts to validate the finding since have been mixed. The other region picked out is in the twist in the centre of chromosome 8. Known as 8q12, it was first signposted in 2005.

Statistically stronger

The latest study involves about three times as many people as the previous largest study, which means it is significantly more statistically robust.

Over the past five years, Sanders has collected blood and saliva samples from 409 pairs of gay brothers, including non-identical twins, from 384 families. This compares, for example, with 40 pairs of brothers recruited for Hamer's study.

The team combed through the samples, looking at the locations of genetic markers called single nucleotide polymorphisms (SNPs) – differences of a single letter in

the genetic code – and measuring the extent to which each of the SNPs were shared by the men in the study.

The only trait unequivocally shared by all 818 men was being gay. All other traits, such as hair colour, height and intelligence, varied by different degrees between each brothers in a pair and between all sets of brothers. Therefore, any SNPs consistently found in the same genetic locations across the group would most likely be associated with sexual orientation.

Only five SNPs stood out and of these, the ones most commonly shared were from the Xq28 and 8q12 regions on the X chromosome and chromosome 8 respectively. But this doesn't mean the study found two "gay genes". Both regions contain many genes, and the next step will be to home in on which ones might be contributing to sexual orientation.

Sanders says he has already completed the work for that next step: he has compared SNPs in those specific regions in gay and straight men to see if there are obvious differences in the gene variants, and is now preparing the results for publication. "Through this study, we have the potential to narrow down to fewer genes," says Sanders.

Not just genetic

Whatever the results, Sanders stresses that complex traits such as sexual orientation depend on multiple factors, both environmental and genetic. Even if he has hit on individual genes, they will likely only have at most a small effect on their own, as has also been seen in studies of the genetic basis for intelligence, for example.

Other researchers who have looked at the biological origins of sexual orientation have welcomed the latest findings, saying they help resolve contradictory results from earlier, smaller studies. "The most pleasing aspect is that the confirmation comes from a team that was in the past somewhat sceptical and critical of the earlier findings," says Andrea Camperio Ciani of the University of Padua in Italy.

"This study knocks another nail into the coffin of the 'chosen lifestyle' theory of homosexuality," says Simon LeVay, the neuroscientist and writer who, in 1991, claimed to have found that a specific brain region, within the hypothalamus, is smaller in gay men. "Yes, we have a choice in life, to be ourselves or to conform to someone else's idea of normality, but being straight, bisexual or gay, or none of these, is a central part of who we are, thanks in part to the DNA we were born with."

"Much hard work now lies ahead to identify the specific genes involved and how they work, as well as to find equivalent genes in women," he adds.

Hamer himself, now a documentary film-maker, is delighted with the result. "Twenty years is a long time to wait for validation, but now it's clear the original results were right," he says. "It's very nice to see it confirmed."

Leader: *"Gay gene discovery has good and bad implications"*

Journal reference: Psychological Medicine, DOI: 10.1017/S0033291714002451

Correction, 18 November 2014: *When this article was first published, we said that all the participants in the study were non-identical twins. They are in fact pairs of brothers, although some are non-identical twins.*

Why I took part in gene study, and what it means to me

As a doctor, I recognise the importance of furthering science through legitimate research. As a gay man, I've known that my sexuality has never been a choice but I could not explain, to myself or anyone else, how I became this way. Genetics and environmental influences seemed logical. This study is an attempt to answer the genetics part of the question.

The results may provide validation for homosexual men who have asked the same questions that I have. They may improve the self-esteem of the many men who have asked "why me?", or have felt ostracised, prejudiced, put down, left out, demonised, or worse. They might possibly change the minds of those who believe homosexuality is a "choice" rather than something predetermined.

However, it is important that the findings be put in context. Inevitable headlines like "Gay gene discovered" or "It's not a choice" over-egg the results. Just because there is a genetic link to homosexuality, it does not necessarily guarantee one will end up gay. The genes, if and when they are identified, may only predispose one to the possibility of being gay, should the required environmental, nutritional or other unknown factors be present at critical stages of development.

On a darker level, some may use the results to justify a belief that homosexuality is the result of a "broken" or "deviant" gene that needs to be fixed. Imagine parents requesting a genetic test on their unborn fetus, or worse, a government rolling out mandatory testing of all unborn children, and using compulsory abortions to cleanse the gene pool. There is enough hate in the world that this concept is not as outrageous as one might think.

Despite this, I remain hopeful that our world will continue to evolve into a safer and more accepting place for everyone. While some countries are going backwards, there is a greater openness around the world to homosexuality. This openness, coupled with scientific fact, will bring a greater understanding of human sexuality to a new generation. **Chad Zawitz**

Chad Zawitz *is a senior physician at a clinic in Chicago, coordinating services for people with HIV and other infectious diseases. He took part in the study with his twin brother*

http://bit.ly/1vsFD10

Sushi Edging Pacific Bluefin Tuna Toward Extinction

The Pacific bluefin tuna, a fish used in sushi and sashimi dishes, is at risk of extinction as the global food market places "unsustainable pressure" on the species and others, a conservation body warned Monday.

The bluefin tuna joined the Chinese pufferfish, American eel, Chinese cobra and Australian black grass-dart butterfly on the International Union for Conservation of Nature's (IUCN) "red list" of threatened species.

The updated list was released by the IUCN at its once-a-decade World Parks Congress in Sydney as it called for better management of protected areas, where some of the decline in species levels has taken place. "Each update of the IUCN 'red list' makes us realise that our planet is constantly losing its incredible diversity of life, largely due to our destructive actions to satisfy our growing appetite for resources," IUCN's director-general Julia Marton-Lefevre said.

"But we have scientific evidence that protected areas can play a central role in reversing this trend," she added. For this year's list, the IUCN assessed 76,199 species, with 22,413 judged to be under threat.

The Pacific bluefin tuna moved from the "least concern" threat category to "vulnerable" as the species is threatened with extinction due to its use in Asia's sushi and sashimi markets, the Swiss-based group said. As most of the fish caught are juveniles that have not yet reproduced, the population has dropped by 19-33 percent over the past 22 years. It called for fisheries to implement conservation and management measures for the Western and Central Pacific Ocean.

The American eel is reeling from the impact of climate change, parasites, pollution, habitat loss and commercial harvesting, as well as having been hit by the high levels of consumption of its counterpart, the Japanese eel. The bluefin is fetching record prices, so prized is its meat. Is using humble mackerel as surrogate parents the way to keep the bluefin from going extinct?

The IUCN categorised the Chinese cobra as "vulnerable" with the population falling 30-50 percent over the past two decades -- another species hurt by its popularity as a food source. "The growing food market is putting unsustainable pressure on these and other species," the IUCN's biodiversity head Jane Smart said. "We urgently need to impose strict limits on harvesting and take appropriate measures to protect habitats."

Another species added to the list was the Malaysian snail Charopa lafargei - named after the French construction giant Lafarge, which has agreed to try and limit its quarrying activities in the snails' habitat - the report said.

Two species, the Malaysian mollusc plectostoma sciaphilum and the St Helena Giant Earwig, were declared extinct due to habitat destruction.

But there was good news for two amphibians in Colombia's Ranita Dorada Reserve -- both members of the poison dart frogs family -- which improved in status and are now categorised as "vulnerable" due to conservation efforts.

The World Parks Congress, which will outline a global agenda for protected areas for the next decade before closing on November 19, comes a month after the member nations of the UN's Convention of Biological Diversity met in South Korea to lay out a roadmap to halt species extinction by 2020.

The World Wildlife Fund said in its Living Planet Report published in September that there has been a 52 percent decline in mammals, birds, reptiles, amphibians and fish overall from 1970 to 2010.

http://bit.ly/1xygkcI

Scientists 'confident' comet lander will wake up (Update)

A burst of sunshine in the spring could be just the wakeup call for Europe's comet lander.

November 17th, 2014 by Frank Jordans in Astronomy & Space / Space Exploration

Scientists raised hopes Monday that as the Philae lander nears the sun its solar panel-powered battery will recharge, and the first spacecraft to touch down on a comet will send a second round of scientific data back to Earth.

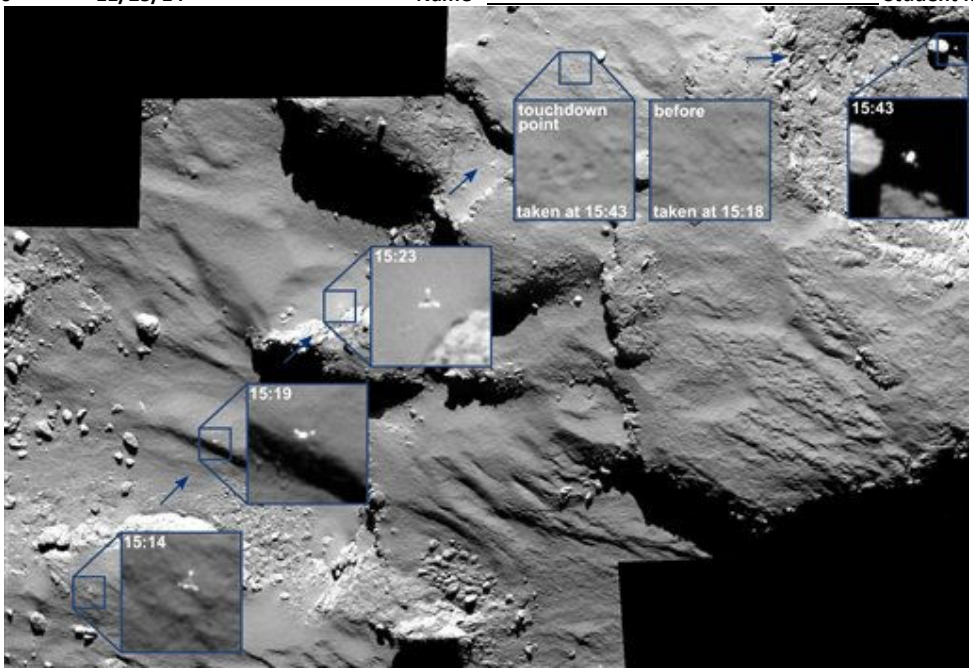
Since landing with a bounce on the comet Wednesday, Philae has already sent back reams of data that scientists are eagerly examining. But there were fears its mission would be cut short because it came to rest in the shadow of a cliff. Its signal went silent Saturday after its primary battery ran out.

Shortly before that happened, the European Space Agency decided to attempt to tilt the lander's biggest solar panel toward the sun - a last-ditch maneuver that scientists believe may have paid off.

"We are very confident at some stage it will wake up again and we can achieve contact," Stephan Ulamec, the lander manager, told The Associated Press.

That should happen next spring, when Philae and the comet it's riding on - called 67P/Churyumov-Gerasimenko - get closer to the sun, warming up a secondary battery on board and bringing it out of its unplanned hibernation. A few days of sunshine on the solar panels should be enough to charge the battery sufficiently to resume collecting scientific data, Ulamec said.

Philae's position in the shadows may even prove to be a blessing in disguise. Shielded from the sun's rays, the lander could survive for longer as the comet approaches perihelion - its closest point to the sun - in August. Before they can say for certain if they'll be able to restore contact with Philae, scientists first need to find out where on the 2.5-mile (4-kilometer)-wide comet the washing machine-sized lander is, he added. New pictures released Monday offered very good clues.



The combination image of several partially enlarged photographs released by the European Space Agency, ESA, Monday Nov. 17, 2014 shows the journey of Rosetta's Philae lander as it approached and then rebounded from its first touchdown on Comet 67P/Churyumov–Gerasimenko on Nov. 12, 2014. The series of images was captured by Rosetta's OSIRIS camera from a distance of 15.5km (9.6 miles) from the comet surface over a 30 minute period spanning the first touchdown. The time of each of image has been marked by source on the corresponding insets and is in GMT. A comparison of the touchdown area shortly before and after first contact with the surface is also provided. From left to right, the images show Philae descending towards and across the comet before touchdown. **(AP Photo/ESA)**

The high-resolution images taken from Philae's mother ship Rosetta show the lander descending toward the comet, then bouncing off when the thrusters and harpoons meant to anchor it to the surface failed. It drifted through the void for two hours before touching down again - after a second, smaller bounce - then coming to rest in a shallow crater.

Scientists at the German Aerospace Center said Monday that an initial review of data the lander sent back 311 million miles to Earth showed the comet's surface is much tougher than previously assumed. There's also evidence of large amounts of ice beneath the lander.

Scientists had speculated the comet's surface could be quite soft, but that has turned out not to be the case. "The strength of the ice found under a layer of dust on the first landing site is surprisingly high," said Klaus Seidensticker of the German Aerospace Center.

