Meat, Egg, Dairy Nutrient Essential for Brain Development

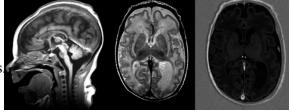
Asparagine, found in foods such as meat, eggs, and dairy products, was until now considered non-essential because it is produced naturally by the body.

Researchers at the University of Montreal and its affiliated CHU Sainte-Justine Hospital found that the amino acid is essential for normal brain development. This is not the case for other organs.

"The cells of the body can do without it because they use asparagine provided through diet. Asparagine, however, is not well transported to the brain via the blood-brain barrier," said senior co-author of the study Dr. Jacques Michaud, who found that brain cells depend on the local synthesis of asparagine to function properly. First co-author José-Mario Capo-Chichi and colleague Grant Mitchell also made major contributions to the study.

In April 2009, a Quebec family experienced the worst tragedy for parents: before the age of one, one of their sons died of a rare genetic disease causing congenital microcephaly,

intellectual disability, cerebral atrophy, and refractory seizures. The event was even more tragic because it was the third infant to die in this family from the same disease.



A genetic defect disrupts brain development by affecting the synthesis of asparagine, an amino acid until now thought to be non-essential. The discovery was made by researchers at CHU Sainte-Justine and the University of Montreal. This image shows MRI Images taken from a child who later died from the defect. (Credit: Universite de Montreal)

This tragedy led Dr. Michaud to discover the genetic abnormality responsible for this developmental disorder. "We are not at the verge of a miracle drug," Michaud said, "but we at least know where to look."

The team identified the gene affected by the mutation code for asparagine synthetase, the enzyme responsible for synthesizing the amino acid asparagine. The study is the first to associate a specific genetic variant with a deficiency of this enzyme. "In healthy subjects, it seems that the level of asparagine synthetase in the brain is sufficient to supply neurons," Michaud said. "In individuals with the disability, the enzyme is not produced in sufficient quantity, and the resulting asparagine depletion affects the proliferation and survival of cells during brain development."

Potential treatment

Children who are carriers of this mutation suffer, to varying degrees, from a variety of symptoms, including intellectual disability and cerebral atrophy, which can lead to death. The Quebec family lost three infant sons to this disorder. Two of their other children are alive and healthy.

Knowledge about gene mutations can be used to develop treatments. "Our results not only open the door to a better understanding of the disease," Michaud said, "but they also give us valuable information about the molecular mechanisms involved in brain development, which is important for the development of new treatments."

For example, asparagine supplement could be given to infants to ensure an adequate level of asparagine in the brain and the latter's normal development. "The amount of supplementation remains to be determined, as well as its effectiveness," said the geneticist. "Since these children are already born with neurological abnormalities, it is uncertain whether this supplementation would correct the neurological deficits."

Creating a pediatric clinical genomics centre

To date, nine children from four different families have been identified as carriers of the mutation: three infants from Quebec, three from a Bengali family living in Toronto, and three Israelis, whose symptoms are less severe. Dr. Michaud's team discovered the genetic mutation by comparing the complete DNA of the Quebec family's children with symptoms of the disease.

The researchers then identified children, among other families, who carried the single candidate gene. The gene was revealed only in the affected children, but not in the unaffected children of the families studied.

The discovery comes at a time when CHU Sainte-Justine Mother and Child University Hospital has reached an agreement with Génome Québec to create the first pediatric clinical genomic centre in Canada.

"This initiative will transform the quality of care for younger patients to ensure better prevention from childhood," says Dr. Michaud. "More than 80% of genetic diseases occur in childhood or adolescence. "This new technology will allow us to sequence all the genes in the genome and obtain a genetic portrait of the children more quickly to know which disease they suffer from and to provide treatment, if available, or when it becomes available."

2 12/2/13 Name

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Alzheimer's and vascular changes in the neck

Association found between a vascular abnormality in the neck and white matter changes in the brain in patients with Alzheimer's disease and mild cognitive impairment

Buffalo, N.Y. – Studies on Alzheimer's disease and other forms of dementia have long focused on what's happening inside the brain. Now an international research team studying Alzheimer's and mild cognitive impairment is reporting potentially significant findings on a vascular abnormality outside the brain. The finding has potential implications for a better understanding of Alzheimer's and other neurological disorders associated with aging.

The pilot study was published in the Journal of Alzheimer's Disease Nov. 8 online ahead of print by researchers from the University at Buffalo, the University of Bradford in the United Kingdom and National Yang-Ming University School of Medicine in Taiwan. The authors caution that the study is small and that the results must be validated in larger, future studies.

They studied a hemodynamic abnormality in the internal jugular veins called jugular venous reflux or JVR. It occurs when the pressure gradient reverses the direction of blood flow in the veins, causing blood to leak backwards into the brain.

JVR occurs in certain physiological situations, if the internal jugular vein valves do not open and close properly, which occurs more frequently in the elderly. This reverse flow is also believed to impair cerebral venous drainage.

The brain's white matter is made of myelin and axons that enable communication between nerve cells. "We were especially interested to find an association between JVR and white matter changes in the brains of patients with Alzheimer's disease and those with mild cognitive impairment," says Robert Zivadinov, MD, PhD, FAAN, professor of neurology at the UB School of Medicine and Biomedical Sciences and senior author. "Age-related white matter changes have long been associated with dementia and faster cognitive decline," he says. "To the best of our knowledge, our study is the first to show that JVR is associated with a higher frequency of white matter changes, which occur in patients with mild cognitive impairment and Alzheimer's disease."

Ching-Ping Chung, the first author on the study and assistant professor of neurology at National Yang-Ming University, adds: "We are the first to observe that JVR may be associated with formation of these lesions in the brain, given the fact that Alzheimer's patients have more white matter lesions than healthy people.

"If this observation is validated in larger studies," she continues, "it could be significant for the development of new diagnostic tools and treatments for pathological white matter lesions developed in Alzheimer's disease and other forms of dementia." White matter changes have been found to have a direct relationship to the buildup of amyloid plaque long seen as central to the development of Alzheimer's disease.

"The accumulation of amyloid plaque may result from the inability of cerebrospinal fluid to be properly cleared from the brain," says Clive Beggs, second author on the study and professor of medical engineering at the University of Bradford. In addition, he says, the study found that JVR appeared to be associated with dirtyappearing white matter, which is thought to represent early stage lesion formation.

"To the best of our knowledge, this is one of the first studies to explore the impact of dirty-appearing white matter in the elderly," Beggs continues. He adds that the significance of dirty-appearing white matter in the elderly needs more study. The research involved 12 patients with Alzheimer's disease, 24 with mild cognitive impairment and 17 age-matched elderly controls. Participants underwent Doppler ultrasound exams and magnetic resonance imaging scans.

The impact of hemodynamic changes in veins from the brain to the neck has been the focus of numerous studies by Zivadinov and colleagues at UB and institutions worldwide.

"Given the major finding of our group in 2011 that both healthy controls and people with a variety of neurological diseases present with structural and hemodynamic changes of the extracranial venous system, we thought it was important to study how they might be involved in the development of Alzheimer's disease and other important neurodegenerative conditions," he explains.

Zivadinov notes that the frequency of JVR increases with aging and its accumulated effects on cerebral circulation may take many years to develop. Patients are likely to be asymptomatic for a long time, which would explain why the condition is seen in both healthy people and those with neurological diseases, he adds. Co-authors besides Zivadinov, Chung and Beggs are Simon Shepherd of the Centre for Infection Control and Biophysics at the University of Bradford; Pei-Ning Wang, Chun-Yu Cheng and Han-Hwa Hu, all of Taipei Veterans General Hospital in Taipei and National Yang-Ming University; and Niels Bergsland, Deepa P. Ramasamy and Michael G. Dwyer all of the Buffalo Neuroimaging Analysis Center in the UB Department of Neurology.

3 12/2/13

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Dying from a food allergy is less likely than being murdered

A person with a food allergy is more likely to be murdered than to die from a severe reaction, according to a new study.

One in 10 children has a food allergy. Many sufferers and their parents experience anxiety about the possibility of a severe and life-threatening allergic reaction, called anaphylaxis, but until now no studies have estimated how common death from such reactions is. Based on data from 13 studies worldwide, researchers at Imperial College London calculated that for any person with a food allergy, the chance of dying from anaphylaxis in one year is 1.81 in a million. For children and young people aged 0-19, the risk is 3.25 in a million.

By comparison, in Europe the risk of being murdered is 11 in a million and of dying from accidental causes is 324 in a million over a year (US figures in notes to editors).

Dr Robert Boyle, from the Department of Medicine at Imperial, who led the study, said: "Everyone has heard stories of people who have died suddenly from a severe allergic reaction, and these stories are frightening. But events like this appear to be very rare, and it's helpful to put that risk in perspective.

"We don't want to belittle the concerns of people with food allergies or their families, and of course people should continue to take reasonable precautions. That said, we want to reassure them that having a food allergy makes a very small difference to someone's overall risk of death.

"Worrying about severe allergic reactions can take a huge toll on someone's quality of life. We should address anxiety and quality of life for food allergic people and their carers, rather than just focus on the risk of death." The study is published in Clinical and Experimental Allergy. It was funded by Lincoln Medical and the National Institute for Health Research (NIHR) Imperial Biomedical Research Centre.

Food allergies appear to be becoming more common. Hospital admissions for children with food allergies have risen five times in the last 20 years, but the reason for this trend is unclear.

Typical allergic reactions involve swelling, rash, or eczema. The reason why severe, life-threatening reactions sometimes occur is not known. The dose of allergen plays a role in determining the risk, but the dose required to trigger anaphylaxis varies widely. Anaphylaxis is most common in young people, but doctors have no way to tell which patients are most susceptible.

http://www.eurekalert.org/pub_releases/2013-11/uota-sep112513.php

Study examines potential evolutionary role of 'sexual regret' in human survival and reproduction

UT Austin study reveals gender differences in sexual regret

AUSTIN, Texas - In the largest, most in-depth study to date on regret surrounding sexual activity, a team of psychology researchers found a stark contrast in remorse between men and women, potentially shedding light on the evolutionary history of human nature.

Researchers for the peer-reviewed study included University of Texas at Austin evolutionary psychologist David Buss. The study was led by Andrew Galperin, a former social psychology doctoral student at the University of California-Los Angeles; and Martie Haselton, a UCLA social psychology professor. It is published in the current issue of Archives of Sexual Behavior.

The findings show how human emotions such as regret can play an important role in survival and reproduction. They suggest that men are more likely to regret not taking action on a potential liaison, and women are more remorseful for engaging in one-time liaisons.

"Prior sex researchers have focused primarily on the emotion of sexual attraction in sexual decisions," Buss says. "These studies point to the importance of a neglected mating emotion -sexual regret - which feels experientially negative but in fact can be highly functional in guiding adaptive sexual decisions." Evolutionary pressures probably explain the gender difference in sexual regret, says Haselton, who earned her Ph.D. in psychology at UT Austin.

"For men throughout evolutionary history, every missed opportunity to have sex with a new partner is potentially a missed reproduce opportunity - a costly loss from an evolutionary perspective." Haselton says. "But for women, reproduction required much more investment in each offspring, including nine months of pregnancy and potentially two additional years of breastfeeding. The consequences of casual sex were so much higher for women than for men, and this is likely to have shaped emotional reactions to sexual liaisons even today."

In three studies the researchers asked participants about their sexual regrets. In the first study, 200 respondents evaluated hypothetical scenarios in which someone regretted pursuing or failing to pursue an opportunity to have sex. They were then asked to rate their remorse on a five-point scale. In the second study, 395 participants

4

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were given a list of common sexual regrets and were asked to indicate which ones they have personally experienced. The last study replicated the second one with a larger sample of 24,230 individuals that included gay, lesbian and bisexual respondents.

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According to the findings:

The top three most common regrets for women are: losing virginity to the wrong partner (24 percent), cheating on a present or past partner (23 percent) and moving too fast sexually (20 percent).

For men, the top three regrets are: being too shy to make a move on a prospective sexual partner (27 percent), not being more sexually adventurous when young (23 percent) and not being more sexually adventurous during their single days (19 percent).

More women (17 percent) than men (10 percent) included "having sex with a physically unattractive partner" as a top regret.

Although rates of actually engaging in casual sex were similar overall among participants (56 percent), women reported more frequent and more intense regrets about it.

Comparing gay men and lesbian women, and bisexual men and bisexual women, a similar pattern held - women tended to regret casual sexual activity more than men did.

Regret comes after the fact, so it's not protective, Haselton notes. But it might help women avoid a potentially costly action again. "One thing that is fascinating about these emotional reactions in the present is that they might be far removed from the reproductive consequences of the ancestral past," Haselton says. "For example, we have reliable methods of contraception. But that doesn't seem to have erased the sex differences in women's and men's responses, which might have a deep evolutionary history."

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Archaeological discoveries confirm early date of Buddha's life Evidence found at world's earliest Buddhist shrine in Nepal

WASHINGTON - Archaeologists working in Nepal have uncovered evidence of a structure at the birthplace of the Buddha dating to the sixth century B.C. This is the first archaeological material linking the life of the Buddha - and thus the first flowering of Buddhism - to a specific century.

Pioneering excavations within the sacred Maya Devi Temple at Lumbini, Nepal, a UNESCO World Heritage site long identified as the birthplace of the Buddha, uncovered the remains of a previously unknown sixth-century B.C. timber structure under a series of brick temples. Laid out on the same design as those above it, the timber structure contains an open space in the center that links to the nativity story of the Buddha himself. Until now, the earliest archaeological evidence of Buddhist structures at Lumbini dated no earlier than the third century B.C., the time of the patronage of the Emperor Asoka, who promoted the spread of Buddhism from present-day Afghanistan to Bangladesh.

"Very little is known about the life of the Buddha, except through textual sources and oral tradition," said archaeologist Professor Robin Coningham of Durham University, U.K., who co-led the investigation. Some scholars, he said, have maintained that the Buddha was born in the third century B.C. "We thought 'why not go back to archaeology to try to answer some of the questions about his birth?' Now, for the first time, we have an archaeological sequence at Lumbini that shows a building there as early as the sixth century B.C." Early Buddhism revealed

The international team of archaeologists, led by Coningham and Kosh Prasad Acharya of the Pashupati Area Development Trust in Nepal, say the discovery contributes to a greater understanding of the early development of Buddhism as well as the spiritual importance of Lumbini. Their peer-reviewed findings are reported in the December 2013 issue of the international journal Antiquity. The research is partly supported by the National Geographic Society.

To determine the dates of the timber shrine and a previously unknown early brick structure above it, fragments of charcoal and grains of sand were tested using a combination of radiocarbon and optically stimulated luminescence techniques. Geoarchaeological research also confirmed the presence of ancient tree roots within the temple's central void.

"UNESCO is very proud to be associated with this important discovery at one of the most holy places for one of the world's oldest religions," said UNESCO Director-General Irina Bokova, who urged "more archaeological research, intensified conservation work and strengthened site management" to ensure Lumbini's protection.

"These discoveries are very important to better understand the birthplace of the Buddha," said Ram Kumar Shrestha, Nepal's minister of culture, tourism and civil aviation. "The government of Nepal will spare no effort to preserve this significant site."

Buddhist tradition records that Queen Maya Devi, the mother of the Buddha, gave birth to him while holding on to the branch of a tree within the Lumbini Garden, midway between the kingdoms of her husband and parents.

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Coningham and his colleagues postulate that the open space in the center of the most ancient, timber shrine may have accommodated a tree. Brick temples built later above the timber shrine also were arranged around the central space, which was unroofed.

Four main Buddhist sites

Lumbini is one of the key sites associated with the life of the Buddha; others are Bodh Gaya, where he became a Buddha or enlightened one; Sarnath, where he first preached; and Kusinagara, where he passed away. At his passing at the age of 80, the Buddha is recorded as having recommended that all Buddhists visit "Lumbini." The shrine was still popular in the middle of the first millennium A.D. and was recorded by Chinese pilgrims as having a shrine beside a tree.

The Maya Devi Temple at Lumbini remains a living shrine; the archaeologists worked alongside meditating monks, nuns and pilgrims.

In the scientific paper in Antiquity, the authors write: "The sequence (of archaeological remains) at Lumbini is a microcosm for the development of Buddhism from a localized cult to a global religion."

Lost and overgrown in the jungles of Nepal in the medieval period, ancient Lumbini was rediscovered in 1896 and identified as the birthplace of the Buddha on account of the presence of a third-century B.C. sandstone pillar. The pillar, which still stands, bears an inscription documenting a visit by Emperor Asoka to the site of the Buddha's birth as well as the site's name - Lumbini.

Despite the rediscovery of the key Buddhist sites, their earliest levels were buried deep or destroyed by later construction, leaving evidence of the very earliest stages of Buddhism inaccessible to archaeological investigation, until now.

Half a billion people around the world are Buddhists, and many hundreds of thousands make a pilgrimage to Lumbini each year. The archaeological investigation there was funded by the government of Japan in partnership with the government of Nepal, under a UNESCO project aimed at strengthening the conservation and management of Lumbini. Along with the National Geographic Society, the research also was supported by Durham University and Stirling University.

Coningham and Acharya were joined on the Antiquity paper by coauthors K.M. Strickland, C.E. Davis, M.J. Manuel, I. A. Simpson, K. Gilliland, J. Tremblay, T.C. Kinnaird and D.C.W. Sanderson.

NOTES: A documentary on Coningham's exploration of the Buddha's life, "Buried Secrets of the Buddha," will premiere in February internationally on National Geographic Channel.

http://www.sciencedaily.com/releases/2013/11/131126092439.htm

Vitamins: Potential Damage to Body's Defences

Vitamin supplements are a billion-dollar industry. We want to stay healthy and fit and help our bodies with this. But perhaps we are achieving precisely the opposite?

"We believe that antioxidants are good for us, since they protect the cells from oxidative stress that may harm our genes. However, our bodies have an enormous inherent ability to handle stress. Recent research results show that the body's responses to stress in fact are important in preventing our DNA from eroding. I fear that the fragile balance in our cells can be upset when we supplement our diet with vitamin pills, says Hilde Nilsen to the research magazine Apollon. Nilsen is heading a research group at the Biotechnology Centre, University of Oslo.

Maintenance of genes

Our DNA - the genetic code that makes us who we are - is constantly exposed to damage.

In each of the hundred trillion cells in our body, up to two hundred thousand instances of damage to the DNA take place every day. These may stem from environmental causes such as smoking, stress, environmental pathogens or UV radiation, but the natural and life-sustaining processes in the organism are the primary sources of damage to our DNA. How can the repair of damage to our DNA help us stay healthy and live long lives?

A small worm provides the answer

To answer this question, Hilde Nilsen and her group of researchers have allied themselves with a small organism - a one millimetre-long nematode called Caenorhabditis elegans (C. elegans). This roundworm, which lives for only 25 days, is surprisingly sophisticated with its 20,000 genes; we humans only have a couple of thousand more.

C. elegans is a fantastically powerful tool, because we can change its hereditary properties. We can increase its ability to repair DNA damage, or we can remove it altogether. We can also monitor what happens when damage to DNA is not repaired in several hundred specimens and through their entire lifespan. Different "repair proteins" take care of various types of damage to the DNA. The most common ones are repaired by "cutting out" and replacing a single damaged base by itself or as part of a larger fragment.

Affecting lifespan with the aid of genes

In some specimens that do not have the ability to repair the damage, the researchers observe that the aging process proceeds far faster than normal. Is it because the damage accumulates in the DNA and prevents the cells from producing the proteins they need for their normal operation? Most researchers have thought so, but Hilde Nilsen doubts it.

One of the genes studied by the researchers has a somewhat shortened lifespan: on average, this mutant lives three days less than normal. Translated into human terms, this means dying at the age of 60 rather than at 70. - "We were surprised when we saw that these mutants do not in fact accumulate the DNA damage that would cause aging. On the contrary: they have less DNA damage. This happens because the little nematode changes its metabolism into low gear and releases its own antioxidant defences. Nature uses this strategy to minimize the negative consequences of its inability to repair the DNA. So why is this not the normal state? Most likely because it comes at a cost: these organisms have less ability to respond to further stress – they are quite fragile. Hilde Nilsen and her colleagues have now -for the very first time -"shown that this response is under active genetic control and is not caused by passive accumulation of damage to the DNA, as has been widely believed. This provides an opportunity to manipulate these processes. And that's exactly what we have done: we have re-established the normal lifespan of a short-lived mutant by removing other proteins that repair damage. Hence, the cause could not be accumulation of damage, since there is no reason to assume that a mutant with no other alternative ways to repair its DNA will be less exposed to damage. There must be something else.

The researchers have gone on to discover that this "something else" in fact is the other repair proteins. They believe that the proteins inhibit damage that they fail to repair completely. The consequence is that they establish a barrier - a road block. This triggers a cascade of signals that reprogram the cell.

Wouldn't this imply that the repair proteins defy their own purpose -"after all, the result is a shorter lifespan? We need to remember that most likely, the purpose of the DNA repairs is to ensure that we produce healthy offspring -"not necessarily that we live as long as possible after our reproductive age interval. Initiating a survival response that reinforces the antioxidant defences means that a lack of ability to repair the DNA has less impact than it would otherwise have on our reproduction. To the species as a whole, it's a small cost that some individuals will be less good at handling stress and have a shorter life.

Because this is an active process within the cells, the researchers refer to it as reprogramming.

"We have found several proteins that trigger this reprogramming. The process has the same effect as a reduction in caloric intake, which we know helps increase the lifespan in many species. In other words, there are two routes to a long life. When we stimulate both of these two routes in our nematode at the same time, we can quadruple its normal lifespan," Nilsen says.

Can do great harm

The balance between oxidants and antioxidants is crucial to our physiology, but exactly where this equilibrium is situated varies from one person to the next.

"This is where I start worrying about the synthetic antioxidants. The cells in our body use this fragile balance to establish the best possible conditions for themselves, and it is specially adapted for each of us. When we take supplements of antioxidants, such as C and E vitamins, we may upset this balance," the researcher warns. "It sounds intuitively correct that intake of a substance that may prevent accumulation of damage would benefit us, and that's why so many of us supplement our diet with vitamins. Our research results indicate that at the same time, we may also cause a lot of harm. The health authorities recommend that instead, we should seek to have an appropriate diet. I'm all in favour of that. It's far safer for us to take our vitamins through the food that we eat, rather than through pills," Hilde Nilsen states emphatically.

http://scitechdaily.com/aerospace-engineers-discover-possible-fix-kepler-spacecraft/

Aerospace Engineers Discover a Possible Fix for the Kepler Spacecraft

Kepler mission and Ball Aerospace engineers believe they have figured out how to get the Kepler Spacecraft working again.

November 26, 2013 by Staff

By maneuvering the spacecraft so that the solar pressure is evenly distributed across the surfaces of the spacecraft, they believe the Sun can as the 'third wheel' to control pointing.

You may have thought that NASA's Kepler spacecraft was finished. Well, think again. A repurposed Kepler Space telescope may soon start searching the sky again.

A new mission concept, dubbed K2, would continue Kepler's search for other worlds, and introduce new opportunities to observe star clusters, young and old stars, active galaxies and supernovae.

In May, the Kepler spacecraft lost the second of four gyroscope-like reaction wheels, which are used to precisely point the spacecraft, ending new data collection for the original mission. The spacecraft required three functioning wheels to maintain the precision pointing necessary to detect the signal of small Earth-sized

exoplanets, which are planets outside our solar system, orbiting stars like our sun in what's known as the habitable zone - the range of distances from a star where the surface temperature of a planet might be suitable for liquid water.

With the failure of a second reaction wheel, the spacecraft can no longer precisely point at the mission's original field of view. The culprit is none other than our own sun.

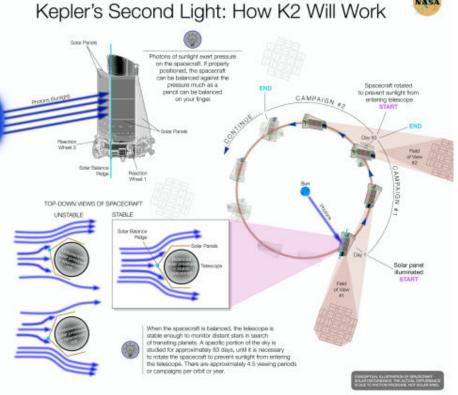
The very body that provides Kepler with its energy needs also pushes the spacecraft around by the pressure exerted when the photons of sunlight strike the spacecraft. Without a third wheel to help counteract the solar pressure, the spacecraft's ultra-precise pointing capability cannot be controlled in all directions.

However, Kepler mission and Ball Aerospace engineers have developed an innovative way of recovering pointing stability by maneuvering

the spacecraft so that the solar pressure is evenly distributed across the surfaces of the spacecraft.

To achieve this level of stability, the orientation of the spacecraft must be nearly parallel to its orbital path around the sun, which is slightly offset from the ecliptic, the orbital plane of Earth. The ecliptic plane defines the band of sky in which lie the constellations of the zodiac.

This technique of using the sun as the 'third wheel' to control pointing is currently being tested on the spacecraft and early results are already coming in. During a pointing performance test in late October, a full frame image of the space telescope's full field of view was captured showing part of the constellation Sagittarius.



This conception illustration depicts how solar pressure can be used to balance NASA's Kepler spacecraft, keeping the telescope stable enough to continue searching for transiting planets around distant stars. NASA Ames/W Stenzel

Photons of light from a distant star field were collected over a 30-minute period and produced an image quality within five percent of the primary mission image quality, which used four reaction wheels to control pointing stability. Additional testing is underway to demonstrate the ability to maintain this level of pointing control for days and weeks.

To capture the telltale signature of a distant planet as it crosses the face of its host star and temporarily blocks the amount of starlight collected by Kepler, the spacecraft must maintain pointing stability over these longer periods.

"This 'second light' image provides a successful first step in a process that may yet result in new observations and continued discoveries from the Kepler space telescope," said Charlie Sobeck, Kepler deputy project manager at NASA Ames Research Center in Moffett Field, California.

The K2 mission concept has been presented to NASA Headquarters. A decision to proceed to the 2014 Senior Review – a biannual assessment of operating missions – and propose for budget to fly K2 is expected by the end of 2013.

Kepler's original mission, which is still in progress to fully process the wealth of data collected, is to determine what percentage of stars like the sun harbor small planets the approximate size and surface temperature of Earth. For four years, the space telescope simultaneously and continuously monitored the brightness of more than 150,000 stars, recording a measurement every 30 minutes.

More than a year of the data collected by Kepler remains to be fully reviewed and analyzed. *Source: Michele Johnson, Ames Research Center; NASA*

Student number Name http://www.livescience.com/41537-t-rex-soft-tissue.html

Controversial T. Rex Soft Tissue Find Finally Explained

The controversial discovery of 68-million-year-old soft tissue from the bones of a Tyrannosaurus rex finally has a physical explanation. According to new research, iron in the dinosaur's body preserved the tissue before it could decay.

By Stephanie Pappas, Senior Writer | November 26, 2013 07:01pm ET

The research, headed by Mary Schweitzer, a molecular paleontologist at North Carolina State University, explains how proteins - and possibly even DNA - can survive millennia. Schweitzer and her colleagues first raised this question in 2005, when they found the seemingly impossible: soft tissue preserved inside the leg of an adolescent T. rex unearthed in Montana.

"What we found was unusual, because it was still soft and still transparent and still flexible," Schweitzer told LiveScience.

T. rextissue?

The find was also controversial, because scientists had thought proteins that make up soft tissue should degrade in less than 1 million years in the best of conditions. In most cases, microbes feast on a dead animal's soft tissue, destroying it within weeks. The tissue must be something else, perhaps the product of a later bacterial invasion, critics argued.

Then, in 2007, Schweitzer and her colleagues analyzed the chemistry of the T. rex proteins. They found the proteins really did come from dinosaur soft tissue. The tissue was collagen, they reported in the journal Science, and it shared similarities with bird collagen - which makes sense, as modern birds evolved from theropod dinosaurs such as T. rex.

The researchers also analyzed other fossils for the presence of soft tissue, and found it was present in about half of their samples going back to the Jurassic Period, which lasted from 145.5 million to 199.6 million years ago, Schweitzer said.

"The problem is, for 300 years, we thought, 'Well, the organics are all gone, so why should we look for something that's not going to be there?' and nobody looks," she said.

The obvious question, though, was how soft, pliable tissue could survive for millions of years. In a new study published today (Nov. 26) in the journal Proceedings of the Royal Society B: Biological Sciences, Schweitzer thinks she has the answer: Iron.

Iron ladv

Iron is an element present in abundance in the body, particularly in the blood, where it is part of the protein that carries oxygen from the lungs to the tissues. Iron is also highly reactive with other molecules, so the body keeps it locked up tight, bound to molecules that prevent it from wreaking havoc on the tissues.

After death, though, iron is let free from its cage. It forms minuscule iron nanoparticles and also generates free radicals, which are highly reactive molecules thought to be involved in aging.

"The free radicals cause proteins and cell membranes to tie in knots," Schweitzer said. "They basically act like formaldehyde."

Formaldehyde, of course, preserves tissue. It works by linking up, or cross-linking, the amino acids that make up proteins, which makes those proteins more resistant to decay.

Schweitzer and her colleagues found that dinosaur soft tissue is closely associated with iron nanoparticles in both the T. rex and another soft-tissue specimen from Brachylophosaurus canadensis, a type of duck-billed dinosaur. They then tested the iron-as-preservative idea using modern ostrich blood vessels. They soaked one group of blood vessels in iron-rich liquid made of red blood cells and another group in water. The blood vessels left in water turned into a disgusting mess within days. The blood vessels soaked in red blood cells remain recognizable after sitting at room temperature for two years.

Dinosaurs' iron-rich blood, combined with a good environment for fossilization, may explain the amazing existence of soft tissue from the Cretaceous (a period that lasted from about 65.5 million to 145.5 million years ago) and even earlier. The specimens Schweitzer works with, including skin, show evidence of excellent preservation. The bones of these various specimens are articulated, not scattered, suggesting they were buried quickly. They're also buried in sandstone, which is porous and may wick away bacteria and reactive enzymes that would otherwise degrade the bone.

Schweitzer is set to search for more dinosaur soft tissue this summer. "I'd like to find a honking big T. rex that's completely articulated that's still in the ground, or something similar," she said. To preserve the chemistry of potential soft tissue, the specimens must not be treated with preservatives or glue, as most fossil bones are, she said. And they need to be tested quickly, as soft tissue could degrade once exposed to modern air and humidity.