Scientists are still waiting to find out whether Philae managed to drill into the comet and extract a sample for analysis. Material beneath the surface of the comet has remained almost unchanged for 4.5 billion years, so the samples would be a cosmic time capsule that scientists are eager to study. One of the things they are most excited about is the possibility that the mission might help confirm that comets brought the building blocks of life - including water - to Earth.

Tantalizingly, one of Philae's instruments was able to "sniff" the presence of organic molecules on the comet, the space center said. A full analysis of the molecules is still underway. The European Space Agency has stressed that even if the lander fails to awaken again, Rosetta will be able to collect about 80 percent of the data scientists are hoping to glean from the $1.6 billion mission.

http://www.eurekalert.org/pub_releases/2014-11/eeco-tst111814.php

Trial shows treatment-resistant advanced non-small cell lung cancer responds to rociletinib

Promising results shown by new drug targeting both common cancer-causing genetic mutations in patients with non-small cell lung cancer, but also a mutation causing resistance to treatment

Barcelona, Spain - A new drug that targets not only common cancer-causing genetic mutations in patients with non-small cell lung cancer (NSCLC), but also a form of the mutation that causes resistance to treatment, has shown promising results in patients in a phase I/II clinical trial. The research will be presented today (Friday) at the 26th EORTC-NCI-AACR Symposium on Molecular Targets and Cancer Therapeutics in Barcelona, Spain.

Approximately 10-15% of Caucasian and 30-35% of Asian patients with NSCLC have a mutation in the epidermal growth factor receptor (EGFR), which can be successfully targeted with EGFR inhibitors called tyrosine kinase inhibitors (TKI), such as erlotinib, gefitinib and afatinib. However, these patients will eventually develop resistance to EGFR TKI therapy and a further EGFR mutation called T790M accounts for 60% of this acquired resistance.

Professor Jean-Charles Soria, Chairman of the Drug Development Department at Gustave Roussy Cancer campus, France, will tell the Symposium: "Currently, there are no approved targeted therapies for mutant EGFR lung cancer patients who develop the T790M mutation, which means their disease inevitably will get worse. Rociletinib (CO-1686) is a new and potent oral EGFR inhibitor designed

to selectively target both the initial activating EGFR mutations as well as the T790M resistance mutation. This compound spares normal (wild-type) EGFR and this means that it causes far fewer toxic side-effects than other EGFR inhibitors. Therefore, it may benefit patients both as a first-line and second- or later-line treatment, by producing a durable clinical benefit and with a reduced toxicity profile compared to current EGFR inhibitor therapies. Current TKIs inhibit the normal EGFR as well as the mutant EGFR, causing acne-like skin rashes and paronychia - an inflammation of the folds of tissue around finger and toe nails - both of which can be very troublesome for patients."

Patients with advanced NSCLC with the EGFR mutation, with or without the T790M resistance mutation, were enrolled in the phase I/II clinical trial in centres in Europe, Australia and the USA; enrolment of patients for the phase I part of the study began in March 2012, and for the phase II part in August 2013. The phase I portion of the study examined two formulations and multiple doses and schedules of rociletinib; 625mg twice a day continuously of hydrobromide (HBr) salt tablet form of rociletinib was identified as the pivotal dose, schedule and formulation for the phase II part of the study.

By October 2014, 179 patients had been treated at therapeutic doses (either 900mg twice a day of freebase formulation, or 500mg or more twice a day of HBr salt tablet). Preliminary results for all of these patients (those with and without the T790M and T790M resistance mutation) include an overall response rate of 46% and a disease control rate of 84%.

Prof Soria will present detailed data to the Symposium on 56 patients who had the T790M resistance mutation and received the pivotal dose and formulation (625mg twice a day) or the reduced dose of 500mg twice a day. The median number of prior therapies for these patients was three; all the patients had been treated previously with at least one other EGFR TKI therapy, and most patients receiving chemotherapy as well. Approximately 80% of these patients were treated immediately after their cancer progressed during treatment with a TKI. The study is ongoing, accruing patients rapidly, and CT scan data are available on 27 of these patients, of whom 18 had a confirmed response to the treatment, giving an overall response rate of 67% and a median progression-free survival of 10.4 months.

Among an additional 11 evaluable patients who did not have the T790M mutation, four had a confirmed response to the treatment (overall response rate of 36%) and this group of patients had a median progression-free survival of 7.5 months. Prof Soria will say: "Re-sensitisation to TKI cannot account for the majority of these responses, since most patients had come off TKI as their immediate prior therapy."

Adverse side-effects of rociletinib were manageable and included asymptomatic hyperglycaemia (high blood sugar levels), nausea and diarrhoea, and these were mostly mild or moderate (grade 1 or 2). Only two patients had any form of rash, which was grade 1 and transient. The most common, more severe adverse event (grade 3) was hyperglycaemia, which was observed in 14% of patients. Hyperglycaemia can usually be managed with a commonly-prescribed oral drug.

Prof Soria will say: "Eventually, almost all lung cancer patients with EGFR mutations will develop resistance to currently available therapies, including TKI, leaving doctors and patients without effective options to treat this deadly disease. The data from the rociletinib clinical trials suggest that we may be able to successfully target and overcome resistance to EGFR inhibitors bring new, targeted treatments to patients who need them the most." The responses seen in the patients who had acquired resistance to earlier TKI treatment but without evidence of T790M mutation was unexpected. Possible explanations include:

the presence of large regions of tumour that do have the T790M mutation, but were missed by the biopsy needle (tumour heterogeneity);

the test is not sensitive enough to detect low levels of the T790M mutation, resulting in a false negative;

rociletinib inhibits an alternative pathway (a "bypass track"), other than EGFR, which drives acquired resistance to EGFR TKI. "Indeed, we now know a metabolite of rociletinib inhibits the IGF1-R pathway, which we believe may account for some of the activity observed in T790M-negative patients," Prof Soria will say.

Professor Josep Tabernero, a member of the scientific committee for the EORTC-NCI-AACR Symposium and head of the medical oncology department at Vall d'Hebron University Hospital and director of the Vall d'Hebron Institute of Oncology, Barcelona, Spain, commented: "Lung cancer is an extremely difficult disease to treat successfully, with only about one in ten patients living for five years or longer. Drug resistance is one of the main problems encountered when trying to treat it; therefore, a therapy that can overcome this resistance in the proportion of patients with non-small cell lung cancer with EGFR mutations is an important step forward. Rociletinib precisely targets the EGFR mutant population with the specific T790M mutation, leaving normal EGFR unaffected, and this means that it offers patients the possibility of a longer life with fewer of the adverse side-effects encountered with other drugs."

EORTC [European Organisation for Research and Treatment of Cancer, NCI [National Cancer Institute], AACR [American Association for Cancer Research]. TKI (tyrosine kinase inhibitors) inhibit tyrosine kinases, which are enzymes that trigger the cancer-causing activity of the epidermal growth factor receptor (EGFR).

http://www.eurekalert.org/pub_releases/2014-11/cwru-bop111814.php

Breakthrough offers promise for spinal cord injury patients to breathe on their own again

Case Western Reserve researcher presents findings that could free patients from ventilators -- even years after injury

Case Western Reserve researchers have developed a procedure that restores function to muscles involved in the control of breathing - even when they have been paralyzed for more than a year. The breakthrough offers hope that one day patients with severe spinal cord injuries will be able to breathe again without the assistance of a ventilator.

Principal investigator Philippa M. Warren, PhD, presented the results Nov. 17 at Neuroscience 2014, the annual meeting of the Society for Neuroscience. The research represents a critical step forward in efforts to reverse even long-term paralysis of muscles within the diaphragm that are activated by nerve fibers that extend from the upper part of the brain stem. When those fibers are damaged in the spinal cord, electrical signals from the brain cannot reach motor nerves that leave the spinal cord to activate muscles that control vital functions. This new research offers a two-step approach to repair the part of the damage that blocks those signals.

"We show that respiratory paralysis can be reversed at long intervals after spinal cord injury," said Warren, a neurosciences researcher at MetroHealth Medical Center, which is affiliated with Case Western Reserve University School of Medicine.

"This has the potential to alleviate the long suffering of currently injured patients, improving their quality, and potentially length, of life."

Investigators focused their research on a group of nerves that extend from the respiratory control center in the brain stem down to the C3 through C5 vertebral levels of the spinal cord located in the middle of the neck. These fibers, or brain axons, control the diaphragm muscle in its critical function of breathing. Any injury to the spinal cord above the C3 vertebra can cause widespread muscle paralysis leading to difficulties in breathing, but also moving, regulation of cardiac output, and sexual function.

Unfortunately, these injuries high in the neck are the most common among sufferers of spinal cord trauma.

Following injury to the spinal cord, damaged nerve fibers die, causing loss of the connections between the brain and muscles of the body. To help preserve tissue immediately after injury, a scar forms at the site of the trauma and extends the distance of several inches up and down the spinal cord. This scar tissue is very dense, contains sugars that inhibit new neuronal growth, and does not reduce in length or intensity over time. The consequence is that new connections cannot form to enable muscle function after injury, which is exceptionally important to breathing.

Spinal cord injury-induced paralysis of the respiratory muscles causes low oxygen in the blood, increases the body's drive to breathe and drives any functioning respiratory muscles to work harder. The breathing capacity of the spinal cord-injured is often not enough to fully support a patient's life.

However, if new nerve fibers or connections can form in the spinal cord, then pathways can be activated to restore respiratory function. So Case Western Reserve researchers devised a technique to treat the injury site with a specially designed enzyme to reopen connections and to apply respiratory therapy to strengthen the remaining functioning respiratory muscles.

In laboratory animals, investigators used the combination technique to restore respiratory function many months after the injury. First, they injected the chondroitinase enzyme at the site of respiratory nerves in the spinal cord to remove the inhibiting sugars from scar tissue.

The action of the enzyme enabled both the formation of new connections and stimulation of latent pathways in the respiratory motor system. Second, the animals were exposed to brief periods of conditions with low oxygen, making them breathe harder and faster to rehabilitate the respiratory muscles. This treatment approach is referred to as intermittent hypoxia.

The combination enzyme injection and intermittent hypoxia treatment boosts levels of serotonin. Commonly known to help relieve anxiety disorders, serotonin also acts more broadly as a neurotransmitter to help stimulate nerve cells. By increasing serotonin at nerve connections and at the specific receptors on the fibers themselves, the researchers were able to help restore diaphragm function back to normal levels in the animals.

This finding is extraordinary not only because function to the paralyzed muscle was completely restored, but also because researchers were able to achieve breathing in animals that had been injured for a year and a half.

"It is remarkable to reactivate the diaphragm and breathing in a chronically injured animal that has had a paralyzed half diaphragm most of its life," said Jerry Silver, PhD, a Case Western Reserve professor of neurosciences who collaborated in the research.

While these results are encouraging, more research is required to perfect the treatment. More than two-thirds of the animals in the study responded to the combined treatment strategy, while the treatment had no effect on the remaining animals. Two thirds of the animals that responded to the combined treatment

resumed normal breathing, while the other third experienced erratic breathing in the injured muscle.

Investigators found that the animals with erratic breathing were flooded with too much serotonin during their treatment. A simple fix involved administering a serotonin receptor blocker, which restored these animals to normal breathing.

Researchers are currently studying further the serotonin-overload phenomenon in animals to expand their knowledge of the chondroitinase enzyme/intermittent hypoxia treatment strategy.

While this treatment strategy holds great promise for use in humans, the technique must first be optimized and shown to be effective in larger animals with spinal cords more similar in size to that of humans.

"Treatment increased the strength of nerve connections, not at the site of injury, but where the diaphragm nerves leave the spinal cord," Warren said. "This may have huge implications for the treatment of sufferers with spinal cord injury. Our work offers new hope that it might be possible in the future to repair paralyzed respiratory muscle activity, even at long time periods after severe spinal injury, allowing patients to breathe normally again."

The work was conducted in the laboratory of Warren J. Alilain, PhD, assistant professor, the Department of Neurosciences, MetroHealth Medical Center and CWRU School of Medicine. This investigation also involved close collaboration with Professor Silver and Peter M. MacFarlane, PhD, assistant professor of pediatrics, CWRU School of Medicine.

The work was funded by Spinal Research (the International Spinal Research Trust), Wings for Life and the Craig H. Neilsen Foundation.

http://www.eurekalert.org/pub_releases/2014-11/icl-gmh111814.php

Gravity may have saved the universe after the Big Bang, say researchers

New research by a team of European physicists could explain why the universe did not collapse immediately after the Big Bang.

Studies of the Higgs particle - discovered at CERN in 2012 and responsible for giving mass to all particles - have suggested that the production of Higgs particles during the accelerating expansion of the very early universe (inflation) should have led to instability and collapse.

Scientists have been trying to find out why this didn't happen, leading to theories that there must be some new physics that will help explain the origins of the universe that has not yet been discovered. Physicists from Imperial College London, and the Universities of Copenhagen and Helsinki, however, believe there is a simpler explanation.

In a new study in Physical Review Letters, the team describe how the spacetime curvature - in effect, gravity - provided the stability needed for the universe to survive expansion in that early period. The team investigated the interaction between the Higgs particles and gravity, taking into account how it would vary with energy.

They show that even a small interaction would have been enough to stabilise the universe against decay.

"The Standard Model of particle physics, which scientists use to explain elementary particles and their interactions, has so far not provided an answer to why the universe did not collapse following the Big Bang," explains Professor Arttu Rajantie, from the Department of Physics at Imperial College London.

"Our research investigates the last unknown parameter in the Standard Model - the interaction between the Higgs particle and gravity. This parameter cannot be measured in particle accelerator experiments, but it has a big effect on the Higgs instability during inflation. Even a relatively small value is enough to explain the survival of the universe without any new physics!"

The team plan to continue their research using cosmological observations to look at this interaction in more detail and explain what effect it would have had on the development of the early universe. In particular, they will use data from current and future European Space Agency missions measuring cosmic microwave background radiation and gravitational waves.

"Our aim is to measure the interaction between gravity and the Higgs field using cosmological data," says Professor Rajantie. "If we are able to do that, we will have supplied the last unknown number in the Standard Model of particle physics and be closer to answering fundamental questions about how we are all here."

The research is funded by the Science and Technology Facilities Council, along with the Villum Foundation, in Denmark, and the Academy of Finland.

http://www.eurekalert.org/pub_releases/2014-11/ps-has111814.php

Herbs and spices enhance heart health as well as flavor

Spices and herbs are rich in antioxidants, which may help improve triglyceride concentrations and other blood lipids, according to Penn State nutritionists.

Triglyceride levels rise after eating a high-fat meal -- which can lead to an increased risk of heart disease.

If a high-antioxidant spice blend is incorporated into the meal, triglyceride levels may be reduced by as much as 30 percent when compared to eating an identical meal without the spice blend. The spiced meal included garlic powder, rosemary, oregano, cinnamon, cloves, paprika, turmeric, ginger and black pepper.

Sheila G. West, professor of biobehavioral health and nutritional sciences, and Ann C. Skulas-Ray, research associate in nutritional sciences, reviewed a variety

of research papers that focused on the effects that spices and herbs have on cardiovascular disease risk. They published their findings in a supplement to the current issue of the journal Nutrition Today, based on papers presented at the McCormick Science Institute Summit held in May 2014.

"The metabolic effects of spices and herbs and their efficacy and safety relative to traditional drug therapy represent an exciting area for future research given the public health significance of cardiovascular disease," the researchers wrote.

West and Skulas-Ray looked at three categories of studies -- spice blends, cinnamon and garlic.

"We live in a world where people consume too many calories every day," said West. "Adding high-antioxidant spices might be a way to reduce calories without sacrificing taste."

West and Skulas-Ray reviewed several cinnamon studies that looked at the effect of the spice on both diabetics and non-diabetics. Cinnamon was shown to help diabetics by significantly reducing cholesterol and other blood lipids in the study participants. However, cinnamon did not appear to have any effect on non-diabetics.

The garlic studies reviewed were inconclusive, but this is likely because the trials had a wide range of garlic doses, from nine milligrams of garlic oil to 10 grams of raw garlic. The reviewers noted that across the studies there was an eight percent decrease in total cholesterol with garlic consumption, which was associated with a 38 percent decrease in risk of heart problems in 50-year-old adults.

In the study West, Skulas-Ray and colleagues conducted, they prepared meals on two separate days for six men between the ages of 30 and 65 who were overweight, but otherwise healthy. The meals were identical -- consisting of chicken, bread and a dessert biscuit -- except that the researchers added two tablespoons of a high-antioxidant culinary spice blend to the test meal.

The researchers followed the participants for three hours after each meal, drawing blood every 30 minutes. Antioxidant activity in the blood increased by 13 percent after the men ate the test meal when compared to the control meal, which may help prevent cardiovascular disease and other chronic diseases.

West and colleagues are currently working on a study to monitor study participants for eight hours after eating a meal with a high-antioxidant spice blend. They want to know what happens to the fat in such a meal.

"If (the fat) isn't being absorbed when spices are included in the meal, it might be excreted instead," said West. "We will examine whether spices affect how rapidly the meal is processed through the stomach and intestines."

The McCormick Science Institute supported this work.

http://www.eurekalert.org/pub_releases/2014-11/sdmc-wna111814.php

Were Neanderthals a sub-species of modern humans? New research says no

Disappearance of Neanderthals likely the result of competition from Homo sapiens, and not from poor adaptation to cold

In an extensive, multi-institution study led by SUNY Downstate Medical Center, researchers have identified new evidence supporting the growing belief that Neanderthals were a distinct species separate from modern humans (Homo sapiens), and not a subspecies of modern humans.

The study looked at the entire nasal complex of Neanderthals and involved researchers with diverse academic backgrounds. Supported by funding from the National Science Foundation and the National Institutes of Health, the research also indicates that the Neanderthal nasal complex was not adaptively inferior to that of modern humans, and that the Neanderthals' extinction was likely due to competition from modern humans and not an inability of the Neanderthal nose to process a colder and drier climate.

Samuel Márquez, PhD, associate professor and co-discipline director of gross anatomy in SUNY Downstate's Department of Cell Biology, and his team of specialists published their findings on the Neanderthal nasal complex in the November issue of The Anatomical Record, which is part of a special issue on The Vertebrate Nose: Evolution, Structure, and Function (now online).

They argue that studies of the Neanderthal nose, which have spanned over a century and a half, have been approaching this anatomical enigma from the wrong perspective. Previous work has compared Neanderthal nasal dimensions to modern human populations such as the Inuit and modern Europeans, whose nasal complexes are adapted to cold and temperate climates.

However, the current study joins a growing body of evidence that the upper respiratory tracts of this extinct group functioned via a different set of rules as a result of a separate evolutionary history and overall cranial bauplan (bodyplan), resulting in a mosaic of features not found among any population of Homo sapiens. Thus Dr. Márquez and his team of paleoanthropologists, comparative anatomists, and an otolaryngologist have contributed to the understanding of two of the most controversial topics in paleoanthropology - were Neanderthals a different species from modern humans and which aspects of their cranial morphology evolved as adaptations to cold stress.

"The strategy was to have a comprehensive examination of the nasal region of diverse modern human population groups and then compare the data with the fossil evidence. We used traditional morphometrics, geometric morphometric

methodology based on 3D coordinate data, and CT imaging," Dr. Márquez explained.

Anthony S. Pagano, PhD, anatomy instructor at NYU Langone Medical Center, a co-author, traveled to many European museums carrying a microscribe digitizer, the instrument used to collect 3D coordinate data from the fossils studied in this work, as spatial information may be missed using traditional morphometric methods. "We interpreted our findings using the different strengths of the team members," Dr. Márquez said, "so that we can have a 'feel' for where these Neanderthals may lie along the modern human spectrum."

Co-author William Lawson, MD, DDS, vice-chair and the Eugen Grabscheid research professor of otolaryngology and director of the Paleorhinology Laboratory of the Icahn School of Medicine at Mount Sinai, notes that the external nasal aperture of the Neanderthals approximates some modern human populations but that their midfacial prognathism (protrusion of the midface) is startlingly different. That difference is one of a number of Neanderthal nasal traits suggesting an evolutionary development distinct from that of modern humans. Dr. Lawson's conclusion is predicated upon nearly four decades of clinical practice, in which he has seen over 7,000 patients representing a rich diversity of human nasal anatomy.

Distinguished Professor Jeffrey T. Laitman, PhD, also of the Icahn School of Medicine and director of the Center for Anatomy and Functional Morphology, and Eric Delson, PhD, director of the New York Consortium in Evolutionary Primatology or NYCEP, are also co-authors and are seasoned paleoanthropologists, each approaching their fifth decade of studying Neanderthals. Dr. Delson has published on various aspects of human evolution since the early 1970's.

Dr. Laitman states that this article is a significant contribution to the question of Neanderthal cold adaptation in the nasal region, especially in its identification of a different mosaic of features than those of cold-adapted modern humans. Dr. Laitman's body of work has shown that there are clear differences in the vocal tract proportions of these fossil humans when compared to modern humans. This current contribution has now identified potentially species-level differences in nasal structure and function.

Dr. Laitman said, "The strength of this new research lies in its taking the totality of the Neanderthal nasal complex into account, rather than looking at a single feature. By looking at the complete morphological pattern, we can conclude that Neanderthals are our close relatives, but they are not us."

Ian Tattersall, PhD, emeritus curator of the Division of Anthropology at the American Museum of Natural History, an expert on Neanderthal anatomy and functional morphology who did not participate in this study, stated, "Márquez and colleagues have carried out a most provocative and intriguing investigation of a very significant complex in the Neanderthal skull that has all too frequently been overlooked." Dr. Tattersall hopes that "with luck, this research will stimulate future research demonstrating once and for all that Homo neanderthalensis deserves a distinctive identity of its own."

The article in The Anatomical Record is entitled, "The Nasal Complex of Neanderthals: An Entry Portal to their Place in Human Ancestry." It is available online at: http://onlinelibrary.wiley.com/doi/10.1002/ar.23040/full.

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http://www.eurekalert.org/pub_releases/2014-11/uos-tcl111814.php

Training can lead to synesthetic experiences, study shows

A new study has shown for the first time how people can be trained to "see" letters of the alphabet as colours in a way that simulates how those with synaesthesia experience their world.

The University of Sussex research, published today (18 November 2014) in Scientific Reports, also found that the training might potentially boost IQ.

Synaesthesia is a fascinating though little-understood neurological condition in which some people (estimated at around 1 in 23) experience an overlap in their senses. They "see" letters as specific colours, or can "taste" words, or associate sounds with different colours.

A critical debate concerns whether the condition is embedded in our genes, or whether it emerges because of particular environmental influences, such as coloured-letter toys in infancy.

While the two possibilities are not mutually exclusive, psychologists at the University's Sackler Centre for Consciousness Science devised a nine-week training programme to see if adults without synaesthesia can develop the key hallmarks of the condition.

They found, in a sample study of 14, that not only were the participants able to develop strong letter-colour associations to pass all the standard tests for synaesthesia, most also experienced sensations such as letters seeming "coloured" or having individual personas (for instance, "x is boring", "w is calm").

One of the most surprising outcomes of the study was that those who underwent the training also saw their IQ jump by an average of 12 points, compared to a control group that didn't undergo training.

Dr Daniel Bor, who co-led the study with Dr Nicolas Rothen, says: "The main implication of our study is that radically new ways of experiencing the world can be brought about simply through extensive perceptual training.

"The cognitive boost, although provisional, may eventually lead to clinical cognitive training tools to support mental function in vulnerable groups, such as Attention Deficit Hyperactivity (ADHD) children, or adults starting to suffer from dementia."

Dr Rothen adds: "It should be emphasised that we are not claiming to have trained non synaesthetes to become genuine synaesthetes. When we retested our participants three months after training, they had largely lost the experience of 'seeing' colours when thinking about the letters. But it does show that synaesthesia is likely to have a major developmental component, starting for many people in childhood."

Adults can be trained to acquire synesthetic experiences', by Daniel Bor, Nicolas Rothen, David Schwartzman, Stephanie Clayton and Anil Seth, is published in Scientific Reports, on 18 November 2014 http://www.nature.com/srep/2014/141118/srep07089/full/srep07089.html

http://www.eurekalert.org/pub_releases/2014-11/uoc-clt111714.php

Calcium loss turning lakes to 'jelly'

New research on a number of Canadian lakes show that historical acid deposits as a result of industry have greatly reduced calcium levels in the water - dramatically impacting populations of calcium-rich plankton such as Daphnia water fleas that dominate these ecosystems.

Falling calcium levels mean Daphnia cannot get the nutrients they need to survive and reproduce, and are consequently consuming less food and becoming more susceptible to predators, leaving more algae for other organisms to feed on.

This has left a small jelly-clad organism called Holopedium to take advantage. Holopedium are plankton competitors of the Daphnia that use less calcium, as well as a jelly coat that affords them greater protection from predators.