Student number

Importantly, Schweitzer and her colleagues have figured out how to remove the iron from their samples, which enables them to analyze the original proteins. They've even found chemicals consistent with being DNA, though Schweitzer is quick to note that she hasn't proven they really are DNA. The iron-removing techniques should allow paleontologists to search more effectively for soft tissue, and to test it when they find it. "Once we can get the chemistry behind some of these soft tissues, there's all sorts of questions we can ask of ancient organisms," Schweitzer said.

http://phys.org/news/2013-11-ancient-reptiles-common-latrine.html

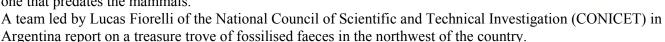
Ancient reptiles gathered in common latrine

Some large, grass-eating mammals, such as elephants, rhinos and camels, gather together not only when they eat, but also when they defecate.

The group poop, researchers have surmised, has several important functions. One is hygiene, to prevent the emergence of new parasites; another is safety, to provide surveillance in numbers against any prowling predator.

Name

But new evidence, turned up in the journal Nature's Scientific Reports, suggests the communal latrine has a much older history, and one that predates the mammals.



They counted no less than 30,000 of faeces, ranging in size from 0.5 to 35 centimetres (0.2 to 14 inches) in length. In some spots, the ancient excreta piled up to 100 per square metre. The faeces-counting may have been exhaustive, but it showed that going through the motions can sometimes bring rewards.

It turns out the communal latrine dates back to 240 million years, a whole 20 million years earlier than the previous record-holder. And close examination of the faeces showed it was deposited by none other than members of the Dicynodontes, "megaherbivore" reptiles which lived cheek-by-jowl with early dinosaurs. "This is the first evidence of megaherbivore communal latrines in non-mammal vertebrates, indicating that this mammal-type behaviour was present in distant relatives of mammals," says the paper.

http://www.eurekalert.org/pub_releases/2013-11/uow-edc112513.php

Economic development can only buy happiness up to a 'sweet spot' of \$36,000 GDP per person, study finds

Economists have shed light on the vexed question of whether economic development can buy happiness – and it seems that life satisfaction actually dips among people living in the wealthiest countries.

Politicians are intensely interested in the link between national wealth and levels of happiness among the population, but it is a subject which is still wide open to debate among economists.

A new analysis led by economists Eugenio Proto in the Centre for Competitive Advantage in the Global Economy at the University of Warwick and Aldo Rustichini, from University of MInnesota finds that as expected, for the poorest countries life satisfaction rises as a country's wealth increases as people are able to meet their basic needs.

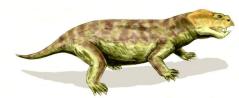
However, the new surprise finding is that once income reaches a certain level – around \$36,000, adjusted for Purchasing Power Parity (PPP) - life satisfaction levels peaks, after which it appears to dip slightly in the very rich countries.

According to the most recent figures, the UK had a PPP-adjusted GDP per capita of roughly \$37,000 dollars. The researchers find suggestive evidence that this happiness dip in the wealthiest countries is because more money creates higher aspirations, leading to disappointment and a drop in life satisfaction if those aspirations are not met. Dr Proto said: "Whether wealth can buy a country's happiness is a major question for governments. Many policy-makers, including in the UK, are interested in official measures of national well-being.

"Our new analysis has one very surprising finding which has not been reported before – that life satisfaction appears to dip beyond a certain level of wealth.

"In our study we see evidence that this is down to changes in the aspiration levels of people living in the richest countries. "As countries get richer, higher levels of GDP lead to higher aspiration. There is a sense of keeping up with the Joneses as people see wealth and opportunity all around them and aspire to having more.

"But this aspiration gap - the difference between actual income and the income we would like - eats away at life satisfaction levels. "In other words, what we aspire to becomes a moving target and one which moves away faster in the richest countries, causing the dip in happiness we see in our analysis."



The study found that people in countries with a GDP per capita of below \$6,700 were 12 per cent less likely to report the highest level of life satisfaction than those in countries with a GDP per capita of around \$18,000. However, once countries reach around \$20,400 GDP per capita, the increase in happiness that higher wealth brings is less obvious. Between this level and the very highest GDP per capita level (\$54,000), the probability of reporting the highest level of life satisfaction changes by no more than two per cent.

Name

This corresponds broadly to the well-known Easterlin Paradox - that the link between life satisfaction and GDP is more or less flat in richer countries.

However, instead of continuing to increase or flatten as other studies have suggested, this new analysis finds a small drop in life satisfaction once countries go beyond a level of GDP per capita of around \$36,000.

The researchers used data on life satisfaction gathered from the World Values Survey and GDP figures which they analysed as quantiles, a new approach to looking at this issue.

By analysing the data this way, they were able to avoid imposing restrictions on the econometric model . Furthermore, they control for country-fixed effects, in order to exclude possible effect due to culture, translation and linguistic issues.

The findings were published in a study entitled A Reassessment of the Relationship Between GDP and Life Satisfaction in the open access journal PLOS ONE.

The paper will be available here once the embargo has lifted <u>http://dx.plos.org/10.1371/journal.pone.0079358</u>

http://www.sciencedaily.com/releases/2013/11/131127115315.htm

Big Brains Are All in the Genes

Scientists have moved a step closer to understanding genetic changes that permitted humans and other mammals to develop such big brains

Scientists have moved a step closer to understanding genetic changes that permitted humans and other mammals to develop such big brains.

During evolution, different mammal species have experienced variable degrees of expansion in brain size. An important goal of neurobiology is to understand the genetic changes underlying these extraordinary adaptations. The process by which some species evolved larger brains -- called encephalization -- is not well understood by scientists.

The puzzle is made more complex because evolving large brains comes at a very high cost.

Dr Humberto Gutierrez, from the School of Life Sciences, University of Lincoln, UK, led research which examined the genomes of 39 species of mammals with the aim of better understanding how brains became larger and more complex in mammals.

To do this, the scientists focussed on the size of gene families across these species. Gene families are groups of related genes which share similar characteristics, often linked with common or related biological functions. It is believed that large changes in the size of gene families can help to explain why related species evolved along different paths.

The researchers found a clear link between increased brain size and the expansion of gene families related to certain biological functions.

Dr Gutierrez said: "We found that brain size variations are associated with changes in gene number in a large proportion of families of closely related genes. These gene families are preferentially involved in cell communication and cell movement as well as immune functions and are prominently expressed in the human brain. Our results suggest that changes in gene family size may have contributed to the evolution of larger brains in mammals."

Mammalian species in general tend to have large brains compared to their body size which represent an evolutionary costly adaptation as they require large amounts of energy to function.

Dr Gutierrez explained: "The brain is an extremely expensive organ consuming a large amount of energy in proportion to its volume, so large brains place severe metabolic demands on animals. Larger brains also demand higher parental investment. For example, humans require many years of nurturing and care before their brains are fully matured."

Dr Gutierrez's research concluded that variations in the size of gene families associated with encephalization provided an evolutionary support for the specific physiological demands associated with increased brain size in mammals.

H. Gutierrez, A. Castillo-Morales, J. Monzon-Sandoval, A. O. Urrutia. Increased brain size in mammals is associated with size variations in gene families with cell signalling, chemotaxis and immune-related functions. Proceedings of the Royal Society B: Biological Sciences, 2013; 281 (1775): 20132428 DOI: 10.1098/rspb.2013.2428

Shingles Vaccine Uptake Disappointing

Although most adults 40 years and older are at risk for herpes zoster infection, also known as shingles, uptake of the vaccine (Zostavax) to prevent infection is disappointing, researchers say. Alice Goodman

NEW ORLEANS - "The risk of herpes zoster is increasing faster than the aging population," said Elizabeth Cohen, MD, from the NYU Langone Medical Center in New York City.

One manifestation of shingles is herpes zoster ophthalmicus - a painful condition with ocular complications that affects 25% of all people infected, she explained.

"Ophthalmologists and other physicians should encourage patients older than 40 to get vaccinated. As renowned bioethicist Arthur Caplan said, 'We have a moral obligation to do the right thing to change behavior'," she said.

Dr. Cohen reviewed the epidemiology of shingles and discussed the underuse of the vaccine here at the American Academy of Ophthalmology (AAO) 2013 Annual Meeting.

More than 90% of the people born in the United States who are older than 40 have had chicken pox. The virus lingers in the body and can be reactivated as shingles many years later.

The risk for reactivation is increasing in the United States and globally, Dr. Cohen explained. The risk goes up after age 40 and rises sharply at age 50. One in 3 people older than 50 will develop shingles, as will 50% of those who live to age 85. Shingles is not necessarily an older person's disease; more than 50% are younger than 60 years at onset, and the mean age of onset is 52 years.

It is not clear why the risk has increased by almost 70% in the past 15 years. "The increase in infection is greater than the increase of the aging population," she said. For unknown reasons, shingles occurs in more women than men.

The risk for postherpetic neuralgia, a painful complication of shingles, also increases with age. In fact, 80% of cases occur in people older than 50 years. Postherpetic neuralgia increases morbidity and quadruples 1-year medical costs, Dr. Cohen noted.

Herpes zoster ophthalmicus increases the risk for stroke 4.5 times within 1 year of diagnosis, and shingles increases the risk for cancer 9-fold in the first year after diagnosis, she reported.

The vaccine was originally approved for patients older than 60, but it is now encouraged for those 50 and older who are not immunocompromised or on immunosuppressant treatment, do not have anaphylactic allergy to gelatin and neomycin, and are not pregnant.

"It is better to get the vaccine in your 50s and 60s, but it is never too late to get it," Dr. Cohen said. Information about the vaccine is available on the Web site of the Centers for Disease Control and Prevention.

The vaccine reportedly reduces the overall burden of disease by 61%, decreases the incidence of shingles by 51%, and lowers the incidence of postherpetic neuralgia by 66%.

Unfortunately, only 15% of people older than 50 are vaccinated, according to the Centers for Disease Control. **Barriers to Vaccination**

Barriers to implementing the vaccine include its high cost, a complex reimbursement process, the need for frozen storage, and availability. In addition, studies have indicated that the lack of a strong recommendation by a physician could be the biggest barrier, Dr. Cohen said.

Dr. Cohen and her team surveyed primary care physicians and consenting patients at the Bellevue Eye Clinic in Washington. Only 69% of the physicians said that the shingles vaccine was important, whereas 94% said the flu vaccine was important.

A second part of the survey compared 100 patients who were vaccinated with 66 eligible patients who chose not to be vaccinated. The demographic characteristics of the 2 groups were similar. The most common reason for refusing the vaccine was wanting to speak to their primary care physician first.

"We need to encourage doctors to recommend the vaccine. Ophthalmologists with a strong relationship with their patients are more likely to recommend it," she stated. In addition to prevention, treating herpes zoster ophthalmicus minimizes complications.

The Zoster Eye Disease Study (ZEDS) is currently enrolling 1050 patients with herpes onset within the previous 6 months, Dr. Cohen reported. The study will evaluate whether prolonged suppression with valacyclovir will reduce complications, including chronic ocular disease and postherpetic neuralgia. Patients will be randomized to 1 year of treatment with valacyclovir 1000 mg/day or placebo. Follow-up will be 18 months.

"More than 1 million cases of shingles occur in the United States each year, and herpes zoster ophthalmicus represents nearly 25% of all cases of shingles," said Kathryn Colby, MD, director of the AAO cornea program.

"Ophthalmologists need to accurately diagnose herpes zoster and recognize the general symptoms of shingles. Timely treatment is critical to minimize complications, protect vision, and reduce nerve pain that can cause patient suffering," said Dr. Colby, who is a cornea surgeon at Massachusetts General Hospital and practices at the Massachusetts Eye and Ear Infirmary in Boston.

Name

"Ophthalmologists can play an important role in ensuring that vulnerable patients are vaccinated," she noted. "Dr. Cohen's presentation highlights the renewed interest in herpes zoster infection, both in the eye and elsewhere," said Elmer Tu, MD, from the Department of Ophthalmology and Visual Science at the University of Illinois Eye and Ear Infirmary in Chicago. "This interest is due to the increased vaccination of children over the last nearly 20 years and the more recent introduction of a vaccine for adults over the age of 50." Dr. Tu noted that shingles is also increasing in those 20 to 50 years of age, but neither the pediatric nor adult version of the vaccine is indicated for this cohort.

"Now that we have additional interventions that have the potential to reduce the recurrence of herpes zoster infection, we are forced to re-examine not only the costs and benefits of vaccination, but the rationale and efficacy of our current treatment regimens," he explained.

"Because of the uncommon but potentially devastating effects of herpes zoster on the eye, the ophthalmologist plays a vital role in educating patients about the benefits of herpes zoster vaccination," he said.

As Dr. Cohen pointed out, "support for a nationwide study on the appropriate treatment of patients with active disease is needed to establish guidelines for the care of this potentially blinding disease," Dr. Tu noted. Dr. Cohen reports financial support from Merck for the study. Dr. Colby reports financial support from Novartis. Dr. Tu reports financial relationships with Bausch + Lomb.

http://bit.ly/1cSnUlz

Are Alzheimer's and diabetes the same disease? The link between obesity and dementia is becoming hard to deny Updated 11:51 28 November 2013 by Jessica Griggs

HAVING type 2 diabetes may mean you are already on the path to Alzheimer's. This startling claim comes from a study linking the two diseases more intimately than ever before.

There is some good news: the same research also offers a way to reverse memory problems associated with diabetes – albeit in rats – which may hint at a new treatment for Alzheimer's.

"Perhaps you should use Alzheimer's drugs at the diabetes stage to prevent cognitive impairment in the first place," says Ewan McNay from the University at Albany in New York.

Alzheimer's cost the US \$130 billion in 2011 alone. One of the biggest risk factors is having type 2 diabetes. This kind of diabetes occurs when liver, muscle and fat cells stop responding efficiently to insulin, the hormone that tells them to absorb glucose from the blood. The illness is usually triggered by eating too many sugary and high-fat foods that cause insulin to spike, desensitising cells to its presence.

As well as causing obesity, insulin resistance can also lead to cognitive problems such as memory loss and confusion.

In 2005, a study by Susanne de la Monte's group at Brown University in Providence, Rhode Island, identified a reason why people with type 2 diabetes had a higher risk of developing Alzheimer's. In this kind of dementia, the hippocampus, a part of the brain involved in learning and memory, seemed to be insensitive to insulin. Not only could your liver, muscle and fat cells be "diabetic" but so it seemed, could your brain.

Feeding animals a diet designed to give them type 2 diabetes leaves their brains riddled with insoluble plaques of a protein called beta-amyloid – one of the calling cards of Alzheimer's. We also know that insulin plays a key role in memory. Taken together, the findings suggest that Alzheimer's might be caused by a type of brain diabetes.

If that is the case, the memory problems that often accompany type 2 diabetes may in fact be early-stage Alzheimer's rather than mere cognitive decline.

Although there is no definitive consensus on the exact causes of Alzheimer's, we do know that brains get clogged with beta-amyloid plaques.

One idea gaining ground is that it is not the plaques themselves that cause the symptoms, but their precursors – small, soluble clumps of beta-amyloid called oligomers. The insoluble plaques could actually be the brain's way of trying to isolate the toxic oligomers.

To investigate whether beta-amyloid might also be a cause of cognitive decline in type 2 diabetes, McNay, Danielle Osborne and their colleagues fed 20 rats a high-fat diet to give them type 2 diabetes. These rats, and another 20 on a healthy diet, were then trained to associate a dark cage with an electric shock.

Whenever the rats were returned to this dark cage, they froze in fear – measuring how long they stayed still is a standard way of inferring how good their memory is.

Name

Memory boost

As expected, the diabetic rats had weaker memories than the healthy ones – they froze in the dark for less than half the time of their healthy counterparts. To figure out whether this was due to the beta-amyloid plaques or the soluble precursors, Pete Tessier at the Rensselaer Polytechnic Institute in Troy, New York, engineered fragments of antibodies that disrupt the action of one or the other.

When the plaque-disrupting antibodies were injected into diabetic rats, no change was seen. However, after receiving antibodies specific for oligomers, they froze for just as long as the healthy rats. "The cognitive deficit brought on by their diabetes is entirely reversed," says McNay.

Until now, the standard explanation for the cognitive decline associated with type 2 diabetes is that it is a result of insulin signalling gone awry. One effect is to reduce the hippocampus's ability to transport energy, or glucose, to neurons during a cognitive task. The fact that amyloid builds up in the brains of diabetic animals – and also in people, was seen as an unhappy consequence of insulin imbalance.

These experiments suggest oligomers are actually to blame. Previous work from other groups has shown that the same enzymes break down both insulin and beta-amyloid oligomers – and that the oligomers prevent insulin binding to its receptors in the hippocampus. So when there is too much insulin around – as there is in someone with type 2 diabetes – those enzymes are working flat out to break it down.

This preferential treatment of insulin leaves the oligomers to form clumps, which then keep insulin from its receptors, causing a vicious spiral of impaired brain insulin signalling coupled with cognitive decline.

"We think that our treatment soaked up the amyloid oligomers, so that they could no longer block insulin from binding to its receptors," says McNay, who presented the preliminary data at the Society for Neuroscience meeting in San Diego earlier this month. "Everyone thinks of amyloid build-up as a consequence of the events that cause cognitive impairment in diabetes, but we're saying it's actually a cause." It means, he says, that the cognitive decline seen in type 2 diabetes may be thought of as early-stage Alzheimer's.

It's a bold claim, and if correct, one with big implications. Given that the number of people with type 2 diabetes is expected to jump from 382 million now to 592 million by 2035, we might expect to see a similar trajectory for associated Alzheimer's (New Scientist, 1 September 2012).

If beta-amyloid build-up can be stopped in people with type 2 diabetes and their cognitive impairment reversed – perhaps many of them will never progress to Alzheimer's.

For the last few years, organisations like the UK's Alzheimer's Society have been backing clinical trials to look for diabetes drugs that may have an effect on Alzheimer's patients. "We're saying that this may be not the only way to think about it," says McNay.

The next step is to repeat the work, and if the results are corroborated, start looking for a drug that would do the same thing as the group's modified antibodies, without having to inject the drug directly into the hippocampus. It will also be necessary to work out just how much amyloid the brain can safely do without, since low levels are important for memory formation.

"The work opens the door to inoculating the most at risk group, people with type 2 diabetes," says Tres Thompson of the University of Texas at Dallas. There have been plenty of failed attempts to use antibodies to relieve Alzheimer's in the past. "But these were all in people with advanced stages of the disease. Vaccinating people much earlier could give better results."

Some researchers are still wary of focusing on beta-amyloid when 20 years of working on a treatment for that particular aspect of the disease has come to nothing.

"I think it's brilliant work – he's using new techniques that seem to be working, but it's still very beta-centric," says Olivier Thibault at the University of Kentucky in Lexington.

He cautiously agrees that McNay's data do seem to suggest a causative link between beta-amyloid and impaired insulin signalling but says the group needs to factor in the effect of ageing – both diabetes and Alzheimer's become more likely as we grow older.

Jessica Smith, spokeswoman for the UK Alzheimer's Society in London welcomes the work. "We need to tease out the difference between those with type 2 diabetes who develop Alzheimer's and those who don't. If people were developing the signs earlier than we thought, then perhaps we can intervene earlier, rather than waiting until they have full clinical Alzheimer's."

Of course, there is another solution to staving off type 2 diabetes and any consequential Alzheimer's that requires no drugs at all. "Go to the gym and eat fewer twinkies," says McNay.

If diabetes causes Alzheimer's, we can beat it

Evidence is growing that Alzheimer's could actually be a late stage of type 2 diabetes – if it is, we all have another big reason to live healthier lives

JUST over 100 years ago, German pathologist Alois Alzheimer dissected the brain of a 57-year-old woman who had died, demented, in a hospital in Kassel. He found tangles of strange fibrous deposits that seemed to have destroyed her brain from within.

Today, the disease that bears his name is a bogeyman stalking our ageing societies. About 35 million people have Alzheimer's; most of them require expensive, exhausting care. By 2050 that number is expected to triple. We still don't really know what causes the disease or how it destroys the brain. There is no way to prevent it and no cure. Dealing with the epidemic will cost trillions.

All it not lost, however. We could be in the midst of a rethink that promises to banish the bogeyman. There is growing evidence that Alzheimer's is actually a late stage of another disease, type 2 diabetes. The link between the two has been noted for a few years and though it remains a hypothesis, the evidence is growing (see "Are Alzheimer's and diabetes the same disease?").

At first glance that sounds like bad news. If the Alzheimer's epidemic is scary, the type 2 diabetes one is truly terrifying. About 270 million people have type 2 diabetes already and their ranks are swelling rapidly – among them adolescents and young adults. If they are destined to progress to Alzheimer's disease, the future looks bleak.

Or perhaps not. Type 2 diabetes is largely a lifestyle disease, caused by obesity, poor diet and lack of exercise. It can be prevented, alleviated and even cured by lifestyle changes, which holds out the hope that we could start to deal with Alzheimer's in a similar way.

Experience tells us, of course, that exhorting people to eat better and exercise more often falls on deaf ears. But with obesity rates levelling off in some parts of the world and falling slightly in others, there is some evidence that the message is getting through.

If the link between diabetes and Alzheimer's is firmed up, there will be even more reason to take heed – and even more reason to keep banging the public health drum. Good news comes in many guises. The possibility that Alzheimer's is "just" diabetes is one of them.

http://bit.ly/1bxEhr6

Oxygen drop makes people with spine injury more mobile

Ask any mountaineer or miner and they'll tell you that time in a low-oxygen environment can be a dangerous business – it can cause altitude sickness and other health problems. But for people with spinal injuries, short periods of hypoxia may be a promising therapy for regaining mobility. 21:00 27 November 2013 by Alyssa Botelho

About 59 per cent of people who experience trauma to the spinal cord suffer incomplete injuries, meaning that some neural pathways between their brain and the neurons that extend to their limbs remain intact. "Strengthening those weak, lingering connections is key to regaining movement," says engineer Randy Trumbower, at Emory University in Atlanta, Georgia. But, often, those networks cannot be fully activated through physical therapy alone. Keen on finding a better way to awaken those dormant connections, he teamed up with Gordon Mitchell at the University of Wisconsin in Madison, and his colleagues, who were studying the effects of sleep apnoea – or interrupted breathing – in mice.

Base camp to summit

Mitchell had found that periodic exposure to low oxygen levels triggers the release of the brain chemical serotonin. This in turn prompts the production of a growth factor that coaxes new connections to form between neurons present in the brainstem and spinal cord that regulate breathing. Trumbower wondered whether there was a way to control the release of this neural growth factor to rejuvenate an injured spine. The idea led his team to run the first test looking into the benefit of controlled hypoxia on people with neurological conditions. The team recruited 19 people with a wide variety of spinal cord injuries, all of whom had difficulty moving around for long periods of time without assistance – from a walker for example – but could walk at least one step unassisted. Half of the subjects wore a breathing mask for 40 minutes a day for five days, alternating between inhaling 9 per cent oxygen for 90 seconds and normal air with 21 per cent oxygen for 60 seconds. "If you were to convert 9 per cent oxygen to altitude, you can think of yourself being at about 26,000 feet – at Mount McKinley but not quite Mount Everest," says Trumbower. He adds that participants do not experience discomfort because they are not exposed to the hypoxic air for long. "You're continuously alternating between the summit and base camp," he says.

15 Faster and further

At the end of the five days, the participants who received low-oxygen treatment walked a 10-metre course an average of 3.8 seconds faster than those who had inhaled air with normal oxygen levels.

Name

The team also found that subjects who attempted to walk for 30 minutes after each of their daily treatments not only walked faster at the end of the regimen, but also walked farther – averaging an extra 100 metres during a 6 minute walk, compared with those who walked for 30 minutes each day after receiving normal oxygen.

"It may seem a little counterintuitive, but these findings make very good sense," says neurosurgeon Michael Fehlings at the University of Toronto, Canada. "Because hypoxia acts as a stress to the nervous system, it can elicit a beneficial response if controlled correctly."

He says he is curious to see if the treatment will provide benefit for people who have more severe spinal cord trauma and cannot walk unassisted.

Although the therapeutic effects from this trial did not last for more than two weeks, Trumbower is hopeful that longer treatment regimens will produce lasting effects.

"Our hope is that this therapy can be used for months, not just days, and enhance the improvements of physical therapy."

Journal reference: Neurology, in press

http://phys.org/news/2013-11-team-quantifies-difficulties.html

Research team quantifies 'the difficulties of reproducibility'

Scientific reproducibility is not as common or as easy as many non-scientists think Phys.org - A key pillar of "the scientific method" is reproducibility, one way to prove another scientist's experimental claims. If the experiment and its results can be reproduced, the validity of the work is considerably strengthened.

But scientific reproducibility is not as common or as easy as many non-scientists think. In a recent study of landmark papers in cancer research, for example, only 11 percent of the studies could be reproduced. In another recent case, a graduate student failed to reproduce the results of a widely cited economic-policy paper – a failure which led to the exposure of significant, but unintentional, errors.

Hoping to quantify just what it takes to reproduce a scientific paper, researchers from three institutions conducted a study of a computational biology paper that analyzed tuberculosis-drug targets.

Philip Bourne, professor of pharmacology at the Skaggs School of Pharmacy and Pharmaceutical Sciences at the University of California San Diego, the principal investigator of the tuberculosis study and co-author of the paper; Daniel Garijo, a doctoral student from the Universidad Politecnica of Madrid; and Yolanda Gil, professor of computer science at the University of Southern California, collaborated to quantify "the difficulties of reproducibility" - and to suggest a possible solution.

Writing in the journal PLOS ONE, Gil and Garijo reported that they had to spend "significant time" reviewing materials from Bourne's lab, and talking to previous lab members, to satisfactorily reconstruct the computational experiments of the original paper.

"We estimated the overall time to reproduce the method at 280 hours for a novice with minimal expertise in bioinformatics," said Garijo, "either because computer scripts were not available, or there were assumptions in the described methods that would not be obvious to a non-expert."

Failure to reproduce a study is rarely the result of fraud, said Bourne, but "mostly lack of a complete record." In this case, he said, "it was not that the work could not be reproduced; the problem was that it took so much time - something all new graduate students in the lab can verify as they pick up previous students' work."

In this day and age, said Bourne, "We should really be doing better. It's unfortunate to say this about my own work – but how many scientists could claim to be doing better?"

One way scientists might do better, said Gil, is to do what she and Garijo did. "As part of the reconstructive work," she said, "we encoded the computational experiment in a semantic workflow, shared as a web object with annotations of its meanings."

These workflow systems are now reaching such a level of maturity, say the researchers, that they're likely to be adopted more broadly. "This should greatly facilitate reproducibility," their report asserts.

Journals and their publishers can also encourage improved reproducibility by insisting that workflows, data, and software to be part of the submission-and-review process, the authors say.

Finally, they note, better reproducibility may eventually be mandated, citing a recent administration memorandum asking all agencies to develop policies to make results of all federally funded research broadly available to scientists, industry, and the public.

Researchers find a missing component in effort to create primitive, synthetic cells *Investigators working to create "protocells" have accomplished an important step towards their goal* A team of Massachusetts General Hospital (MGH) investigators working to create "protocells" – primitive synthetic cells consisting of a nucleic acid strand encased within a membrane-bound compartment – have accomplished an important step towards their goal. In the November 28 issue of Science, the investigators describe a solution to what could have been a critical problem – the potential incompatibility between a chemical requirement of RNA copying and the stability of the protocell membrane.

"For the first time, we've been able to do nonenzymatic RNA copying inside fatty acid vesicles," says Jack Szostak, PhD, of the MGH Department of Molecular Biology and the Center for Computational and Integrative Biology. "We've found a solution to a longstanding problem in the origin of cellular life: RNA copying chemistry requires the presence of the magnesium ion Mg2+, but high Mg2+ levels can break down the simple, fatty acid membranes that probably surrounded the first living cells."

Szostak's team has been working for more than a decade to understand how the first cells developed from a "primordial soup" of chemicals into living organisms capable of copying their genetic material and reproducing. Part of that work is developing a model protocell made from components probably present in the primitive Earth environment. They have made significant progress towards developing cell membranes from the kind of fatty acids that would have been abundant and naturally form themselves into bubble-like vesicles when concentrated in water. But the genetic component – an RNA or DNA molecule capable of replication – has been missing.

Since the primitive environment in which such cells could have developed would not have had the kind of complex enzymes that modern cells use in replicating nucleic acids, Szostak and lead author Katarzyna Adamala, PhD, then a graduate student in Szostak's lab, investigated whether simple chemical processes could drive nonenzymatic replication of RNA, which many scientists believe was the first nucleic acid to develop. To address the incompatibility between the need for Mg2+ to drive assembly of the RNA molecule and the ion's ability to degrade fatty acid membranes, they tested several chelators – small molecules that bind tightly to metal ions – for their ability to protect fatty acid vesicles from the potentially destabilizing effects of Mg2+. Citrate and several other chelators were found to be effective in protecting the membranes of fatty acid vesicles from disruption.

To test whether the presence of the tested chelators would allow Mg2+-catalyzed RNA assembly, the investigators placed molecules consisting of short primer RNA strands bound to longer RNA templates into fatty acid vesicles. The unbound, single-strand portion of the template consisted of a sequence of cytosine (C) nucleotides. In the presence of Mg2+ and one of four chelating molecules, one of which was citrate, the researchers then added activated G, the nucleotide that base-pairs with C in nucleic acids.

The desired reaction – diffusion of G nucleotides through the vesicle membrane to complete a double-stranded RNA molecule by binding to the C nucleotides of the template – proceeded fastest in the presence of citrate. In fact two of the other tested chelators completely prevented extension of the RNA primer.

"While other molecules can protect membranes against the magnesium ion," Szostak explains, "they don't let RNA chemistry go on. We think that citrate is able both to protect membranes and to allow RNA copying to proceed by covering only one face to the magnesium ion, protecting the membrane while allowing RNA chemistry to work." He and Adamala also found that continually refreshing the activated guanine nucleotide solution by flushing out broken down molecules and adding fresh nucleotides improved the efficiency of RNA replication.

Szostak notes that, while citrate may be appropriate for creating artificial cells in a laboratory environment, which he and his team are pursuing, it probably would not have been present in sufficient quantities in the early earth. "We have shown there is at least one way to make RNA replication chemistry compatible with primitive, fatty-acid-based cell membranes, but this opens up new questions. Our current best guess is there must have been some sort of simple peptides that acted in a similar way to citrate, and finding such peptides is something we are working on now."

A co-recipient of the 2009 Nobel Prize in Physiology or Medicine for his contribution to the discovery of the enzyme telomerase, Szostak is a professor of Genetics at Harvard Medical School and a Howard Hughes Medical Institute investigator. Adamala, who worked in Szostak's lab as part of her doctoral studies at Roma Tre University in Italy, is now a postdoctoral fellow at Massachusetts Institute of Technology. The study was supported, in part, by NASA Exobiology grant NNX07AJ09G.