Lakes across eastern Canada have seen Holopedium populations explode in the last thirty years; particularly in lakes in the province of Ontario that have seen a recent Eurasian invasion of the spiny water flea - which also favours hunting Daphnia, affording Holopedium even more room in these ecosystems to expand.

Researchers say the average population of these small invertebrate jellies in many Ontario lakes doubled between the mid-1980s and the mid-2000s. They warn that the increasing 'jellification' of Canada's lakes will prevent vital nutrients being passed up the food chain to fish stocks, as well as clogging filtration systems that help the lakes contribute drinking water to many residents in these areas.

The team used data from monthly surveys of lakes that recorded water chemistry and plankton populations for over 30 years, and used the latest statistical techniques to map all the cause-and-effect relationships in these ecosystems to determine that falling water calcium was causing the jelly boom. The results are published today in the journal Proceedings of the Royal Society B.

"As calcium declines, the increasing concentrations of jelly in the middle of these lakes will reduce energy and nutrient transport right across the food chain, and will likely impede the withdrawal of lake water for residential, municipal and industrial uses," said study co-author Dr Andrew Tanentzap, from the University of Cambridge's Department of Plant Sciences.

This is a Holopedium. **Credit: Michael Arts, Canada Centre for Inland WatersDepartment of Plant Sciences.**

"In Ontario, 20% of government-monitored drinking water systems now come from landscapes containing lakes with depleted calcium concentrations that favour Holopedium, and this is only set to increase."

Historically, a lot of acid was deposited throughout the northern hemisphere due to industrialisation. The acid displaced calcium from soil, says Tanentzap. Over a long period, this process pushed all the calcium out of drainage areas that feed these lakes, causing dramatic declines in the water calcium levels.

"Pollution control may have stopped acid deposits in the landscape, but it's only now that we are discovering the damage wasn't entirely reversed," he said.

Daphniids have a heavily calcified exoskeleton, so need much higher levels of calcium and phosphorous. In low calcium water, Daphnia are much more vulnerable to at least one key predator - the larval phantom midge, or Chaoborus - as their ability to produce defences such as larger bodies, stronger exoskeletons and projecting neck teeth is compromised. Additionally, the Daphniids phosphorous requirements mean they need to eat a lot more.

Holopedium, however, have no exoskeleton, and require only half the phosphorous of Daphnia and just one-tenth the amount of calcium, and the jelly capsule in which Holopedium are contained largely protects them from the predators that live on plankton of this size.

Calcium loss isn't the only bad news for Daphniids. The team suggest that climate change is causing oxygen decline deep in the lakes, creating better conditions and increasing populations of larval midges - the main predator of Daphnia.

The team also investigated how far back the jellification of these lakes began. By analysing sediment cores and fossil records, they show that Holopedium have been steadily increasing ever since around 1850 - a time of early industrialisation

and consequent acid deposit increases. But the process really gathered pace from the 1980s onwards. "It may take thousands of years to return to historic lake water calcium concentrations solely from natural weathering of surrounding watersheds," said Tanentzap.

"In the meanwhile, while we've stopped acid rain and improved the pH of many of these lakes, we cannot claim complete recovery from acidification. Instead, we many have pushed these lakes into an entirely new ecological state."

http://www.eurekalert.org/pub_releases/2014-11/uow-sgt111814.php

Scientists get to the heart of fool's gold as a solar material

As the installation of photovoltaic solar cells continues to accelerate, scientists are looking for inexpensive materials beyond the traditional silicon that can efficiently convert sunlight into electricity.

MADISON, Wis. - Theoretically, iron pyrite -- a cheap compound that makes a common mineral known as fool's gold -- could do the job, but when it works at all, the conversion efficiency remains frustratingly low. Now, a University of Wisconsin-Madison research team explains why that is, in a discovery that suggests how improvements in this promising material could lead to inexpensive yet efficient solar cells.

"We think we now understand why pyrite hasn't worked," says chemistry Professor Song Jin, "and that provides the hope, based on our understanding, for figuring out how to make it work. This could be even more difficult, but exciting and rewarding." Although most commercial photovoltaic cells nowadays are based on silicon, the light-collecting film must be relatively thick and pure, which makes the production process costly and energy-intensive, says Jin.

A film of iron pyrite -- a compound built of iron and sulfur atoms -- could be 1,000 times thinner than silicon and still efficiently absorb sunlight.

Like silicon, iron and sulfur are common elements in the Earth's crust, so solar cells made of iron pyrite could have a significant material cost advantage in large scale deployment. In fact, previous research that balanced factors like theoretical efficiency, materials availability, and extraction cost put iron pyrite at the top of the list of candidates for low-cost and large-scale photovoltaic materials.

In the current online edition of the Journal of the American Chemical Society, Jin and first author Miguel Cabán-Acevedo, a chemistry Ph.D. student, together with other scientists at UW-Madison, explain how they identified defects in the body of the iron pyrite material as the source of inefficiency. The research was supported by the U.S. Department of Energy.

In a photovoltaic material, absorption of sunlight creates oppositely charged carriers, called electrons and holes, that must be separated in order for sunlight to be converted to electricity. The efficiency of a photovoltaic solar cell can be judged by three parameters, Jin says, and the solar cells made of pyrite were almost totally deficient in one: voltage. Without a voltage, a cell cannot produce any power, he points out. Yet based on its essential parameters, iron pyrite should be a reasonably good solar material. "We wanted to know, why is the photovoltage so low," Jin says.

"We did a lot of different measurements and studies to look comprehensively at the problem," says Cabán-Acevedo, "and we think we have fully and definitively shown why pyrite, as a solar material, has not been efficient."

In exploring why pyrite was practically unable to make photovoltaic electricity, many researchers have looked at the surface of the crystals, but Cabán-Acevedo and Jin also looked inside. "If you think of this as a body, many have focused on the skin, but we also looked at the heart," says Cabán-Acevedo, "and we think the major problems lie inside, although there are also problems on the skin."

The internal problems, called "bulk defects," occur when a sulfur atom is missing from its expected place in the crystal structure. These defects are intrinsic to the material properties of iron pyrite and are present even in ultra-pure crystals. Their presence in large numbers eventually leads to the lack of photovoltage for solar cells based on iron pyrite crystals.

Science advances by comprehending causes, Jin says. "Our message is that now we understand why pyrite does not work. If you don't understand something, you must try to solve it by trial and error. Once you understand it, you can use rational design to overcome the obstacle. You don't have to stumble around in the dark."

http://scitechdaily.com/study-shows-calorie-restricting-diets-slow-aging/

Study Shows Calorie-Restricting Diets Slow Aging

Calorie-Restricting Diets Slow Aging

Neuroscientists show that calorie-reduced diets stop the normal rise and fall in activity levels of close to 900 different genes linked to aging and memory formation in mice.

The adage ‘you are what you eat’ has been around for years. Now, important new research provides another reason to be careful with your calories.

In a presentation prepared for the Society for Neuroscience annual meeting in Washington, D.C., on November 17, researchers say their experimental results, conducted in female mice, suggest how diets with fewer calories derived from carbohydrates likely deter some aspects of aging and chronic diseases in mammals, including humans.

“Our study shows how calorie restriction practically arrests gene expression levels involved in the aging phenotype — how some genes determine the behavior of mice, people, and other mammals as they get old,” says senior study investigator and NYU Langone neuroscientist, Stephen D. Ginsberg, PhD. Ginsberg cautions

that the study does not mean calorie restriction is the "fountain of youth," but that it does "add evidence for the role of diet in delaying the effects of aging and age-related disease."

While restrictive dietary regimens have been well-known for decades to prolong the lives of rodents and other mammals, their effects in humans have not been well understood. Benefits of these diets have been touted to include reduced risk of human heart disease, hypertension, and stroke, Ginsberg notes, but the widespread genetic impact on the memory and learning regions of aging brains has not before been shown. Previous studies, he notes, have only assessed the dietary impact on one or two genes at a time, but his analysis encompassed more than 10,000 genes.

Ginsberg, an associate professor at NYU Langone and its affiliated Nathan S. Kline Institute for Psychiatric Research, says the research "widens the door to further study into calorie restriction and anti-aging genetics."

For the study, female mice, which like people are more prone to dementia than males, were fed food pellets that had 30 percent fewer calories than those fed to other mice. Tissue analyses of the hippocampal region, an area of the brain affected earliest in Alzheimer's disease, were performed on mice in middle and late adulthood to assess any difference in gene expression over time.

Funding support for the study was provided primarily by the US National Institutes of Health. Corresponding federal grant numbers are RR029893, TR000038, GM007238, R01 AG043375, P01 AG014449, and P01 AG017617. Additional funding support was provided by Alzheimer's Association grant IIRG-12-237253.

Besides Ginsberg, other NYU Langone researchers involved in these experiments were lead study investigator Marissa Schafer, PhD; and co-investigators Igor Dolgalev, MS, and Adriana Heguy, PhD.

http://bit.ly/1uvCypW

People Tend to Run Marathons Before Big Birthdays

The search for achievement and meaning at the end of a decade increases suicide and cheating, too

By Shannon Palus

About to wrap up your 30s, 40s, or 50s? However arbitrary that age may be, you're probably taking stock of what you've accomplished in the past ten years. "The imminent approach of a new decade signals the end of one era and the beginning of another," writes a group of psychologists in a new paper.

All that reflection, they found, has consequences. It can lead to either adaptive or maladaptive behaviors: actions that increase meaning or ones that tend towards a "lol, nothing matters" outlook on life. It's similar to the way that the New Year brings on a slew of debauchery, diets and gym memberships.

In the adaptive behavior category, the authors looked at marathon runners. People on the brink of a big birthday—"9-enders," as the study authors call them—were much more likely to sign up to run a marathon. Those that were regular marathoners tended to run faster in their something-9 year—on average 2.3 percent faster than if they were something-7 years old.

As for maladaptive behavior, 9-enders were more likely to commit suicide. And 9-enders were more likely to be registered on a dating website that caters to people seeking an affair—nearly 20 percent more than if the ages were randomly distributed. (And, since dating websites do not verify ages, the authors also conducted a quick study to check if non-9-enders were more likely to lie about being a 9-ender than any other age. They weren't.)

"Although some of these effects were small," the authors write, "they occur in domains with consequential life outcomes." And if you're a 9-ender, the findings from another paper, on the meaning of meaning, are worth thinking about: "Satisfying one's needs and wants increased happiness but was largely irrelevant to meaningfulness. Happiness was largely present-oriented, whereas meaningfulness involves integrating past, present, and future."

http://www.eurekalert.org/pub_releases/2014-11/aha-sht110514.php

Speedy heart transplant for kids better than waiting for perfect match

Children receiving a heart transplant as soon as a suitable donor is available are predicted to have better quality-adjusted survival than children who wait for a donor to which they do not have antibodies

Children who receive a heart transplant as soon as a suitable donor is available are predicted to have better quality-adjusted survival - even if they have antibodies that may attack the new heart - than children who wait for a donor to which they do not have antibodies according to research presented at the American Heart Association's Scientific Sessions 2014.

When the costs of care while waiting for an urgent transplant are considered, transplantation with the first suitable heart is also cheaper than waiting for a better-matched organ, researchers said.

In the same way that a vaccine activates the body's immune response to fight off a virus, a donated organ can trigger antibodies to fight off foreign tissue. Because of the risk of severe rejection after transplantation, experts traditionally believed that children with these antibodies should wait for a heart that won't activate an antibody response.

But patients with the antibodies in their blood are at high risk of dying while waiting for a perfect match, said Brian Feingold, M.D., M.S., study lead author

and medical director of Pediatric Heart and Heart-Lung Transplantation at Children's Hospital of Pittsburgh of UPMC and associate professor of the University of Pittsburgh School of Medicine in Pennsylvania. He noted that as many as 20 percent of children waiting for a heart transplant may have antibodies. Researchers examined data of more than 2,700 children listed for transplant since 1999.Patients' average age was 5 years and 45 percent were female. More than half were Caucasian, 23 percent were African American and 15 percent were Hispanic. About half of the children were born with heart disease and all urgently needed a heart transplant.

Researchers compared 10-year survival after being listed for transplant using two opposing strategies: waiting for a donor heart to which the candidate does not have antibodies or taking the first suitable offer, regardless of potential problems that antibodies may pose. The study found that accepting the first suitable offer, regardless of antibody concerns, is predicted to:

increase survival from the time of listing by more than 1 year (adjusted for quality of life) as compared to waiting for transplantation based on antibody status.

cost an average $122,856 less than waiting for transplantation based on antibody status.