Memories are 'geotagged' with spatial information, Penn researchers say Brain cells that encode spatial information form "geotags" for specific memories and are activated immediately before those memories are recalled

Using a video game in which people navigate through a virtual town delivering objects to specific locations, a team of neuroscientists from the University of Pennsylvania and Freiburg University has discovered how brain cells that encode spatial information form "geotags" for specific memories and are activated immediately before those memories are recalled.

Their work shows how spatial information is incorporated into memories and why remembering an experience can quickly bring to mind other events that happened in the same place.

"These findings provide the first direct neural evidence for the idea that the human memory system tags memories with information about where and when they were formed and that the act of recall involves the reinstatement of these tags," said Michael Kahana, professor of psychology in Penn's School of Arts and Sciences.

The study was led by Kahana and professor Andreas Schulze-Bonhage of Freiberg. Jonathan F. Miller, Alec Solway, Max Merkow and Sean M. Polyn, all members of Kahana's lab, and Markus Neufang, Armin Brandt, Michael Trippel, Irina Mader and Stefan Hefft, all members of Schulze-Bonhage's lab, contributed to the study. They also collaborated with Drexel University's Joshua Jacobs.

Their study was published in the journal Science.

Kahana and his colleagues have long conducted research with epilepsy patients who have electrodes implanted in their brains as part of their treatment. The electrodes directly capture electrical activity from throughout the brain while the patients participate in experiments from their hospital beds.

As with earlier spatial memory experiments conducted by Kahana's group, this study involved playing a simple video game on a bedside computer. The game in this experiment involved making deliveries to stores in a virtual city. The participants were first given a period where they were allowed to freely explore the city and learn the stores' locations. When the game began, participants were only instructed where their next stop was, without being told what they were delivering. After they reached their destination, the game would reveal the item that had been delivered, and then give the participant their next stop.

After 13 deliveries, the screen went blank and participants were asked to remember and name as many of the items they had delivered in the order they came to mind.

This allowed the researchers to correlate the neural activation associated with the formation of spatial memories (the locations of the stores) and the recall of episodic memories: (the list of items that had been delivered).

"A challenge in studying memory in naturalistic settings is that we cannot create a realistic experience where

the experimenter retains control over and can measure every aspect of what the participant does and sees. Virtual reality solves that problem," Kahana said. "Having these patients play our games allows us to record every action they take in the game and to measure the responses of neurons both during spatial navigation and then later during verbal recall."

By asking participants to recall the items they delivered instead of the stores they visited, the researchers could test whether their spatial memory systems were being activated even when episodic memories were being accessed. The map-like nature of the neurons associated with spatial memory made this comparison possible.

"During navigation, neurons in the hippocampus and neighboring regions can often represent the patient's virtual location within the town, kind of like a brain GPS device," Kahana said. "These so-called 'place cells' are perhaps the most striking example of a neuron that encodes an abstract cognitive representation."



Using a video game in which people navigate through a virtual town delivering objects to specific locations, a team of neuroscientists from the University of Pennsylvania and Freiburg University has discovered how brain cells that encode spatial information form "geotags" for specific memories and are activated immediately before those memories are recalled. Their work shows how spatial information is incorporated into memories and why remembering an experience can quickly bring to mind other events that happened in the same place. This overhead map of the virtual city is overlaid with the areas where a participants place cells were activated. Different colors represent different cells, showing how these neurons help track spatial information and memories. University of Pennsylvania

_Student number

Using the brain recordings generated while the participants navigated the city, the researchers were able to develop a neural map that corresponded to the city's layout. As participants passed by a particular store, the researchers correlated their spatial memory of that location with the pattern of place cell activation recorded. To avoid confounding the episodic memories of the items delivered with the spatial memory of a store's location, the researchers excluded trips that were directly to or from that store when placing it on the neural map. With maps of place cell activations in hand, the researchers were able to cross-reference each participant's spatial memories as they accessed their episodic memories of the delivered items. The researchers found that the neurons associated with a particular region of the map activated immediately before a participant named the item that was delivered to a store in that region.

"This means that if we were given just the place cell activations of a participant," Kahana said, "we could predict, with better than chance accuracy, the item he or she was recalling. And while we cannot distinguish whether these spatial memories are actually helping the participants access their episodic memories or are just coming along for the ride, we're seeing that this place cell activation plays a role in the memory retrieval processes."

Earlier neuroscience research in both human and animal cognition had suggested the hippocampus has two distinct roles: the role of cartographer, tracking location information for spatial memory, and the role of scribe, recording events for episodic memory. This experiment provides further evidence that these roles are intertwined.

"Our finding that spontaneous recall of a memory activates its neural geotag suggests that spatial and episodic memory functions of the hippocampus are intimately related and may reflect a common functional architecture," Kahana said.

The research was supported by the U.S. National Institutes of Health, the German Research Foundation and Germany's Federal Ministry of Education and Research.

http://phys.org/news/2013-11-reversible-wound-closure-dissolvable-dendritic.html

Reversible wound closure: Dissolvable dendritic thioester hydrogel for sealing wounds *In first-aid situations, wounds must be quickly and effectively closed to stop blood loss and prevent infection.*

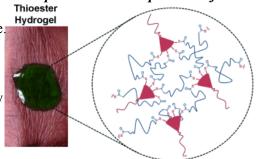
For treatment on arrival in a hospital, the temporary seal must be reopened, which often causes additional damage to the injured tissue. In the journal Angewandte Chemie, American scientists have now introduced a novel gel for sealing wounds. The gel can later be dissolved and gently removed.

Injuries sustained in remote areas, far from civilization, or in military action can often not be treated in a clinic until hours later. In such scenarios, a temporary wound closure system is desirable. Such a system should: 1) stop the bleeding for several hours, 2) adhere to the tissue, 3) be easy to apply, and 4) be easily removable in a

controlled manner to make the wound accessible during surgical treatment. No single wound-closure systems currently available meet all of these requirements. Removal of blood-clotting agents or dressings requires tearing or surgical excision, both of which can increase the size of the wound and make it worse. Scientists working at Boston University and the Beth Israel Deaconess Medical Center in Boston have now developed a wound-closure system based on a synthetic biocompatible gel that meets the requirements listed above. The gel is cross-linked through branched thioesters. The team, led by Mark W. Grinstaff, uses a chemical reaction known as thiol-thioester exchange in order to dissolve these gels for removal. In this reaction, a thioester bond reacts with a thiolate anion to produce new thioester and thiolate products. The advantage of this reaction is that it takes place in an aqueous environment under physiological conditions. This type of reaction also occurs in natural biological processes. When the thioester gel is treated with cysteine methyl ester, the thioester bridges are rapidly split and the gel dissolves.

Wounds may be treated by simply mixing and applying two starting materials. The gel forms within seconds, adheres to the skin even when stress is applied and remains intact for several days. The gel absorbs any liquid exiting the wound. Treatment with cysteine methyl ester causes the wound closure to reopen within 30 minutes. To simulate injury to a vein, the researchers filled a section of bovine jugular vein with buffer solution and punctured it. Once the gel was applied, the damaged vein was completely sealed; after dissolution of the gel, the buffer solution flowed out again.

More information: Mark W. Grinstaff, A Dendritic Thioester Hydrogel Based on Thiol–Thioester Exchange as a Dissolvable Sealant System for Wound Closure, Angewandte Chemie International Edition, dx.doi.org/10.1002/anie.201308007



Study suggests why, in some species, mere presence of males shortens females' lifespan Researchers at the Stanford University School of Medicine have discovered that males of the laboratory roundworm secrete signaling molecules that significantly shorten the lifespan of the opposite sex.

The scientists speculate that, if carried out after reproduction, this "male-induced demise" could serve to conserve precious resources for a male's offspring or to decrease the supply of mates for other males. For several years, it's been known that the presence of some male worms and flies can shorten the lifespan of their female or hermaphroditic counterparts. But it's not been clear why. Some researchers have speculated that the physical stress of mating may lead to their early death.

The Stanford research, however, suggests something more than sex is to blame - specifically, that the males are carrying out a calculated plan at the molecular level to off the baby-makers after they've done their jobs. The researchers studied the common laboratory roundworm, known as Caenorhabditis elegans, or C. elegans. The 1-millimeter-long, translucent worms generally live for about 20 days, and a normal population consists of

about 0.01 to 0.1 percent males. The remainder consists of hermaphrodites, which have both male and female reproductive organs. Although hermaphrodites can self-fertilize, they can produce more offspring if they mate with a male.

"We've found that males induce the expression of a large number of genes involved in sensation and signaling in hermaphrodites," said Anne Brunet, PhD, associate professor of genetics. "This raises the possibility that the male-induced demise is not just due to the physical stress of copulation but instead involves some degree of active signaling. Indeed, we found that just placing hermaphrodites on plates where males had previously been present was sufficient to induce the premature demise of hermaphrodites."

Brunet is the senior author of the study, which will be published Nov. 28 in Science Express. Postdoctoral scholar Travis Maures, PhD, is the lead author.

Brunet's research shows that males can initiate the killing process even across distances. But as tempting as it is to extend the findings to mammals and - dare we say it? - humans, it would likely backfire in situations where mothers, or parents, are needed to rear the young.

"In worms, once the male has mated and eggs are produced, the hermaphrodite mother can be discarded," Brunet said. "The C. elegans mother is not needed to care for the baby worms. Why should it be allowed to stay around and eat? Also, if she dies, no other male can get to her and thus introduce his genes into the gene pool." The researchers found that the continuous presence of young males shortened the average lifespan of C. elegans hermaphrodites by more than 20 percent. This effect persisted even when the genders were prevented from comingling, or when the hermaphrodites were sterile - indicating that neither the physical stress of copulation nor the energy demands of producing offspring were entirely responsible for early death. Affected hermaphrodites also displayed symptoms of aging, including slower movement, an increased incidence of paralysis, general decrepitude and structural decline.

"Even long-lived and stress-resistant hermaphrodites were highly susceptible to the premature demise induced by males," said Brunet, who specializes in the study of longevity in worms and other animals.

Finally, when the researchers placed hermaphrodites on laboratory dishes that had formerly contained male worms, those hermaphrodites also exhibited a shortened lifespan, indicating that the males had left behind some substance that was affecting the hermaphrodites.

There was one way to ameliorate the effect, however.

"Males that are deficient in pheromone production no longer induce a strong premature demise of hermaphrodites," Brunet said, "and hermaphrodites that cannot sense pheromones are resistant to male-induced demise."

Pheromones are soluble, diffusible chemical compounds produced by many animals to trigger social or behavioral reactions among members of the same species across distances. Although it's as yet unclear how or when the roundworm males secrete the unidentified pheromones, the effect was evident when the researchers investigated the gene expression profiles of the affected hermaphrodites.

In particular, they observed large changes in hermaphrodite gene expression that occurred only in the presence of males. Many of these changes affected genes expressed in neurons or involved in neurodegenerative diseases. Blocking the expression of one gene in particular, an insulin-like peptide known as INS-11, specifically impeded male-induced demise.

The studies conducted by the researchers in C. elegans used a much higher proportion of males to hermaphrodites than normally occurs. However, Brunet feels the effect of even a few males may be exacerbated once mating has occurred - particularly if the unknown pheromones are secreted in the seminal fluid. The effect

on non-mating populations (the hermaphrodites added to the plate after the males have been removed) could be explained by the seminal fluid secreted by the males that, without access to nearby hermaphrodites, attempt unsuccessfully to copulate with other, nearby males.

Name

Although the researchers first studied a domesticated strain of C. elegans, they were also able to observe maleinduced demise in a wild strain of C. elegans, as well as in two other, distantly related species of worm confirming that the phenomenon has been conserved over about 20 to 30 million years of evolution. The maleinduced demise even occurred in species of roundworm that have true males and true females in an equal mix (similar to mammals), suggesting that this phenomenon is not just due to idiosyncrasies of C. elegans such as hermaphroditism or a low proportion of males.

"The observation that this male-induced demise is present in several species of worms and has also been shown in flies suggests that it could have some adaptive benefits," Brunet said. "It will be interesting, of course, to determine whether males also affect the lifespan of females in other species, particularly mammals." *"Males Shorten the Life Span of C. elegans Hermaphrodites via Secreted Compounds," by T.J. Maures et al. Science, 2013.*

http://www.bbc.co.uk/news/science-environment-25129117

'Love-test' identifies newly-weds true feelings

Scientists have devised a new "love test" that they believe is a better guide to the success of a relationship than the good intentions of newly-weds.

By Pallab Ghosh Science correspondent, BBC News

The research suggests that a subconscious response to an image of a partner could be a useful predictor of marriage outcomes. Those who had a negative gut reaction were more likely be unhappy several years later. The study is published in the Journal Science.

The lead author, Prof James McNulty from Florida State University, says that the new test gauges the true feelings of newly-weds towards each other, rather than what they say to other people or even admit to themselves. "These immediate gut level responses seem to be pretty powerful in predicting whether people stay happy," he told BBC News.

His team interviewed 135 newly-wed couples just after their nuptials. The researchers asked them to evaluate their marriage related to positive and negative adjectives such as "good", "bad", "satisfying" and "dissatisfying". They then measured their gut reaction to each other using their intriguing "love test".

This involved showing one partner a photograph of the other for a fleeting third of a second. They then had to answer as quickly as possible, whether certain words such as "great", "awesome", "horrible" and "scary" were positive or negative words.

The speed with which they answered was an indication of their true feelings, say the researchers.

The test is based on the psychological principle of association. The theory is that after fleetingly seeing a picture of their partner, the newlywed is in a positive or negative state of mind.

Awesome or scary?

If they are in a positive state of mind they will identify positive words such as "great" or "awesome" more quickly than negative words such as "scary and horrible" and vice versa. Prof McNulty and his team found that the conscious answers of the newly-weds were all positive and very happy about their relationships, as you might imagine. But the gut reactions from the love test varied considerably.

The researchers interviewed the couples every six months for the next four years. They found that on average, those who had negative gut reactions were more likely to say that they were unhappy as the marriage wore on. Some even divorced.

"Everyone wants to believe they are in a good relationship and people can convince themselves that they are but these gut-level reactions are more indicative of how people feel immediately about their relationships," he said.

The test, according to the authors, measures the presence or absence of negative emotions. "People can have love and negative emotions at the same time and this test probably taps into both of those," said Prof McNulty. However, he was at pains to state that the research was not developed enough to be able to offer it to people before they tie the knot.

He pointed out that overall the scientists found a trend, but some of those who had a negative response stayed happy, while others who had a positive gut reaction became unhappy.

For those about to take the leap, Prof McNulty said that gut reaction could be something they listen to. "I think the best advice would be to attend to your gut level responses about how you think about seeing your partner. I don't think that should be the only factor people should consider, but it should be one of them" Experts 'prefer cheaper champagne'

Champagne experts prefer a £40 brand to one costing £400, a study indicates.

By Victoria Gill Science reporter, BBC News

Scientists from the Universities of Oxford and London carried out blind taste tests. They asked the participants to score six different champagnes and one English sparkling wine - ranging in price from £18 to £400. The champagne experts who took part scored the £40 bottle most highly. The results are published in Flavour Journal.

The panel of tasters included:

four experts, who work in the champagne industry six "intermediates", who work in the wine trade but do not specialise in champagne five novice champagne tasters, or "social drinkers"

As well as disguising the bottles, the researchers gave the participants black tasting glasses to disguise the colour of the champagne. The primary aim of the study was to work out if tasters could estimate the relative proportions of red or white grapes. This ratio is said to give each champagne and sparkling wine a distinctive flavour. As well as finding that the experts were not able to accurately identify this ratio, the study also suggested preference was unrelated to cost.

Prof Charles Spence, from the University of Oxford, who led the study, told BBC News: "We found no correlation between the price and how much the participants liked the drink. "Experts and intermediates preferred a £40 bottle of champagne. "And in fact the novices - the social drinkers - preferred a slightly more expensive £75 bottle of champagne."

Champagne chefs

Prof Spence thinks the results suggest the cellar masters or "chefs de cave" who create the final blend of champagne are so skilled they can create blends that taste like "more than the sum of their parts". But, he added, the study did not necessarily mean people were wasting their money if they bought expensive champagne. There was, he said, a kind of placebo effect with cost.

"If you know how much something costs - the psychology of that high cost seems to make things taste better," said Prof Spence. "It might be that you're paying more for certain characteristics that are interesting, but not necessarily linked with preference."

James Hutchinson, a wine expert from the Royal Society of Chemistry, pointed out that, with just 15 participants, this was "quite a small study". But, he said, it did show the complexity and subtlety of champagne and sparkling wine. "They're produced in colder climates, so you don't get the big, robust fruity flavour [from] very ripe fruit aromas," he said. "Even the experts can have a hard time finding those subtle differences." A high price, he added, was linked with many things aside from flavour, including rarity, reputation of the winemaker, and even how big the marketing budget was. "But ultimately wine is very complex - there are 400 different chemical compounds in wine," he said. "For champagne, I would say that complexity of flavour is an indication of something amazing. "Something that feels hand-crafted - with lots of layers of flavour."

even more options onto the market that cost much less than champagne.

"There are lots of great sparkling wines out there, so I'd suggest that people try different things," he said.

http://phys.org/news/2013-11-astro-virology.html

Astro-virology

In HG Wells' 'The War of the Worlds', the invading Martians were beaten by that most unassuming of combatants – the common cold. Could the reverse happen and alien viruses pose a threat to human astronauts when they land on Mars?

This intriguing question is asked by Dale Griffin in a new paper for the journal Astrobiology, who also asks whether our first evidence of extraterrestrial life could come in the shape of viruses.

Biologists do not consider viruses to be alive. They are smaller than bacteria (20-300 nanometers compared to 500-1500 nanometers) and cannot replicate on their own - instead they must invade a host cell and use its genetic tools to aid its replication. Yet viruses completely dominate the planet - hypochondriacs might tremble at the fact that there are ten million trillion trillion viruses existing on Earth right now, with a tenth of them found in the oceans. Given their total dependence on cellular life in order for them to replicate, it is little surprise that wherever life is found on our planet, viruses are right there with them.

Griffin, a microbiologist at the US Geological Survey at St Petersburg in Florida, thinks that we can expect to find a similar situation where life exists on other planets. "I would think that the evolution of cellular life on

Student number

another planet would be very similar to what occurred on Earth," he says. "If this is the case and cellular life is present on any given planetary body then I would expect viruses to also be present in superior numbers." Yet he doesn't believe that astrobiologists have fully cottoned onto this fact and that exo-viruses are just as viable a topic for speculative study as extraterrestrial cellular life. The reason for this may be partly because the study of viruses on Earth beyond those that cause illness in humans and animals has really only taken off in the last decade or so. As Stedman alludes to, studying viruses is not easy.

"It is only recently in the historical microbiology record that we've had the molecular tools that have allowed us to determine the number and the extent of the diversity of viruses on Earth," says Griffin. Part of the problem is that viruses on Earth have for the most part evolved symbiotic relationships with specific hosts - it's the reason you usually cannot catch a cold from a dog, for example, as the viruses that cause colds in dogs have developed to work with canine cells, and vice versa. So to really study viruses in detail one must culture a host cell, usually a bacterium, in the laboratory and for many viruses we have not identified their host(s). This has slowed research into the vast range and diversity of viruses on Earth considerably. Says Professor Chris Impey of the University of Arizona, who has written several books on astrobiology, "Because of the difficulty of culturing most bacterial species, we're still ignorant of the full range of symbiotic relationships between bacteria and viruses."

Now that things are changing, Griffin thinks it is time to consider extraterrestrial viruses. Stedman agrees. "Life on Earth is clearly heavily influenced by viruses," he says. "The jury is still out on whether viruses are essential for life, but certainly life on Earth would be very different without viruses. Finding life in the absence of viruses would surprise me, but would be very interesting."

The key point, Griffin argues, isn't that viruses will exist where life also exists, as we will have noticed that life long before we find the accompanying viruses, but rather that multiple host types of viruses could be found at both the beginning and the end stages of life on a planet.

Nobody really knows when viruses first developed on Earth, but the smart money suggests they are very ancient and could even have given evolution the required push towards cellularity. When a virus invades a cell, it brings with it its own genetic material and can add it to the cell's genome. When the virus replicates inside the cell, it takes some of the cell's genetic information with it, transferring it from cell to cell, organism to organism. Hence genes are swapped around, driving evolution.

Of course, viruses can cause damage but they can also bring benefits to the cell. For instance, if the cell has been damaged by ultraviolet light, a virus carrying ultraviolet-resistant genes can introduce those genes to the damaged cell, helping it to repair. Vice-versa, damaged viruses can have their replicating abilities restored if a cell is infected by multiple viruses that swap enough genetic information to produce a complete undamaged viral genome.

This makes viruses extremely hardy. "Viruses are robust and adaptable and they may be able to survive a long time in a dormant state," says Impey. Although viruses are inert outside of host cells, on Earth they have been seen to survive in extreme conditions. For example, viruses have been found in hot spring water in Yellowstone National Park in temperatures reaching 93 degrees Celsius. Conversely, viruses have been discovered in briny sea-water at minus 12 degrees Celsius, while the influenza virus is stored in secure laboratories at temperatures as low as minus 70 degrees, with no apparent damage to the virus itself. Nor do they require water to survive outside of the cell – they simply remain inert down and assuming no damage from radiation for example, they just re-awaken when they invade a new cell.

What happens however on a planet where all life has long ago disappeared? This may be the case for Mars, although it remains to be seen whether life did ever exist in the brief warm and wet periods of the red planet's ancient history. Let us suppose that Mars did once harbor primitive microbial life, with the requisite viruses accompanying it. As we have seen, on Earth most viruses are host specific – Griffin argues that the same would hold for extraterrestrial viruses too. However, when Martian life died off, or at least became rare, viruses would have been faced with a problem. If they remained host specific, they would disappear along with their hosts. If, however, the adapted and became generalized they could inhabit whatever cells they came across, sharing genetic information to survive at a subsistence level.

As such, if there was once life on Mars, today all that may be left could be the generalized viruses, capable of infecting most cells they come across. These viruses could therefore present a biohazard for any future astronauts that land on Mars and perhaps while we are looking for life on Mars, we should also equip our robotic rovers to search for viruses too.

Griffin has some ideas about how we may begin searching for extraterrestrial viruses. One tool would be a MEMS-based concentrator [Micro-Electro-Mechanical Systems] which is a micron-sized technology that can be used in chromatography [the separating of mixtures in a laboratory setting] and spectroscopy techniques.

This could be used in conjunction with particle-sized separators, microscopic imaging systems and nucleic sequencers to sift through the Martian dirt and analyze it for virus-like structures and nucleics.

"This approach would allow you to screen samples for cellular life, to image any virus-like particles or cells that may be present - are they similar to known variants on Earth? - and to sequence segments of their genome if it is structurally similar to our own, i.e. RNA or DNA," says Griffin.

There is one other location in the Solar System where viruses could remain the last holdout of organic entities, but it is only to be found in the far future, two billion years hence when our Sun has brightened. At this time Earth would have warmed, the plants withered and died, the oceans boiled away and life all but eradicated. Even here, in this smoldering world, viruses could remain. With little cellular material remaining the viruses could evolve to share genes altruistically, regardless of the host. As such, suggests Griffin, altruism in the microbial world may be the last state of life to be found on our planet, before the Sun grows so hot that even the viruses are snuffed out. Viruses and cells working in unison at the beginning and end of life stages on our planet – with billions of years of competitive conflict in between.

In the meantime, research into exo-viruses could be the seed for a new revolution in astrobiology. "The biological research community will continue to refine our understanding of viruses," says Impey. "Meanwhile, it is useful to start seriously considering their role in the wider landscape of exobiology. Griffin's excellent paper is a good starting point."

http://phys.org/news/2013-11-reveals-black-china-paper-authoring.html

Investigation reveals black market in China for research paper authoring The journal Science has uncovered, via investigation, a thriving black market in science paper authoring -

people are paying to have their names added to papers that have been written to describe research efforts. Phys.org - Mara Hvistendahl was the lead investigator and author of a paper published by Science, describing the operation and what was found.

There have been reports of unscrupulous journals printing research papers without proper vetting, and other reports suggesting that there exists a black market in paper authorship. This new investigation by Science, is the first to publish direct evidence of such a black market operating in China.

Hvistendahl reports on one instance where a suspected black-marketeer was contacted to inquire about having a name applied to an existing research paper. The contact quoted different prices for having a name included, depending on whether the person paying wished to be listed as the primary writer, or as merely a co-author, or even as just one of the team members. No money changed hands, as that would have been unethical for a Science reporter, but Hvistendahl reports that the paper that had been part of the earlier investigation showed up at a later date published in a reputable journal, along with different names attributed to the research effort - names of people that had all bought their way on.

Hvistendahl notes that such a black market has arisen in China due to the enormous pressure Chinese researchers are feeling to publish something. In that country, it appears having one's name attached to a research paper, matters more than actually conducting research. Hvistendahl also reports that people in China are willing to pay tens of thousands of dollars for the "honor" of having their name printed as an author on a research paper.

Hvistendahl writes that Science's undercover investigation revealed a thriving black market in China for paper authorization, which includes "shady agencies, corrupt scientists, and compromised editors." The undercover operation was conducted over a five month period and resulted in numerous examples of people at all levels of research in China participating in the black market in one way or another. They also found that it was possible to pay for someone to write a paper, attach a name and then submit and have it published in a reputable international journal - so long as the research it described passed a traditional vetting process.

The investigative team also found doctors and others engaged in medical research that were willing to openly admit that the black market for research papers is thriving in China. All in all, the investigative team contacted 27 agencies involved in helping researchers get their work published - only five of them refused an offer to pay for adding a name to a research paper.

China's Publication Bazaar, Science 29 November 2013: Vol. 342 no. 6162 pp. 1035-1039. DOI: 10.1126/science.342.6162.1035

http://www.bbc.co.uk/news/25141597

Why China is fixated on the Moon

The Moon could be a "beautiful" source of minerals and energy, a top Chinese scientist has told the BBC. Exotic materials including helium-3 and the potential for solar power could prove invaluable for humankind, he says. The comments come from Prof Ouyang Ziyuan of the department of lunar and deep space exploration. His first interview with the foreign media provides insights into China's usually secretive space programme.

Prof Ouyang was speaking ahead of the first Chinese attempt to land an unmanned spacecraft on the lunar surface. The Chang'e 3 lander is due to launch imminently, perhaps as soon as Sunday evening, UK time. It will be the first to make a soft touchdown on the Moon since an unmanned Russian mission in 1976.

No humans have set foot on the lunar surface since America's Apollo missions ended in 1972.

Prof Ouyang is an adviser to the mission and his comments reveal the scale of Chinese thinking about the Moon. He said the forthcoming venture would land in an ancient crater 400km wide called Sinus Iridum, thought to be relatively flat and clear of rocks, and explore its geology.

He explained that there were three motivations behind the drive to investigate the Moon. "First, to develop our technology because lunar exploration requires many types of technology, including communications, computers, all kinds of IT skills and the use of different kinds of materials. This is the key reason," he told BBC News. "Second, in terms of the science, besides Earth we also need to know our brothers and sisters like the Moon, its origin and evolution and then from that we can know about our Earth. "Third, in terms of the talents, China needs its own intellectual team who can explore the whole lunar and solar system - that is also our main purpose."

After the first two Chang'e craft orbited the Moon, the next two missions will try to land on it and the following two will attempt to bring samples back to Earth.

Manned expeditions will then take place, according to Prof Ouyang. "After all of this work, which is that China can make the achievement of arriving at the Moon and safely landing and that we can bring samples back; and once we finish all these unmanned projects, we will send Man there."

A rationale for this long-term programme is that "there are many ways humans can use the Moon", and he outlined a startling vision for its exploitation. With no air on the Moon, solar panels would operate far more efficiently, he believes, and a "belt" of them could "support the whole world".

The Moon is also "so rich" in helium-3, which is a possible fuel for nuclear fusion, that this could "solve human beings' energy demand for around 10,000 years at least".

Prof Ouyang highlighted the combination of an extremely thin atmosphere and massive temperature extremes offering a unique possibility for manufacturing that does not exist on Earth. He also spelled out the potential riches in lunar minerals and metals - a feature highlighted in an exhibition about the Moon which I visited in his home city of Guiyang. "The Moon is full of resources - mainly rare earth elements, titanium, and uranium, which the Earth is really short of, and these resources can be used without limitation. "But it's unnecessary to get them now because it's very costly."

Prof Ouyang summed up his vision for the goal of lunar exploration: "There are so many potential developments - it's beautiful - so we hope we can fully utilize the Moon to support sustainable development for humans and society." Coming from a representative of a poorer, less ambitious nation, these ideas might be seen as purely wishful thinking. But China has been methodically and patiently building up the key elements needed for an advanced space programme - from launchers to manned missions in Earth orbit to unmanned planetary craft - and it is investing heavily.

This comes as China is seen by neighbouring countries in Asia as flexing its muscles, most recently over control of airspace over the South China Sea. Chinese officials stress their desire to cooperate on space projects but lunar exploration is also regarded as a statement of national prowess.

Ouyang has himself been blunt about this in the past, as here in 2006: "Lunar exploration is a reflection of a country's comprehensive national power," he said in an interview with the official newspaper People's Daily. "It is significant for raising our international prestige and increasing our people's cohesion."

One leading British space scientist, Prof Richard Holdaway of the government-funded laboratory RAL Space, has long experience of working with China. He believes China could have astronauts on the lunar surface by 2025."They started from a long way back but now they're catching up fast - they want to monitor what's happening on the ground, they want to be part of the analysis of climate change and a much bigger programme looking at the Moon for mining or as a staging post to other parts of the Solar System."

I asked him if the idea of a Chinese moonbase extracting minerals was remotely plausible.

"It's perfectly plausible from the technical point of view, absolutely plausible from the finance point of view because they have great buying power, so I think, yes, there's nothing at all to stop them doing that probably within something like 10 years.

So a great deal is riding on the Chang'e 3 launch - national prestige, the quest for technological prowess and the desire to harness all available natural resources.

If all goes according to plan, the spacecraft will take six days to reach the Moon and then face the challenge of a soft landing. But it is clear that a successful mission will pave the way for the next boots to walk on the lunar surface to be worn by Chinese astronauts.

Name ______Student number ______ http://www.sciencedaily.com/releases/2013/11/131129101905.htm

Controversy Over Use of Roman Ingots to Investigate Dark Matter, Neutrinos The properties of these lead bricks recovered from ancient shipwrecks are ideal for experiments in particle

physics.