"Our analysis shows that denial of listing for transplant, solely on the basis of having too many antibodies, is unwarranted," Feingold said. "One of the next questions is whether low levels of antibodies identified using modern antibody detection techniques are clinically meaningful. Are they a harbinger of problems to come, or just a 'false positive' that potentially alters our care of patients with important effects on survival and costs of care?"

For their study, researchers obtained 1999-2009 patient data from the Organ Procurement and Transplantation Network. Cost data came from the Children's Hospital of Pittsburgh of UPMC and the public Healthcare Cost Utilization Project Kids' Inpatient Database.

Researchers were able to control for antibody status, wait-list time and wait-list survival, post-transplant survival in the presence or absence of a positive crossmatch, and costs. They didn't specifically examine rejection rates, nor did they examine treatments other than heart transplant or outcome among patients without antibodies. As of June 2013, nearly 3,500 patients were waiting for a heart transplant, according to American Heart Association statistics.

Co-authors are Steven A. Webber, M.B.Ch.B., M.R.C.P.; Cindy L. Bryce, Ph.D.; Heather E. Tomko, M.S.; Seo Y. Park, Ph.D.; William T. Mahle, M.D.; and Kenneth J. Smith, M.D.

Author disclosures are on the manuscript.

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http://www.eurekalert.org/pub_releases/2014-11/ez-ajb111914.php

A jettisoned black hole?

When the central black holes in merging galaxies combine, a "kick" launches the merged black hole on a wide orbit taking it far from the galaxy's core

In his general theory of relativity, Albert Einstein predicted that there are such things as gravitational waves. In fact, the very existence of these waves is the linchpin of the entire theory. Despite the great lengths that physicists have gone to in recent decades, however, they still have not managed to detect them directly with a measurement. This could largely be due to the fact that this requires a level of precision that it is practically impossible to achieve with today's measuring devices. Ultimately, it is all about measuring the tiniest of compressions and extensions of space which, according to Einstein's theory, arise when gravitational waves pass through it. And even using the high-precision measuring equipment of the future, only waves with a corresponding level of intensity may well be detectable, such as those formed during the fusion of two merging black holes.

If two galaxies head towards each other in space and eventually collide, they merge into one. The two supermassive black holes in the centre of the two galaxies also fuse. In this process, if the general theory of relativity holds true, gravitational waves are formed and spread out in space. If the black holes have unequal masses or are spinning at different speeds, the gravitational waves will be emitted asymmetrically - giving the fused black hole a "kick" that propels it in the opposite direction. In some cases, this recoil kick is relatively weak and the fused black hole drifts back into the centre. In other cases, however, the kick is strong enough to propel the black hole out of the galaxy entirely, where it will forever wander through the universe.

Remnant of a Collision Between Two Galaxies...

Astronomers have been searching for such recoiling black holes, but have not found any strong candidates yet. An international team of scientists including Kevin Schawinski, a professor at the Institute for Astronomy at ETH Zurich, and Michael Koss, a Swiss National Science Foundation Ambizione Fellow working with the Schawinski group, discovered an object that may in fact be a recoiling black hole. The object, named SDSS1133, lies around 90 million light years from Earth, which is nearby in astronomical terms. Researchers from the University of Hawaii, the University of Maryland, the Jet Propulsion Laboratory in Pasadena, California, the University of Arizona, the University of Copenhagen, the University of California, Berkeley, and the Ohio State University have also worked on the discovery.

The researchers first realized that SDSS1133 was a unique object last year, while observing it with a reflecting telescope at the Keck Observatory in Hawaii.

Comparisons with an astronomical map from 2001 showed that it was already ten times weaker last year than in 2001 - and although the object was visible on maps from the 1950s and 1990s, it could only be seen very weakly. SDSS1133 shone very brightly in 2001 but did not go completely dark afterwards, which showed that it cannot be a normal supernova - the life-ending explosion of a star - because supernovae tend to be detectable for only a few months before fading significantly. From a comparison of the wavelength spectrum of the light emitted by SDSS1133 and a nearby dwarf galaxy the scientists concluded that the object might be a black hole that belonged to this dwarf galaxy at one stage and was jettisoned out of it.

... Or One of the Longest-lived Supernovae?

And yet the researchers are far from certain, mainly because there is a second, more exotic possibility: SDSS1133 could be a new type of long-duration outburst before a supernova within a giant star. This giant star would have lost much of its mass in a series of eruptions over the course of at least 50 years before its final explosion.

Scientists have already observed stars changing in this fashion: Eta Carinae, one of the most massive stars in our own galaxy, briefly became the second-brightest star in the sky in 1843. If this type of activity were also the explanation for SDSS1133, that would make it the longest continuous outbursts ever observed before a supernova.

Answers on the Horizon

ETH scientists will have the opportunity to search for answers to these questions next year. Black holes and supernovae both emit ultraviolet light, but with differing wavelengths. The researchers have been allocated observation time with the Hubble Space Telescope in October 2015 in order to measure this spectrum more precisely.

Changes in the object's brightness in the coming years will also give scientists clues as to whether they are dealing with a jettisoned black hole or an exploding mega-star: for a recoiling black hole they expect to see variable brightness, whereas the brightness of a supernova explosion should generally decrease over time. "Whether SDSS1133 is a recoiling black hole or an exploding mega-star, we are observing something that has never before been seen in the universe", says Michael Koss.

And should they discover that the object is in fact a recoiling black hole, that would considerably increase the odds of one day being able to detect gravitational waves. The scientists estimate that the recoil, if confirmed, occurred around ten million years ago. Consequently, it is not this object in itself that would be important for the concrete measurement of gravitational waves, but rather the fact that it is existing. "Dwarf galaxies are very common," says Koss. "Therefore it would be highly probable that other recoil events would appear before too long. The hope is that we would be able to observe one near Earth and measure the gravitational waves."

ESA missions to search for gravitational waves

The European Space Agency (ESA) will use space probes and laser interferometers to detect gravitational waves in space during one of its next large-scale missions, "eLISA". The launch of the probes has been scheduled for 2034. However, the preparatory mission LISA Pathfinder is already due to blast off next year with a view to testing key technologies for eLISA. ETH Zurich is also involved in LISA Pathfinder.

Koss M, Blecha L, Mushotzky R, Hung CL, Veilleux S, Trakhtenbrot B, Schawinski K, Stern D, Smith N, Li Y, Man A, Filippenko AV, Mauerhan JC, Stanek K, Sanders D: An Unusually Persistent Transient in a Nearby Dwarf Galaxy, 2014, Monthly Notices of the Royal Astronomical Society 2014. 445: 515.

http://www.eurekalert.org/pub_releases/2014-11/pu-uso111914.php

Unique sense of 'touch' gives a prolific bacterium its ability to infect anything

New research has found that one of the world's most prolific bacteria manages to afflict humans, animals and even plants by way of a mechanism not before seen in any infectious microorganism -- a sense of touch.

This unique ability helps make the bacteria *Pseudomonas aeruginosa* ubiquitous, but it also might leave these antibiotic-resistant organisms vulnerable to a new form of treatment.

Pseudomonas is the first pathogen found to initiate infection after merely attaching to the surface of a host, Princeton University and Dartmouth College researchers report in the journal the *Proceedings of the National Academy of Sciences*. This mechanism means that the bacteria, unlike most pathogens, do not rely on a chemical signal specific to any one host, and just have to make contact with any organism that's ripe for infection.

The researchers found, however, that the bacteria could not infect another organism when a protein on their surface known as PilY1 was disabled. This suggests a possible treatment that, instead of attempting to kill the pathogen, targets the bacteria's own mechanisms for infection.

Corresponding author Zemer Gitai, a Princeton associate professor of molecular biology, explained that the majority of bacteria, viruses and other disease-causing agents depend on "taste," as in they respond to chemical signals unique to the hosts with which they typically co-evolved. *Pseudomonas*, however, through their sense of touch, are able to thrive on humans, plants, animals, numerous human-

made surfaces, and in water and soil. They can cause potentially fatal organ infections in humans, and are the culprit in many hospital-acquired illnesses such as sepsis. The bacteria are largely unfazed by antibiotics.

"*Pseudomonas*' ability to infect anything was known before. What was not known was how it's able to detect so many types of hosts," Gitai said. "That's the key piece of this research -- by using this sense of touch, as opposed to taste, *Pseudomonas* can equally identify any kind of suitable host and initiate infection in an attempt to kill it."

The researchers found that only two conditions must be satisfied for *Pseudomonas* to launch an infection: Surface attachment and "quorum sensing," a common bacterial mechanism wherein the organisms can detect that a large concentration of their kind is present. The researchers focused on the surface-attachment cue because it truly sets *Pseudomonas* apart, said Gitai, who worked with first author Albert Siryaporn, a postdoctoral researcher in Gitai's group; George O'Toole, a professor of microbiology and immunology at Dartmouth; and Sherry Kuchma, a senior scientist in O'Toole's laboratory.

To demonstrate the bacteria's wide-ranging lethality, Siryaporn infected ivy cells with the bacteria then introduced amoebas to the same sample; *Pseudomonas* immediately detected and quickly overwhelmed the single-celled animals. "The bacteria don't know what kind of host it's sitting on," Siryaporn said. "All they know is that they're on something, so they're on the offensive. It doesn't draw a distinction between one host or another."

When Siryaporn deleted the protein PilY1 from the bacteria's surface, however, the bacteria lost their ability to infect and thus kill the test host, an amoeba. "We believe that this protein is the sensor of surfaces," Siryaporn said. "When we deleted the protein, the bacteria were still on a surface, but they didn't know they were on a surface, so they never initiated virulence."

Because PilY1 is on a *Pseudomonas* bacterium's surface and required for virulence, it presents a comprehensive and easily accessible target for developing drugs to treat *Pseudomonas* infection, Gitai said. Many drugs are developed to target components in a pathogen's more protected interior, he said.

Kerwyn Huang, a Stanford University assistant professor of bioengineering, said that the research is an important demonstration of an emerging approach to treating pathogens -- by disabling rather than killing them.

"This work indicates that the PilY1 sensor is a sort of lynchpin for the entire virulence response, opening the door to therapeutic design that specifically disrupts the mechanical cues for activating virulence," said Huang, who is familiar with the research but had no role in it.

"This is a key example of what I think will become the paradigm in antivirals and antimicrobials in the future -- that trying to kill the microbes is not necessarily the best strategy for dealing with an infection," Huang said. "[The researchers'] discovery of the molecular factor that detects the mechanical cues is critical for designing such compounds."

Targeting proteins such as PilY1 offers an avenue for combating the growing problem of antibiotic resistance among bacteria, Gitai said. Disabling the protein in *Pseudomonas* did not hinder the bacteria's ability to multiply, only to infect. Antibiotic resistance results when a drug kills all of its target organisms, but leaves behind bacteria that developed a resistance to the drug. These mutants, previously in the minority, multiply at an astounding rate -- doubling their numbers roughly every 30 minutes -- and become the dominant strain of pathogen, Gitai said. If bacteria had their ability to infect disabled, but were not killed, the mutant organisms would be unlikely to take over, he said.

"I'm very optimistic that we can use drugs that target PilY1 to inhibit the whole virulence process instead of killing off bacteria piecemeal," Gitai said. "This could be a whole new strategy. Really what people should be doing is screening drugs that inhibit virulence but preserve growth. This protein presents a possible route by which to do that."

PilY1 also is found in other bacteria with a range of hosts, Gitai said, including Neisseria gonorrhoeae or the large bacteria genus Burkholderia, which, respectively, cause gonorrhea in humans and are, along with *Pseudomonas*, a leading cause of lung infection in people with cystic fibrosis. It is possible that PilY1 has a similar role in detecting surfaces and initiating infection for these other bacteria, and thus could be a treatment target.

Frederick Ausubel, a professor of genetics at Harvard Medical School, said that the research could help explain how opportunistic pathogens are able to infect multiple types of hosts. Recent research has revealed a lot about how bacteria initiate an infection, particularly via quorum sensing and chemical signals, but the question about how that's done across a spectrum of unrelated hosts has remained unanswered, said Ausubel, who is familiar with the research but had no role in it.

"A broad host-range pathogen such as *Pseudomonas* cannot rely solely on chemical cues to alert it to the presence of a suitable host," Ausubel said.

"It makes sense that *Pseudomonas* would use surface attachment as one of the major inputs to activating virulence, especially if attachment to surfaces in general rather than to a particular surface is the signal," he said. "There is probably an advantage to activating virulence only when attached to a host cell, and it is certainly possible that other broad host-range opportunistic pathogens utilize a similar strategy."

The paper, "Surface attachment induces Pseudomonas aeruginosa *virulence," was published online Nov. 10 by the* Proceedings of the National Academy of Sciences*. The work was supported by a National Institutes of Health Director's New Innovator Award (grant no. 1DP2OD004389); the National Science Foundation (grant no. 1330288); an NIH National Institute of Allergy and Infectious Diseases postdoctoral fellowship (no. F32AI095002) and grant (no. R37-AI83256-06); and the Human Frontiers in Science Program.*

http://www.eurekalert.org/pub_releases/2014-11/uow-irf112014.php

Imagination, reality flow in opposite directions in the brain

As real as that daydream may seem, its path through your brain runs opposite reality.

MADISON, Wis. - Aiming to discern discrete neural circuits, researchers at the University of Wisconsin-Madison have tracked electrical activity in the brains of people who alternately imagined scenes or watched videos.

"A really important problem in brain research is understanding how different parts of the brain are functionally connected. What areas are interacting? What is the direction of communication?" says Barry Van Veen, a UW-Madison professor of electrical and computer engineering. "We know that the brain does not function as a set of independent areas, but as a network of specialized areas that collaborate."

Van Veen, along with Giulio Tononi, a UW-Madison psychiatry professor and neuroscientist, Daniela Dentico, a scientist at UW-Madison's Waisman Center, and collaborators from the University of Liege in Belgium, published results recently in the journal NeuroImage. Their work could lead to the development of new tools to help Tononi untangle what happens in the brain during sleep and dreaming, while Van Veen hopes to apply the study's new methods to understand how the brain uses networks to encode short-term memory.

During imagination, the researchers found an increase in the flow of information from the parietal lobe of the brain to the occipital lobe - from a higher-order region that combines inputs from several of the senses out to a lower-order region. In contrast, visual information taken in by the eyes tends to flow from the occipital lobe - which makes up much of the brain's visual cortex - "up" to the parietal lobe.

"There seems to be a lot in our brains and animal brains that is directional, that neural signals move in a particular direction, then stop, and start somewhere else," says. "I think this is really a new theme that had not been explored."

The researchers approached the study as an opportunity to test the power of electroencephalography (EEG) - which uses sensors on the scalp to measure underlying electrical activity - to discriminate between different parts of the brain's network.

Brains are rarely quiet, though, and EEG tends to record plenty of activity not necessarily related to a particular process researchers want to study.

To zero in on a set of target circuits, the researchers asked their subjects to watch short video clips before trying to replay the action from memory in their heads. Others were asked to imagine traveling on a magic bicycle -- focusing on the details of shapes, colors and textures -- before watching a short video of silent nature scenes.

Using an algorithm Van Veen developed to parse the detailed EEG data, the researchers were able to compile strong evidence of the directional flow of information.

"We were very interested in seeing if our signal-processing methods were sensitive enough to discriminate between these conditions," says Van Veen, whose work is supported by the National Institute of Biomedical Imaging and Bioengineering. "These types of demonstrations are important for gaining confidence in new tools."

http://www.eurekalert.org/pub_releases/2014-11/uoz-bci111914.php

Business culture in banking industry favors dishonest behavior

In the past years, there have often been cases of fraud in the banking industry, which have led to a considerable loss of image for banks.

Are bank employees by nature less honest people? Or does the business culture in the banking sector favor dishonest behavior? These questions formed the basis for a new study by Alain Cohn, Ernst Fehr, and Michel Maréchal from the Department of Economics at the University of Zurich. Their results show that bank employees are in principle not more dishonest than their colleagues in other industries. The findings indicate, however, that the business culture in the banking sector implicitly favors dishonest behavior. The results suggest that the implementation of a healthy business culture is of great importance in order to restore trust in the banking industry.

Occupational Norms Implicitly Favor Dishonest Behavior in Bankers

The scientists recruited approximately 200 bank employees, 128 from a large international bank and 80 from other banks. Each person was then randomly assigned to one of two experimental conditions. In the experimental group, the participants were reminded of their occupational role and the associated behavioral norms with appropriate questions. In contrast, the subjects in the control group were reminded of their non-occupational role in their leisure time and the associated norms. Subsequently, all participants completed a task that would allow them to increase their income by up to two hundred US dollars if they behaved dishonestly. The result was that bank employees in the experimental

group, where their occupational role in the banking sector was made salient, behaved significantly more dishonestly.

A very similar study was then conducted with employees from various other industries. In this case as well, either the employees' occupational roles or those associated with leisure time were activated. Unlike the bankers, however, the employees in these other industries were not more dishonest when reminded of their occupational role. "Our results suggest that the social norms in the banking sector tend to be more lenient towards dishonest behavior and thus contribute to the reputational loss in the industry," says Michel Maréchal, Professor for Experimental Economic Research at the University of Zurich.

A Change in Norms is Needed in the Banking Industry

Social norms that are implicitly more lenient towards dishonesty are problematic, because the people's trust in bank employees' behavior is of great importance for the long-term stability of the financial services industry. Alain Cohn, who recently joined the Booth School of Business at the University of Chicago as a postdoctoral scholar, suggests concrete measures that could counteract the problem: "The banks could encourage honest behavior by changing the industry's implicit social norms. Several experts and supervisory authorities suggest, for example, that bank employees should take a professional oath, similar to the Hippocratic Oath for physicians." If an oath like this were supported with a corresponding training program in ethics and appropriate financial incentives, this could lead bank employees to focus more strongly on the long-term, social effects of their behavior instead of concentrating on their own, short-term gains.

Alain Cohn, Ernst Fehr and Michel André Maréchal. Business culture and dishonesty in the banking industry. Nature. November 19, 2014. doi: 10.1038/nature13977

http://www.eurekalert.org/pub_releases/2014-11/si-ssd111714.php

Salk scientists deliver a promising one-two punch for lung cancer

Scientists at the Salk Institute have discovered a powerful one-two punch for countering a common genetic mutation that often leads to drug-resistant cancers.

LA JOLLA - The dual-drug therapy--with analogs already in use for other diseases--doubled the survival rate of mice with lung cancer and halted cancer in pancreatic cells.

Lung cancer, which affects nonsmokers as well as smokers, is the most common cancer worldwide, causing 1.6 million deaths a year, far more than pancreatic, breast and colon cancer combined. About 30 percent of the most common type of lung cancer (non-small) contains a mutation in a gene called KRAS. This mutation can also lead to hard-to-treat cancer in the pancreas, thyroid and colon.

"There really have been no effective treatments to target the KRAS mutation so far," says Inder Verma, a professor in the Laboratory of Genetics and American Cancer Society Professor of Molecular Biology. "We found a drug combination that successfully targets KRAS and stops tumor growth in the mouse model."

The new discovery, detailed November 19 in Science Translational Medicine, shows how the two-pronged attack successfully hindered KRAS and other cellular processes to halt or shrink tumor growth.

When activated, mutated KRAS clings to cell membranes and recruits proteins to ramp up cancer growth. Researchers have developed drugs to disable enzymes that tether KRAS to the cell membrane, but these drugs typically ended up being toxic because those enzymes are needed in the body for normal functions.

"The Achilles' heel of KRAS is its movement to the membrane," says Verma, who is also holder of Salk's Irwin and Joan Jacobs Chair in Exemplary Life Science.

The researchers took a new approach to targeting this membrane interaction when they noticed that a drug called Zometa, typically used to stop the breakdown and growth of cells in bone disease, also interfered with cell membrane interactions. In previous work, the team added carbon chains to a molecule similar to Zometa, to create a lipophilic bisphosphonate (BP) that blocked KRAS from attaching to the cell membrane. "For the first time, we had the ability to interfere with KRAS without being completely toxic," says Verma.

This, however, wasn't enough. Tumors were still proliferating, in part because the new BP led to failed attempts of a process called autophagy, where cells, under stress, self-destruct and break down into nutrients that can be used by other cells. Autophagy can be both good and bad in fighting cancer: in some cases, autophagy prompts cancer cells to die; in other settings, it creates a cellular environment that helps tumors thrive. With the BP treatment, cells began the process of autophagy but failed, leading to junk protein accumulation and an inflamed environment that helped the tumors to survive.

But, as demonstrated in the new work, when the researchers added a chemical called rapamycin, cells were able to carry out autophagy successfully and prevented tumor cells from proliferating. Rapamycin, discovered in the 1970s, is used in the clinic for preventing organ rejection and has also been linked to anti-cancer effects.

"We found if we also activated autophagy--with the rapamycin--and combine it with the inhibitor of the cell membrane--the BP--there were significant cell deaths in the tumors," says Yifeng Xia, Salk researcher and first author of the new work. When they injected the combination in mouse lung tumors, tumors shrunk or stopped growing. The study also found that a pancreatic cancer cell line responded to the dual treatment. Next, the team plans to test toxicity of the new

BP. The group is also working with the University of California, San Diego, Moores Cancer Center to design human clinical trials to test the dual therapy. "Those two drugs have not been used together as far as we know for KRAS-related cancer treatment," adds Xia. "We are excited about the potential and that these molecules are already being used in clinical trials in some form."

In addition to Verma and Xia, authors on the paper included Shen Shen, Narayana Yeddula, Wolfgang Fischer and William Low of the Salk Institute; Yi-Liang Liu, Wei Zhu, Francisco Guerra and Eric Oldfield of the University of Illinois at Urbana-Champaign; and Yonghua Xie, Xiaoying Zhou and Yonghui Zhang of the Tsinghua University.

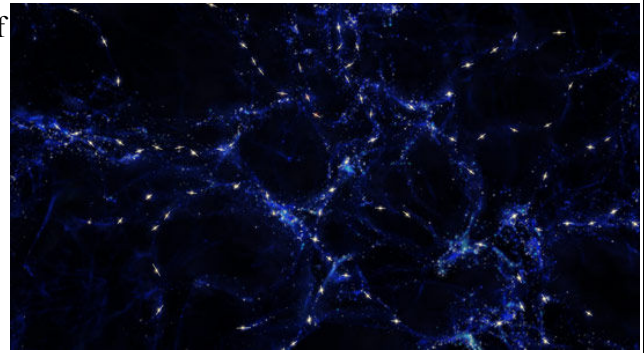
The work was funded by the National Institutes of Health, Ipsen Biomeasure, the H.N. and Frances C. Berger Foundation and the Leona M. and Harry B. Helmsley Charitable Trust.

http://bit.ly/1r0xS0y

VLT Reveals Alignment of Quasars Across Billions of Light-Years

Using data from ESO's Very Large Telescope, astronomers have discovered alignments between supermassive black hole axes separated by billions of light-years.

New observations with ESO's Very Large Telescope (VLT) in Chile have revealed alignments over the largest structures ever discovered in the Universe. A European research team has found that the rotation axes of the central supermassive black holes in a sample of quasars are parallel to each other over distances of billions of light-years. The team has also found that the rotation axes of these quasars tend to be aligned with the vast structures in the cosmic web in which they reside.



This artist's impression shows schematically the mysterious alignments between the spin axes of quasars and the large-scale structures that they inhabit that observations with ESO's Very Large Telescope have revealed. These alignments are over billions of light-years and are the largest known in the Universe. The large-scale structure is shown in blue and quasars are marked in white with the rotation axes of their black holes indicated with a line.

Quasars are galaxies with very active supermassive black holes at their centers. These black holes are surrounded by spinning discs of extremely hot material that is often spewed out in long jets along their axes of rotation. Quasars can shine more brightly than all the stars in the rest of their host galaxies put together.

A team led by Damien Hutsemékers from the University of Liège in Belgium used the FORS instrument on the VLT to study 93 quasars that were known to form huge groupings spread over billions of light-years, seen at a time when the Universe was about one third of its current age.

"The first odd thing we noticed was that some of the quasars' rotation axes were aligned with each other — despite the fact that these quasars are separated by billions of light-years," said Hutsemékers.

The team then went further and looked to see if the rotation axes were linked, not just to each other, but also to the structure of the Universe on large scales at that time.

When astronomers look at the distribution of galaxies on scales of billions of light-years they find that they are not evenly distributed. They form a cosmic web of filaments and clumps around huge voids where galaxies are scarce. This intriguing and beautiful arrangement of material is known as large-scale structure.

The new VLT results indicate that the rotation axes of the quasars tend to be parallel to the large-scale structures in which they find themselves. So, if the quasars are in a long filament then the spins of the central black holes will point along the filament. The researchers estimate that the probability that these alignments are simply the result of chance is less than 1%.

"A correlation between the orientation of quasars and the structure they belong to is an important prediction of numerical models of evolution of our Universe. Our data provide the first observational confirmation of this effect, on scales much larger that what had been observed to date for normal galaxies," adds Dominique Sluse of the Argelander-Institut für Astronomie in Bonn, Germany and University of Liège.