Scientists from the CDMS dark matter detection project in Minnesota (USA) and from the CUORE neutrino observatory at the Gran Sasso Laboratory in Italy have begun to use them, but archaeologists have raised alarm about the destruction and trading of cultural heritage that lies behind this.

Two thousand years ago, a Roman vessel with ingots of lead extracted from the Sierra of Cartagena sank across the waters from the coast of Sardinia. Since 2011, more than a hundred of these ingots have been used to build the 'Cryogenic Underground Observatory for Rare Events' (CUORE), an advanced detector of neutrinos -- almost weightless subatomic particles -- at the Gran Sasso National Laboratory in Italy.

In the 18th century, another ship loaded with lead ingots was wrecked on the French coast. A company of treasure hunters retrieved this material and, despite problems with French authorities, managed to sell it to the Cryogenic Dark Matter Search (CDMS) team. This detector located in a mine in Minnesota (USA) looks for signs of the enigmatic dark matter, which is believed to constitute a quarter of the universe.

These two examples have served as reference for the discussion that two researchers have opened between archaeologists, worried by the destruction of underwater cultural heritage, and particle physicists, pleased to have found a unique material for research on neutrinos and dark matter.

As Elena Perez-Alvaro from the University of Birmingham explains: "Roman lead is essential for conducting these experiments because it offers purity and such low levels of radioactivity -- all the more so the longer it has spent underwater -- which current methods for producing this metal cannot reach."

"Lead extracted today is naturally contaminated with the isotope Pb-210, which prevents it from being used as shielding for particle detectors," adds physicist Fernando González Zalba from the University of Cambridge. The two researchers have published a study in the journal 'Rosetta', also commented upon this month in 'Science', which poses a dilemma: Should we sacrifice part of our cultural heritage in order to achieve greater knowledge of the universe and the origin of humankind? Should we yield part of our past to discover more about our future?

"Underwater archaeologists see destruction of heritage as a loss of our past, our history, whilst physicists support basic research to look for answers we do not yet have," remarks Perez-Alvaro, "although this has led to situations in which, for example, private companies like Odyssey trade lead recovered from sunken ships." This is the company that had to return the treasure of the frigate Nuestra Señora de las Mercedes to Spain.

Dialogue between underwater archaeologists and particle physicists

The underwater archaeologist and the physicist are encouraging dialogue between both collectives, as well as developing legislation that regulates these kinds of activities, without limiting them exclusively to archaeologists, and including scientists. "Recovery for knowledge in both fields, and not merely for commercial reasons," the scientists stress.

The jury is still out. In the case of the CUORE detector, for example, in principle the lead from the least wellpreserved Roman ingots is used, although their inscriptions are cut and preserved. Some archaeologists also suggests that there are other pieces of valuable metal, such as anchor stocks, rings or tackles for fishing that we should assess whether or not to "sacrifice for science."

The problem is that they are protected by UNESCO's 2001 Convention on the protection of underwater cultural heritage if they have been under water more than 10 years and the 2003 Convention for safeguarding intangible cultural heritage.

Regarding the habitual use that Romans made of these ingots, Pérez Álvaro points out that there are many theories, "but they were generally used as water-resistant material for pipes, water tanks or roofs, but also in the manufacture of arms and ammunition."

A special case are the large lead bricks recovered from the largest Roman ship of the excavation of the Mediterranean, the wreck of the Bou Ferrer, which sunk very close to the port of La Vila Joiosa (Alicante). A series of engravings enable specialists to determine that their owner was the Emperor of Rome himself, probably Caligula, Claudius or Nero.

26 12/2/13

Student number Name http://scitechdaily.com/unexpected-discovery-galaxy-messier-101/

An Unexpected Discovery in Galaxy Messier 101

Using data from the Gemini Observatory, researchers discovered that a small black hole can sustain a hugely voracious appetite while consuming material in an efficient and tidy manner – something previously thought impossible.

November 29, 2013 by Staff

Observations of a black hole powering an energetic X-ray source in a galaxy some 22 million light-years away could change our thinking about how some black holes consume matter. The findings indicate that this particular black hole, thought to be the engine behind the X-ray source's high-energy light output, is unexpectedly lightweight, and, despite the generous amount of dust and gas being fed to it by a massive stellar companion, it swallows this material in a surprisingly orderly fashion.

"It has elegant manners," says research team member Stephen Justham, of the National Astronomical Observatories of China, Chinese Academy of Sciences. Such lightweights, he explains, must devour matter at close to their theoretical limits of consumption to sustain the kind of energy output observed. "We thought that when small black holes were pushed to these limits, they would not be able to maintain such refined ways of consuming matter," Justham explains. "We expected them to display more complicated behavior when eating so quickly. Apparently we were wrong."

A Surprising Twist

X-ray sources give off high- and low-energy X-rays, which astronomers call hard and soft X-rays, respectively. In what might seem like a contradiction, larger black holes tend to produce more soft X-rays, while smaller black holes tend to produce relatively more hard X-rays. This source, called M101 ULX-1, is dominated by soft X-rays, so researchers expected to find a larger black hole as its energy source.

In a surprising twist, however, the new observations made at the Gemini Observatory, and published in the November 28th issue of the journal Nature, indicate that M101 ULX-1's black hole is on the small side, and astrophysicists don't understand why.

In theoretical models of how matter falls into black holes and radiates energy, the soft X-rays come primarily from the accretion disk (see illustration), while hard X-rays are typically generated by a high-energy "corona" around the disk. The models show that the corona's emission strength should increase as the rate of accretion gets closer to the theoretical limit of consumption. Interactions between the disk and corona are also expected to become more complex.

Based on the size of the black hole found in this work, the region around M101-ULX-1 should, theoretically, be dominated by hard X-rays and appear structurally more complicated. However, that isn't the case.

"Theories have been suggested which allow such low-mass black holes to eat this quickly and shine this brightly in X-rays. But those mechanisms leave signatures in the emitted X-ray spectrum, which this system does not display," says lead author Jifeng Liu, of the National Astronomical Observatories of China, Chinese Academy of Sciences. "Somehow this black hole, with a mass only 20-30 times the mass of our Sun, is able to eat at a rate near to its theoretical maximum while remaining relatively placid. It's amazing. Theory now needs to somehow explain what's going on."

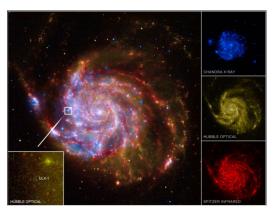


Figure 2. ULX-1 is located near a spiral arm of M101. The image for M101 is composed from X-ray (Chandra X-ray Observatory; Purple), Infrared (Spitzer Satellite; Red), Optical (Hubble Space Telescope; Yellow) and Ultraviolet (GALEX satellite; Blue). Credit: Chandra X-ray Observatory, Spitzer Satellite, Hubble Space Telescope, and GALEX Satellite.

An Intermediate-mass Black Hole Dilemma

The discovery also delivers a blow to astronomers hoping to find conclusive evidence for an "intermediatemass" black hole in M101 ULX-1. Such black holes would have masses roughly between 100 and 1000 times the mass of the Sun, placing them between normal stellar-mass black holes and the monstrous supermassive black holes that reside in the centers of galaxies. So far these objects have been frustratingly elusive, with potential candidates but no broadly-accepted detection. Ultra-luminous X-ray sources (ULXs) have been one of the main proposed hiding places for intermediate-mass black holes, and M101 ULX-1 was one of the most promising-looking contenders.

"Astronomers hoping to study these objects will now have to focus on other locations for which indirect evidence of this class of black holes has been suggested, either in the even brighter 'hyper-luminous' X-ray sources or inside some dense clusters of stars," explains research team member Joel Bregman of the University of Michigan.

"Many scientists thought it was just a matter of time until we had evidence for an intermediate-mass black hole in M101 ULX-1," says Liu. But the new Gemini findings both take away some of that hope to solve an old puzzle and adds the fresh mystery of how this stellar-mass black hole can consume matter so calmly. To determine the mass of the black hole, the researchers used the Gemini Multi-Object Spectrograph at the Gemini North telescope on Mauna Kea, Hawai'i to measure the motion of the companion. This star, which feeds matter to the black hole, is of the Wolf-Rayet variety. Such stars emit strong stellar winds, from which the black hole can then draw in material. This study also revealed that the black hole in M101 ULX-1 can capture more material from that stellar wind than astronomers had anticipated.

M101 ULX-1 is ultra-luminous, shining a million times more brightly than the Sun in both X-rays (from the black hole accretion disk) and in the ultraviolet (from the companion star). Co-author Paul Crowther from the University of Sheffield in the United Kingdom adds, "Although this isn't the first Wolf-Rayet black hole binary ever discovered, at some 22 million light-years away, it does set a new distance record for such a system. The Wolf-Rayet star will have died in a small fraction of the time it has taken for light to reach us, so this system is now likely a double black hole binary."

"Studying objects like M101 ULX-1 in distant galaxies gives us a vastly larger sampling of the diversity of objects in our universe," says Bregman. "It's absolutely amazing that we have the technology to observe a star orbiting a black hole in another galaxy this far away."

Publication: Ji-Feng Liu, et al., "Puzzling accretion onto a black hole in the ultraluminous X-ray source M 101 ULX-1," Nature 503, 500–503, (28 November 2013); doi:10.1038/nature12762

http://nyti.ms/180d0U2

Comet ISON, Presumed Dead, Shows New Life

Comet ISON passed within a million miles of the sun's surface at 1:37 p.m. Eastern time on Thursday - by which time observers had already glumly concluded that the comet had disintegrated and vaporized. By KENNETH CHANG

NASA posted on Twitter, "It's likely it didn't survive." ISON, which spent several billion years at the frigid edge of the solar system before starting a long journey toward the sun, had been billed as a possible "comet of the century." Its demise seemed to be an anticlimactic ending to the story.

But "then it appears again," said Karl Battams, an astrophysicist at the Naval Research Laboratory who has been observing the comet from Kitt Peak National Observatory in Arizona. "We see something come out." Images taken by spacecraft showed an increasingly bright point at the head of the comet. Dr. Battams said that current data could not offer a definitive answer, but it appeared Friday that part of ISON's nucleus was still holding together.

"It's definitely maybe alive," Dr. Battams said. "There's a strong definite chance it might be, may be alive."

Additional observations by spacecraft and ground-based telescopes could provide a clearer picture over the next few days. The Hubble Space Telescope should be able to take a close look in a couple of weeks.

On his Twitter account, Dr. Battams mused, "So, umm ... did I mention that comets are like cats??"

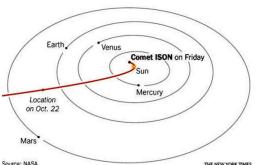
Comet ISON may have survived its brush with the sun.

Even more uncertain is whether there will be much to see in the night sky in early December, when ISON is to pass through Earth's neighborhood. (One thing is certain, astronomers say: There is no possibility that it will strike Earth.)

The apparent resurrection raised the question: if ISON is not dead, why did it disappear during its close approach to the sun?

"At this point, we don't have an answer to that," said C. Alex Young, associate director for science in the heliophysics division at NASA's Goddard Spaceflight Center in Greenbelt, Md.

The tale has gathered a wide following on the Internet, with Dr. Battams juggling media interviews and Twitter postings while also trying to digest the stream of data. "We've got spotlights on us, literally," he said in an interview, adding that he had slept only a couple of hours. "It's a lot of pressure because at present we have a lot more questions than answers. But it's fabulous. It's an amazing event we're witnessing."



On Thursday, Dr. Battams and Dr. Young answered questions in a NASA-organized chat room on Google as ISON neared the sun.

NASA's Solar Dynamics Observatory spacecraft sent back an image that was expected to show the comet within the corona. It showed nothing, but it was possible the comet was not close enough yet. "We thought maybe we wouldn't see something right away," Dr. Young said.

Half an hour later, another image came back, again with no sign of ISON.

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"We didn't see anything - nothing - and we expected we would see at least a little bit," Dr. Young said. A much smaller comet last year had given an impressive show, and scientists expected that even if ISON started falling apart, there would still be big pieces left for the observatory to detect.

"We were extremely let down by the lack of a show," Dr. Young said.

But a couple of hours later, another NASA spacecraft spotted something emerging from the other side of the sun. At first it seemed to be nothing more than debris from the comet's tail. Dr. Young left for home thinking the day had been a bust.

As he was driving, he heard his cellphone buzzing as text messages poured in. He pulled over to take a look at the data. More images were showing indications of a surviving nucleus. He headed to a diner that was closed for Thanksgiving but whose Wi-Fi network was on. "I pulled out my laptop to see what I could see," he said. The news that reports of ISON's death were premature ricocheted around Twitter.

Richard Branson, the British billionaire who founded the constellation of Virgin companies, posted on Friday: "Our sun melts most of comet #ISON. A little survives to fly on."

Scientists hope that observations of ISON will also provide information about the early solar system when ISON formed. By now, comet experts are cautious about saying what they expect to happen next.

ISON, Dr. Battams said, "is taking every opportunity to do everything we didn't expect it to do."

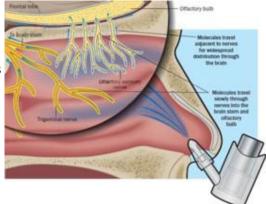
http://www.scientificamerican.com/article.cfm?id=inhaled-stem-cells-might-replace-lost-neurons

Inhaled Stem Cells Might Replace Lost Neurons Intranasal stem cell therapy may one day treat brain disorders

By Caitlin Shure

Many diseases of the central nervous system involve the death of neurons - so, theoretically, the replacement of dead cells should improve symptoms of degenerative disorders such as Parkinson's, Huntington's, amyotrophic lateral sclerosis (ALS) and Alzheimer's, as well as stroke and brain tumors. Stem cell therapy may do just that even though evidence of its effectiveness is mixed.

In any cell transplant procedure, the host organ - in this case, the brain - may reject its new additions. Further, it is unclear whether grafted cells can truly integrate into complex neural circuitry. Finally, current procedures require invasive surgical implantation, which can be expensive and risky. The surgery can cause neural inflammation, and the implanted cells may quickly die.



Inhaler Jim Kopp

Intranasal administration may address at least some of these issues. Most important, it eliminates the need for surgery. Further, some research suggests that stem cells delivered intranasally are "smart" - they do not spread through the brain indiscriminately but instead target damaged cells.

Although it is difficult to predict when medical practice will adopt stem cell therapy for the brain, animal studies have produced some promising results. In a rat model of Parkinson's, for example, treatment with intranasal stem cells appeared to improve motor function and slow the neurological deterioration associated with the disease.

http://www.wired.com/wiredscience/2013/11/surf-for-cystic-fybrosis/

The Surfing Solution: How Seawater Can Help Treat Cystic Fybrosis Cystic fibrosis, it turns out, doesn't like salt water. By Ariel Ramchandani

Inhaling it rehydrates the airways, allowing mucus to flow more easily and be dislodged by coughing. Patients have used saline nebulizers to achieve this effect for years, and new units are under development that people can use during sleep. But CF sufferers who would rather catch waves than z's might get some of the same benefits (albeit at a much lower concentration) by hopping on a surfboard.

The effect of salt water on cystic fibrosis was actually discovered more than a decade ago, when Australian researchers noticed that patients in Sydney reported feeling better after spending time in the ocean.