The team could not see the rotation axes or the jets of the quasars directly. Instead they measured the polarization of the light from each quasar and, for 19 of them, found a significantly polarized signal. The direction of this polarization, combined with other information, could be used to deduce the angle of the accretion disc and hence the direction of the spin axis of the quasar.

"The alignments in the new data, on scales even bigger than current predictions from simulations, may be a hint that there is a missing ingredient in our current models of the cosmos," concludes Dominique Sluse.

***Publication**: D. Hutsemekers, et al., "Alignment of quasar polarizations with large-scale structures," A&A, Volume 572, December 2014, A18; doi:10.1051/0004-6361/201424631*

***PDF Copy of the Study**: Alignment of quasar polarizations with large-scale structures*

http://nyti.ms/11qmUoT

Viruses as a Cure

New research hints that some viruses may actually be keeping us healthy

Carl Zimmer

When we talk about viruses, usually we focus on the suffering caused by Ebola, influenza and the like. But our bodies are home to trillions of viruses, and new research hints that some of them may actually be keeping us healthy.

"Viruses have gotten a bad rap," said Ken Cadwell, an immunologist at New York University School of Medicine. "They don't always cause disease."

Dr. Cadwell stumbled by accident onto the first clues about the healing power of viruses. At the time, he was studying the microbiome, the community of 100 trillion microbes living in our bodies. Scientists have long known that the microbiome is important to our health.

One of its crucial functions is ensuring that our intestines develop normally. In a healthy gut, the inner wall is lined with a dense mat of fingerlike projections called villi. When scientists raise germ-free mice in sterile cages, their intestinal villi turn out to be sparse and thin.

Germ-free mice also fail to develop a normal supply of the immune cells nestled in the lining of the gut, which attack pathogens but not harmless microbes. As a result, the germ-free mouse's gut becomes vulnerable to injuries and infections.

In order for the gut to develop normally, an intimate chemical conversation must take place between the microbiome and ho
st cells. Genetic mutations can disrupt this tête-à-tête, causing immune cells in the gut to attack beneficial bacteria as if they were enemies. A number of experiments suggest that ailments like inflammatory bowel disease may be the result of discord between microbes and their hosts.

Dr. Cadwell set out to understand exactly how it happens. He and his colleagues created mice with a genetic mutation known to increase the risk of inflammatory bowel disease in humans. Then the researchers examined the animals' immune cells and guts.

In the midst of his research, Dr. Cadwell moved his mice to a new lab. And something odd happened: The move cured the mice.

Dr. Cadwell eventually figured out that the two labs differed in one important way. The old one was contaminated with a virus called murine norovirus, and the new one was virus-free.

Murine norovirus is related to the nasty human strain that causes vomiting and diarrhea - and has ruined so many cruises. The virus is harmless in healthy mice, but Dr. Cadwell found that when he gave it to his mutant mice, it triggered inflammatory bowel disease.

Dr. Cadwell was struck by how much the virus mimicked the microbiome: harmless in normal mice but triggering disease those with mutant genes. He wondered if the similarity went even further, if the virus served a purpose.

After setting up a new lab at N.Y.U. in 2011, Dr. Cadwell launched an experiment to find out.

When he and his colleagues infected germ-free mice with murine norovirus, the animals developed intestines and an immune system that were fairly normal. "It's just one virus, but it's doing many of the things that an entire community of bacteria is doing," said Dr. Cadwell.

Dr. Cadwell wondered if viruses can restore the gut when it has been disturbed in other ways. Heavy doses of antibiotics, which kill off much of the microbiome, can lead to drastic changes in the gut.

Some villi die, and the population of immune cells drops. But as bacteria return to the gut, the damage gets fixed.

To see whether viruses have a similar effect, Dr. Cadwell and his colleagues gave antibiotics to normal adult mice for two weeks. When they infected the mice with murine norovirus, their guts returned to normal. Dr. Cadwell and his colleagues published the results of their experiment in Nature on Thursday.

Kristine Wylie, a research instructor of pediatrics at Washington University School of Medicine who was not involved in the research, speculated that in real life, certain viruses might be important partners with the microbiome.

"It isn't hard to imagine that the viral exposures we get as children are important to our development," she said.

In recent years, medical researchers have been investigating how to harness the microbiome to attack diseases. But Dr. Cadwell doesn't expect we'll be taking pills full of viruses to treat immune disorders. In some people, ordinarily harmless viruses might turn out to be dangerous.

Instead, Dr. Cadwell wants to figure out the molecular tricks thatthe viruses are using to improve the health of their hosts. "You're probably going to come up with things you never imagined," he said.

At the moment, Dr. Cadwell doesn't know for sure how the viruses nurture the mice, but he and his colleagues have found one important clue.

When they prevented germ-free mice from making a receptor on the surface of their cells, infection with norovirus didn't lead to an improvement in their guts.

That receptor only latches onto one type of molecule. It's called Type 1 interferon, and it's produced by cells when they're invaded by viruses.

Taken together, findings suggest that some viruses may be working to keep us healthy. "They did a very good job of starting to crack that nut," said Julie K.

Pfeiffer, a virologist at University of Texas Southwestern Medical Center who was not involved in the new study.

David T. Pride, a microbiologist at the University of California, San Diego, said that the new study would spur other researchers to see if they can find similar results in humans.

"The hunt for natural viruses that are beneficial to our immune systems has officially begun," he said.

http://www.eurekalert.org/pub_releases/2014-11/uol-hdc112014.php

Hand dryers can spread bacteria in public toilets, research finds

Modern hand dryers are much worse than paper towels when it comes to spreading germs, according to new University of Leeds research.

Scientists from the University of Leeds have found that high-powered 'jet-air' and warm air hand dryers can spread bacteria in public toilets.

Airborne germ counts were 27 times higher around jet air dryers in comparison with the air around paper towel dispensers.

The study shows that both jet and warm air hand dryers spread bacteria into the air and onto users and those nearby.

The research team, led by Professor Mark Wilcox of the School of Medicine, contaminated hands with a harmless type of bacteria called Lactobacillus, which is not normally found in public bathrooms. This was done to mimic hands that have been poorly washed.

Subsequent detection of the Lactobacillus in the air proved that it must have come from the hands during drying. The experts collected air samples around the hand dryers and also at distances of one and two metres away.

Air bacterial counts close to jet air dryers were found to be 4.5 times higher than around warm air dryers and 27 times higher compared with the air when using paper towels.

Next to the dryers, bacteria persisted in the air well beyond the 15 second hand-drying time, with approximately half (48%) of the Lactobacilli collected more than five minutes after drying ended. Lactobacilli were still detected in the air 15 minutes after hand drying.

Professor Wilcox said: "Next time you dry your hands in a public toilet using an electric hand dryer, you may be spreading bacteria without knowing it. You may also be splattered with bugs from other people's hands.

"These findings are important for understanding the ways in which bacteria spread, with the potential to transmit illness and disease."

The research, funded by the European Tissue Symposium, was published in the Journal of Hospital Infection and presented at the Healthcare Infection Society (HIS) International Conference in Lyon, France.

http://www.eurekalert.org/pub_releases/2014-11/jhm-sof112014.php

Study: Obesity fuels silent heart damage

Evidence of heart muscle damage seen even among symptom-free people

Fast facts:

The study shows that obesity leads to subclinical heart muscle injury and increases the risk for heart failure even among people without overt heart disease and independently of other cardiovascular risk factors such as diabetes, high blood pressure and high cholesterol.

The silent heart damage was detected by using an ultrasensitive test that measures the levels of a protein released by the cells of the heart muscle during injury.

The findings suggest that obesity is an independent driver of heart muscle damage, and that obese individuals, even when free of cardiac symptoms, warrant vigilant monitoring.

Using an ultrasensitive blood test to detect the presence of a protein that heralds heart muscle injury, researchers from Johns Hopkins and elsewhere have found that obese people without overt heart disease experience silent cardiac damage that fuels their risk for heart failure down the road.

The findings of the federally funded study, published ahead of print in the Journal of the American College of Cardiology: Heart Failure, challenge the commonly held belief that much of the cardiovascular disease seen in severely overweight people is driven by diabetes and high blood pressure, both well-known cardiac risk factors and both occurring frequently among the obese.

Specifically, the research showed that obese people had elevated levels of a heart enzyme known as troponin T, released by injured heart muscle cells. Increases in levels of this enzyme corresponded to increases in people's body mass index (BMI) -- a measure of body fat based on a person's weight-to-height ratio. Levels of the enzyme rose proportionally as BMI went up.

Troponin T is the gold standard for diagnosing acute or recent heart attacks and is widely used in emergency rooms to test patients with chest pain and other symptoms suggestive of a heart attack. The test used in the current study works in much the same way, but is calibrated to detect troponin levels far below the ranges of the clinical test for diagnosing a heart attack.

"Obesity is a well-known 'accomplice' in the development of heart disease, but our findings suggest it may be a solo player that drives heart failure independently of other risk factors that are often found among those with excess weight," says lead investigator Chiadi Ndumele, M.D., M.H.S., an assistant professor at the Johns Hopkins Ciccarone Center for the Prevention of Heart Disease. "The direct relationship we found between obesity and subclinical heart damage is quite

potent and truly concerning from a public health standpoint given the growing number of obese people in the United States and worldwide."

For the study, investigators measured the BMIs and cardiac troponin levels of more than 9,500 heart disease-free men and women, aged 53 to 75, living in Maryland, Mississippi, North Carolina and Minnesota. The researchers then tracked the participants' health for more than 12 years. During the follow-up, 869 people developed heart failure.

People who were severely obese -- those with a BMI above 35 -- had more than twice the risk of developing heart failure, compared with people of normal weight, the researchers found. That risk rose incrementally with BMI, growing by 32 percent for every five-unit increase in BMI. Thus, a 6-foot, 225-pound man with a BMI of 30 was 32 percent more likely to develop heart failure than a 6-foot, 188-pound man with a BMI of 25. All people with elevated troponin levels, regardless of BMI, had higher risk of developing heart failure over a decade. In other words, extra weight and high troponin each independently signaled higher heart disease risk.

When the researchers calculated the combined effects of elevated troponin and severe obesity, the predictive power was striking. Severely obese people with elevated troponin levels were nine times more likely to develop heart failure than people with normal weight and undetectable troponin levels. The elevated risk persisted even when investigators accounted for other possible causes of heart damage, including diabetes, hypertension and high cholesterol.

Public health experts deem heart failure -- a condition in which the heart muscle doesn't pump efficiently -- a looming epidemic. The disease has been on a steady rise and is expected to affect one in five adults by 2030.

Ndumele and team say the findings underscore the dangers of obesity and should be heeded as an alarm bell for clinicians to monitor their obese patients rigorously for emerging sings heart disease.

"These results are a wake-up call that obesity may further fuel the growing rate of heart failure, and clinicians who care for obese people should not be lulled into a false sense of security by the absence of traditional risk factors, such as high cholesterol, diabetes and hypertension," says Roger Blumenthal, M.D., director of the Johns Hopkins Ciccarone Center for the Prevention of Heart Disease. "Obese people, even when free of cardiovascular symptoms, should be monitored for the earliest signs of heart failure and counseled on ways to improve their lifestyle habits."

The investigators say their next step is to study the precise mechanism by which obesity causes subclinical heart muscle damage, and whether reduction in weight would lower the risk for heart failure.

The research was funded by the National Heart, Lung and Blood Institute.

Other Johns Hopkins investigators involved in the study included Josef Coresh, Mariana Lazo and Elizabeth Selvin. Other institutions involved in the study included Baylor College of Medicine, the University of Minnesota, the Michael E. DeBakey VA Medical Center in Houston and the Houston Methodist DeBakey Heart & Vascular Center.

http://www.eurekalert.org/pub_releases/2014-11/wtsi-bwi111914.php

Brain-dwelling worm in UK man's head sequenced

Tapeworm removed from UK resident's brain reveals genetic secrets of an elusive Far East parasite

For the first time, the genome of a rarely seen tapeworm has been sequenced. The genetic information of this invasive parasite, which lived for four years in a UK resident's brain, offers new opportunities to diagnose and treat this invasive parasite.

The tapeworm, Spirometra erinaceieuropaei, has been reported only 300 times worldwide since 1953 and has never been seen before in the UK. The worm causes sparganosis: inflammation of the body's tissues in response to the parasite. When this occurs in the brain, it can cause seizures, memory loss and headaches. The worm's rarity means that little is known about its complex lifecycle and biology, however it is thought that people may be infected by accidently consuming tiny infected crustaceans from lakes, eating raw meat from reptiles and amphibians, or by using a raw frog poultice - a Chinese remedy to calm sore eyes.

Before the 1cm-long parasite was diagnosed and successfully removed by surgery, it had travelled 5cm from the right side of the brain to the left. The tapeworm was placed on a histology slide by the hospital to confirm the clinical diagnosis. The patient is now systemically well.

"The clinical histology slide offered us a great opportunity to generate the first genome sequence of this elusive class of tapeworms," says Dr Hayley Bennett, first author of the study from the Wellcome Trust Sanger Institute. "However, we only had a minute amount of DNA available to work with - just 40 billionths of a gram. So we had to make difficult decisions as to what we wanted to find out from the DNA we had."

To identify the exact species of worm, the researchers sequenced one particular gene, the so-called "barcode of life". Fortunately for the patient, the gene's DNA sequence revealed that the parasite was the more benign of the two sparganosis-causing worm species. Remarkably, the team also were able to generate sufficient DNA sequence data using standard next-generation sequencing techniques to piece together a draft genome. This is now being used to investigate known and potential treatment targets, which may help patients in the future.

"We did not expect to see an infection of this kind in the UK, but global travel means that unfamiliar parasites do sometimes appear," says Dr Effrossyni Gkrania-Klotsas, study author from the Department of Infectious Disease, Addenbrooke's NHS Trust. "We can now diagnose sparganosis using MRI scans, but this does not give us the information we need to identify the exact tapeworm species and its vulnerabilities. Our work shows that, even with only tiny amounts of DNA from clinical samples, we can find out all we need to identify and characterise the parasite.

"This emphasises just how important a global database of worm genomes is to allow us to identify the parasite and determine the best course of treatment. Additionally, this information can be paired with our work in global travellers' infection to give additional insights in what infections other patients can get in specific destinations. We are really lucky to be able to work closely with such an excellent facility as the Wellcome Trust Sanger Institute."

Spirometra erinaceieuropaei's genome is 1.26Gb long, making it ten times larger than other tapeworm genomes and one-third the size of the human genome. Some of this seems to come from an increase in the number of genes that may help the parasite to break up proteins and invade its host, coupled with the fact that the genome is much more repetitive than other tapeworm genomes.

The team also used this draft sequence to look for similarities and differences from other, previously sequenced, tapeworm species in the GeneDB pathogen database. This has revealed more about Spirometra erinaceieuropaei's biology than ever before. For example, the worm has a large selection of molecular motors for moving proteins around the cell, which could underpin the large changes in body shape and environmental adaptions that the worm undergoes during its complicated lifecycle.

"For this uncharted group of tapeworms, this is the first genome to be sequenced and has allowed us to make some predictions about the likely activity of known drugs," says Dr Matt Berriman, senior author and member of Faculty of the Sanger Institute. "The genome sequence suggests that the parasite is naturally resistant to albendazole - an existing anti-tapeworm drug. However, many new drug targets that are being explored for other tapeworms are present in this parasite and could offer future clinical possibilities."

Because such an elusive worm is rare, discovering targets in the genome to existing licensed drugs could prove to be the best way to treat this rare disease. These data contribute to the growing global database for identifying parasites and parasite provenance and will serve as a resource for identifying new treatments for sparganosis.

http://www.bbc.com/news/health-30138097

Eye specialists call for NHS to use Avastin

A drug that prevents elderly people losing their sight should be routinely available on the NHS, says the Royal College of Ophthalmologists.

By Adam Brimelow Health Correspondent, BBC News

Avastin has been found in clinical trials to be safe and effective for patients with wet Age-Related Macular Degeneration (AMD), a major cause of sight loss in older people. The Royal College says switching to the drug could save the NHS £100m. Avastin is cheaper than the officially approved treatment, Lucentis. Both drugs are made by Roche - but Lucentis is marketed by Novartis in the UK.

Effective and safe

Lucentis typically costs about £700 for an injection, but the price for Avastin is about £70. Recent studies have concluded Avastin is just as effective and safe as Lucentis. Doctors can prescribe it "off-label", but they are only supposed to do that if there is no suitable licensed drug.

Writing in the British Medical Journal, experts from the Royal College say regulators should find a way of getting round what they call the "bureaucratic hurdles" that prevent its use, and called for the General Medical Council and National Institute for Health and Care Excellence.

"Without unequivocal GMC and NICE support, ophthalmologists are understandably concerned that they may be assuming unacceptable personal liability by using an unlicensed drug when a licensed alternative exists," they write.

Hospital eye services are struggling to cope with demand, they warn. Consequently, patients may not be getting treatment when they need it and not getting the best results. The money saved by switching to bevacizumab (Avastin) could facilitate investment in these services. "Either the regulators must find a way to license a drug without the sponsorship of the company that owns it or NICE must find a way to consider an off-label drug that is not being submitted for appraisal by its owners."

'Proper protection'

Cathy Yelf from the Macular Society said it agreed with the Royal College's view, and had been campaigning for for regulators to carry out an appraisal of Avastin for use in ophthalmology since 2010.

"We are aware that some Clinical Commissioning Groups are looking at ways of using Avastin. However, it is individual doctors who are legally accountable if an unlicensed drug is prescribed. It is not right that clinicians should be pressurised by the NHS to use Avastin without proper legal protection."

In a statement, Novartis, which markets Lucentis in the UK, said it was aware of the BMJ editorial. "Lucentis and Avastin are different molecules. Each was approved by EU regulatory authorities for different usages. Lucentis is approved for use in the eye. Avastin is intended for use with cancer patients. These are real differences that are clearly established and recognized by regulatory authorities." The company said patient safety was of paramount importance.

"Novartis believes that patients in the UK deserve access to medicines which are prescribed and used according to medical need and approved indications."

A spokesperson for the Department of Health in England said:

"Age-related macular degeneration is a very serious condition and there are already other licensed and NICE-recommended drugs available to treat this condition. Avastin is not licensed for this purpose and only the manufacturer is able to apply for a new licence. "Doctors are free to prescribe unlicensed medicines and licensed products off label if they feel they are clinically appropriate for their patients."

http://nyti.ms/1tpRKEG

F.D.A. Approves Hysingla, a Powerful Painkiller

The Food and Drug Administration on Thursday approved a powerful long-acting opioid painkiller, alarming some addiction experts who fear that its widespread use may contribute to the rising tide of prescription drug overdoses.

By RONI CARYN RABIN NOV. 20, 2014

The new drug, Hysingla, and another drug approved earlier this year, Zohydro, contain pure hydrocodone, a narcotic, without the acetaminophen used in other opioids. But Hysingla is to be made available as an "abuse-deterrent" tablet that cannot easily be broken or crushed by addicts looking to snort or inject it.

Nearly half of the nation's overdose deaths involved painkillers like hydrocodone and oxycodone, according to a 2010 study by the Centers for Disease Control and Prevention. More than 12 million people used prescription painkillers for nonmedical reasons that year, according to the study.

Prescription opioid abuse kills more adults annually than heroin and cocaine combined, and sends 420,000 Americans to emergency rooms every year, according to the C.D.C.

Hysingla, however, will not be not abuse-proof, said officials at the F.D.A. and the drug's manufacturer, Purdue Pharma. Its extended-release formulation, a pill to be taken once every 24 hours by patients requiring round-the-clock pain relief, will contain as much as 120 milligrams of hydrocodone.

The F.D.A. warned that doses of 80 milligrams or more "should not be prescribed to people who have not previously taken an opioid medication," but officials described the abuse-deterrent formulation as a step forward.

"For patients who benefit from hydrocodone alone for the treatment of pain severe enough to need an opioid, this offers the advantage of once-a-day dosing in a formulation that we expect will reduce abuse and misuse," said Dr. Douglas Throckmorton, deputy center director for regulatory programs at the F.D.A.

An official with Purdue Pharma said concerns about prescription-painkiller deaths were what motivated the company to develop abuse-deterrent products.

The tablets are hard and difficult to crush, and when mixed with water or other fluids, they become a "gelatinous, gooey mass that doesn't pull into a syringe easily," said Dr. David Haddox, the company's chief of health policy.

"This is coming on to a market that is currently flooded with products that do not have abuse-deterrent options," added Raul Damas, the company's spokesman.

Dr. Andrew Kolodny, the chief medical officer at Phoenix House, a group of nonprofit addiction-treatment centers, said he was disturbed by the drug's approval and disappointed that the F.D.A. did not seek input from an advisory committee of experts.

Dr. Kolodny said that addicts knew how to break down abuse-deterrent products for oral use, and that the 120-milligram tablets were particularly dangerous because they "pack an enormous amount of hydrocodone."

The F.D.A. approved Zohydro last year despite the recommendation of its own expert advisory committee, which had voted against approval.

In July, the F.D.A. approved another Purdue Pharma abuse-deterrent painkiller, Targiniq, without review by an expert advisory committee. Targiniq contains oxycodone and naloxone.

http://bit.ly/1r1vS8e

Saturn's calming nature keeps Earth friendly to life

Earth's comfortable temperatures may be thanks to Saturn's good behaviour. If the ringed giant's orbit had been slightly different, Earth's orbit could have been wildly elongated, like that of a long-period comet.

16:56 21 November 2014 by Jeff Hecht

Our solar system is a tidy sort of place: planetary orbits here tend to be circular and lie in the same plane, unlike the highly eccentric orbits of many exoplanets.

Elke Pilat-Lohinger of the University of Vienna, Austria, was interested in the idea that the combined influence of Jupiter and Saturn – the solar system's heavyweights – could have shaped other planets' orbits. She used computer models to study how changing the orbits of these two giant planets might affect the Earth.

Earth's orbit is so nearly circular that its distance from the sun only varies between 147 and 152 million kilometres, or around 2 per cent about the average. Moving Saturn's orbit just 10 percent closer in would disrupt that by creating a resonance

– essentially a periodic tug – that would stretch out the Earth's orbit by tens of millions of kilometres. That would result in the Earth spending part of each year outside the habitable zone, the ring around the sun where temperatures are right for liquid water.

Tilting Saturn's orbit would also stretch out Earth's orbit. According to a simple model that did not include other inner planets, the greater the tilt, the more the elongation increased. Adding Venus and Mars to the model stabilised the orbits of all three planets, but the elongation nonetheless rose as Saturn's orbit got more tilted. Pilat-Lohinger says a 20-degree tilt would bring the innermost part of Earth's orbit closer to the sun than Venus.

Booted out

Away from such simulations, the circularity of every planet's orbit does fluctuate over time. If the orbit is already highly elongated, such fluctuations would allow a planet to escape the sun's gravity. A 20-degree tilt of Saturn's orbit could eventually boot Mars out, while Earth would require a 30-degree tilt.

Pilat-Lohinger's methods are sound and her conclusions well supported, says Rory Barnes at the University of Washington in Seattle. But he notes that the implications for life in the universe are unclear. For one thing, we know the orbital inclination of only two planets outside the solar system: both orbit the star Upsilon Andromedae, with orbits inclined by 30 degrees to the star's equator.

What the elongation of an orbit means for life is uncertain, too. "At some point, the eccentricity of a planet impacts its potential to support life, but it's hard to say where that boundary is," says Barnes. A planet with an orbit shuttling it between Earth's distance from the sun and that of Mercury would be quite different from the Earth, he says, "but I don't think it would prevent life from originating".

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http://bit.ly/1r1vS8e

One Idea to Get to Mars: Fill the Walls of a Spaceship With Water

The insulation from radiation would also be drinkable

By Shannon Palus

NASA isn't particualrly close to sending humans to Mars. Whatever Mars One says, as a society, we're closer to the brainstorming phase of how a piloted mission to the red planet would even work. One of the weird ideas that NASA architects have sketched out? Spaceship walls that are filled with water.

Vice talked to NASA's chief technologist, David Miller, about the concept:

Line your space shuttle with water and hey presto: you both help protect against radiation during the journey and transport a vital resource for your astronauts.

“Water with hydrogen content absorbs radiation to some degree,” Miller explained. “Seeing as you need to take water, maybe you could line the walls of your capsule with water. So it’s used for drinking as well as shielding.”

The water would be replenished with purified astronaut waste. The design combines radiation protection, thermal control, and life support—not just in the form of drinking water but also algae, grown for food—thus reducing the overall weight of the spacecraft.

Plus, this system would not need to be that complex. So it's less likely to break down than other schemes for shielding the craft and supplying water. The only mechanical aspect are the pumps that push water from toilet waste through membranes to purify it.

Astronauts in low Earth orbit have been drinking recycled waste water since 2010. (It's purified with a spinning keg-sized device.) But even with an innovative water purifying system, astronauts would likely shower the way they do on the International Space Station: with babywipes.