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Experimentation later revealed that it was, indeed, the salt water; that's why there are saline inhalers today. Now a group called Mauli Ola ("breath of life" in Hawaiian) is reviving the therapy—it has helped around 2,000 children and young adults with cystic fibrosis learn to surf with professional riders. "Surfing is my way of overcoming cystic fibrosis," says Jacob Venditti, a 20-year-old patient who hopes to catch a 30-footer one day. "It's my physical therapy every day that there are waves." Gnarly, brah.

http://www.eurekalert.org/pub_releases/2013-12/uoc--vf112613.php

'Nanosponge vaccine' fights MRSA toxins

Nanosponges that soak up a dangerous pore-forming toxin produced by MRSA (methicillin-resistant Staphylococcus aureus) could serve as a safe and effective vaccine against this toxin.

This "nanosponge vaccine" enabled the immune systems of mice to block the adverse effects of the alpha-haemolysin toxin from MRSA—both within the bloodstream and on the skin. Nanoengineers from the University of California, San Diego described the safety and efficacy of this nanosponge vaccine in the December 1 issue of Nature Nanotechnology.

The nanosponges at the foundation of the experimental "toxoid vaccine" platform are bio-compatible particles made of a polymer core wrapped in a red-blood-cell membrane. Each nanosponge's red-blood-cell membrane seizes and detains the Staphylococcus aureus (staph) toxin alpha-haemolysin without compromising the toxin's structural integrity through heating or chemical processing. These toxin-studded nanosponges served as vaccines capable of triggering neutralizing antibodies and fighting off otherwise lethal doses of the toxin in mice.



The nanosponges at the foundation of the experimental "toxoid vaccine" platform are bio-compatible particles made of a polymer core (light-blue-green color) wrapped in a red-blood-cell membrane (orange). Each nanosponge's redblood-cell membrane seizes and detains the Staphylococcus aureus (staph) toxin alpha-haemolysin (blue) without compromising the toxin's structural integrity through heating or chemical processing. These toxin-studded nanosponges served as vaccines capable of triggering neutralizing antibodies and fighting off otherwise lethal doses of the toxin in mice.

Nanosponges that soak up a dangerous pore-forming toxin produced by MRSA (methicillin-resistant Staphylococcus aureus) could serve as a safe and effective vaccine against this toxin. This "nanosponge vaccine" enabled the immune systems of mice to block the adverse effects of the alpha-haemolysin toxin from MRSA -- both within the bloodstream and on the skin. Nanoengineers from UC San Diego described the safety and efficacy of this nanosponge vaccine in the Dec. 1 issue of Nature Nanotechnology. UC San Diego Department of NanoEngineering

Toxoid vaccines protect against a toxin or set of toxins, rather than the organism that produces the toxin(s). As the problem of antibiotic resistance worsens, toxoid vaccines offer a promising approach to fight infections without reliance on antibiotics.

"With our toxoid vaccine, we don't have to worry about antibiotic resistance. We directly target the alphahaemolysin toxin," said Liangfang Zhang, a nanoengineering professor at UC San Diego Jacobs School of Engineering and the senior author on the paper.

Targeting the alpha-haemolysin toxin directly has another perk. "These toxins create a toxic environment that serves as a defense mechanism which makes it harder for the immune system to fight Staph bacteria," explained Zhang.

Beyond MRSA and other staph infections, the nanosponge vaccine approach could be used to create vaccines that protect against a wide range of toxins, including those produced by E. coli and H. pylori.

This work from Zhang's Nanomaterials and Nanomedicine Laboratory at the UC San Diego included nanoengineering post-doctoral researcher Che-Ming "Jack" Hu, nanoengineering graduate student Ronnie Fang, and bioengineering graduate student Brian Luk.

The researchers found that their nanosponge vaccine was safe and more effective than toxoid vaccines made from heat-treated staph toxin.

After one injection, just 10 percent of staph-infected mice treated with the heated version survived, compared to 50 percent for those who received the nanosponge vaccine. With two more booster shots, survival rates with the nanosponge vaccine were up to 100 percent, compared to 90 percent with the heat-treated toxin.

"The nanosponge vaccine was also able to completely prevent the toxin's damages in the skin, where MRSA infections frequently take place," said Zhang, who is also affiliated with the Moores Cancer Center at UC San Diego.

Fighting Pore-Forming Toxins

This work is a twist on a project the UC San Diego nanoengineers presented earlier this year: a nanosponge that can sop up a variety of pore-forming toxins—from bacterial proteins to snake venom—in the body.

Pore-forming toxins work by punching holes in a cell's membrane and letting the cell essentially leak to death. But when toxins attack the red blood cell membrane draped over the nanoparticle, "nothing will happen. It just locks the toxin there," Zhang explained.

The nanoengineers wondered what would happen if they loaded one of their nanosponges with staph toxin in this way, and presented the whole package to an essential part of the immune system called dendritic cells. Could the loaded particles trigger an immune response and work as a toxoid vaccine?

Staph toxin is so powerful that it kills immune cells in its unaltered form. Most vaccine candidates, therefore, use a heat or chemically processed version of the toxin that unravels some of its proteins and makes it a little weaker. But this process also makes the immune response to the toxin a little weaker.

"The more you heat it, the safer the toxin is, but the more you heat it, the more you damage the structure of the protein," Zhang explained. "And this structure is what the immune cell recognizes, and builds its antibodies against."

The nanosponge toxoid vaccine gets around this problem by detaining—but not changing—the staph toxin. Like a dangerous but handcuffed prisoner, the staph toxin can be led to the dendritic cells of the immune system without causing any harm.

Before this, "there was no way you could deliver a native toxin to the immune cells without damaging the cells," Zhang said. "But this technology allows us to do this."

Each vaccine particle is approximately 85 nanometers in diameter; for comparison, about 1000 of them would fit across the width of a single human hair. They are cleared from the body after injection in about two weeks, the researchers found.

Staphylococcus aureus

Staph bacteria are one of the most common causes of skin infections, and can cause blood poisoning and surgical infections as well as pneumonia. According to the Centers for Disease Control and Prevention, about 80,000 Americans suffer from invasive MRSA infections each year, and over 11,000 of those individuals die. At the moment, there are no vaccines approved to protect humans against the toxins associated with staph infections, including those caused by MRSA strains.

The idea for a staph vaccine came about when the researchers considered the success of their nanosponge. If the particle was so good at collecting toxins, they wondered, what were the potential uses of a particle full of toxin? "To be honest, we never thought about the vaccine use from the beginning," Zhang noted. "But when we do research, we always want to look at a problem in reverse."

In a way, the toxoid vaccine hearkens back to their first use for the particles, as a cancer drug delivery device, Zhang noted.

The particles "work so beautifully," Zhang said, that it might be possible to detain several toxins at once on them, creating "one vaccine against many types of pore-forming toxins," from staph to snake venom.

The research was funded by the National Institute of Diabetes and Digestive and Kidney Diseases of the National Institutes of Health (award no. R01DK095168) and by the National Science Foundation (grant DMR-1216461).

"Nanoparticle-detained toxins for safe and effective vaccination," by Che-Ming J. Hu, Ronnie H. Fang and Liangfang Zhang in the Department of NanoEngineering at the University of California, San Diego; and Brian T. Luk in the Department of Bioengineering at the University of California, San Diego.

http://www.eurekalert.org/pub_releases/2013-12/cumc-hsc112713.php

Human stem cells converted to functional lung cells

Possibility of generating lung tissue for transplant using a patient's own cells

NEW YORK, NY - For the first time, scientists have succeeded in transforming human stem cells into functional lung and airway cells. The advance, reported by Columbia University Medical Center (CUMC) researchers, has significant potential for modeling lung disease, screening drugs, studying human lung development, and, ultimately, generating lung tissue for transplantation. The study was published today in the journal Nature Biotechnology.

"Researchers have had relative success in turning human stem cells into heart cells, pancreatic beta cells, intestinal cells, liver cells, and nerve cells, raising all sorts of possibilities for regenerative medicine," said study leader Hans-Willem Snoeck, MD, PhD, professor of medicine (in microbiology & immunology) and affiliated with the Columbia Center for Translational Immunology and the Columbia Stem Cell Initiative. "Now, we are finally able to make lung and airway cells. This is important because lung transplants have a particularly poor prognosis. Although any clinical application is still many years away, we can begin thinking about making

31 12/2/13

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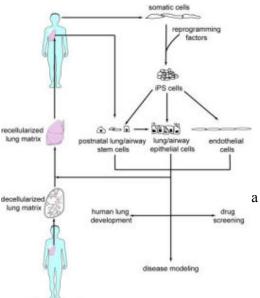
autologous lung transplants—that is, transplants that use a patient's own skin cells to generate functional lung tissue."

The research builds on Dr. Snoeck's 2011 discovery of a set of chemical factors that can turn human embryonic stem (ES) cells or human induced pluripotent stem (iPS) cells into anterior foregut endoderm—precursors of lung and airway cells. (Human iPS cells closely resemble human ES cells but are generated from skin cells, by coaxing them into taking a developmental step backwards. Human iPS cells can then be stimulated to differentiate into specialized cells—offering researchers an alternative to human ES cells.)

In the current study, Dr. Snoeck and his colleagues found new factors that can complete the transformation of human ES or iPS cells into functional lung epithelial cells (cells that cover the lung surface). The resultant cells were found to express markers of at least six types of lung and airway epithelial cells, particularly markers of type 2 alveolar epithelial cells. Type 2 cells are important because they produce surfactant, a substance critical

to maintain the lung alveoli, where gas exchange takes place; they also participate in repair of the lung after injury and damage. The findings have implications for the study of a number of lung diseases, including idiopathic pulmonary fibrosis (IPF), in which type 2 alveolar epithelial cells are thought to play a central role. "No one knows what causes the disease, and there's no way to treat it," says Dr. Snoeck. "Using this technology, researchers will finally be able to create laboratory models of IPF, study the disease at the molecular level, and screen drugs for possible treatments or cures."

"In the longer term, we hope to use this technology to make an autologous lung graft," Dr. Snoeck said. "This would entail taking decendentized lung from a donor; removing all the lung cells, leaving only the lung scaffold; and seeding the scaffold with new lung cells derived from the patient. In this way, rejection problems could be avoided." Dr. Snoeck is investigating this approach in collaboration with researchers in the Columbia University Department of Biomedical Engineering.



neic donor

Human skin cells can now be reprogrammed into induced pluripotent stem cells, which are similar to human embryonic stem cells, and then into functional lung and airway cells. Such cells have potential for use in disease modeling, drug screening, the study of lung development, and the generation of lung tissue for transplantation. Hans-Willem Snoeck, M.D., Ph.D., Columbia University Medical Center

"I am excited about this collaboration with Hans Snoeck, integrating stem cell science with bioengineering in the search for new treatments for lung disease," said Gordana Vunjak-Novakovic, co-author of the paper and Mikati Foundation Professor of Biomedical Engineering at Columbia's Engineering School and professor of medical sciences at Columbia University College of Physicians and Surgeons.

The paper is titled, "Highly efficient generation of airway and lung epithelial cells from human pluripotent stem cells." The other contributors are Sarah X.L. Huang, Mohammad Naimul Islam, John O'Neill, Zheng Hu, Yong-Guang Yang, Ya-Wen Chen, Melanie Mumau, Michael D. Green, and Jahar Bhattacharya (all at CUMC).

Columbia University has filed for a patent relating to the generation of lung and airway epithelium from human pluripotent stem cells and uses thereof. The authors declare no other financial or other conflicts of interests.

The study was supported by startup funds from CUMC and the New York Stem Cell Foundation.

http://www.newscientist.com/article/dn24678-astrophile-europas-choppy-ocean-looks-friendly-to-life.html

Astrophile: Europa's choppy ocean looks friendly to life

Object: Europa's subsurface ocean

Interesting property: Intense turbulence

18:00 01 December 2013 by Nicola Guttridge

As moons go, Europa is doing pretty well in the looks department. While other wrinkled and pockmarked planetary bodies look their age, Jupiter's moon, despite being billions of years old, is one of the smoothest objects in our solar system. However, this moon is far from flawless. Europa is suspected to have a perpetually dark, liquid water ocean enclosed beneath a thick shell of water ice – around 40 per cent of which is covered with long, dark scratches and scars.

The prospect of liquid water places Europa near the top of the list of places in our solar system that might host alien life. However, it is hard to know what's actually going on in the sub-surface ocean. Does it teem with alien microbes – perhaps even bigger creatures – or is it a vast, inky, sterile wasteland?

32 12/2/13

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The only window we have on the ocean is its icy surface, so scientists try to read its criss-cross scars for clues. But whether this so-called chaos terrain can tell us about what's going on underneath such a thick layer of ice is hotly disputed.

Oceanic chaos

The criss-cross pattern is likely to be caused by warmer and thinner regions of ice breaking and refreezing. It is much more abundant around the moon's equatorial regions than at its poles – but why should this be the case? Unlike on Earth, the temperature difference between the equator and poles cannot be explained by the effects of the sun because its light is too faint and Europa's surface too reflective.

Another theory involves Jupiter's gravitational pull, which would produce tectonic forces and heat up Europa's ocean – but models have shown that this would heat the poles than the equator.

The latest study suggests that turbulence in Europa's ocean sculpts the chaos terrain on the icy surface. It was previously assumed that an effect caused by the moon's rotation – known as the Coriolis force Movie Camera – dominates the ocean's flow, funnelling heat to high latitudes. The new model instead relies on ocean currents caused by convection of the moon's internal heat.

Mix for life

The team found that this produces a chaos terrain very similar to the one seen on Europa. "The resulting flow is less organised, but more vigorous in the equatorial region," says researcher Johannes Wicht of the Max Planck Institute for Solar System Research in Lindau, Germany. "This correlates nicely with the distribution of chaos terrain."

The model suggests that the ocean is extremely turbulent, with three strong ocean jets. So despite the thickness of Europa's icy shell, it seems that properties of its ocean are writ in the ice. "We may be able to understand Europa's ocean just by looking at the surface," says Wicht's colleague Britney Schmidt of the Georgia Institute of Technology in Atlanta.

A turbulent ocean would be beneficial for any life there because it would help shift nutrients from the sea floor into the rest of the ocean, says Schmidt's other colleague, Krista Soderlund of the University of Texas at Austin. Microbes can live in stagnant water, but knowing the ocean is turbulent makes life much more likely.

Upcoming missions such as the European Space Agency's Jupiter Icy Moons Explorer (JUICE), will map Europa's chaos terrain via fly-bys in 2030, and potentially NASA's Europa Clipper, will find out more – although it is just a concept at the moment.

Studies of Europa's chaos terrain may have relevance beyond Jupiter's moon. "Icy subsurface oceans may be commonplace in the outer solar system," says Leigh Fletcher of the University of Oxford, a member of the ESA Science Working Team for JUICE. "JUICE will also search for any active plumes and vents, just like on Saturn's moon Enceladus, to offer a glimpse into this icy ocean. This will be a great test of this sort of model for the icy worlds of our solar system."

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http://nyti.ms/1hsr2uc

Comet, Thought Dead but Found Alive, Is Now Fading Away, Astronomers Say A comet that once promised to light up the night skies — then all but vanished, and later seemed to blaze back to life — is now fading away, astronomers say. By KENNETH CHANG

"I think for the most part it's dead," said C. Alex Young, the associate director for science in the heliophysics division at NASA's Goddard Space Flight Center in Greenbelt, Md. "The folks are finally pretty confident that's the case."

En route to its demise, the comet, ISON, has provided one twist after another. On Thursday, when it made its closest approach to the sun, the comet eluded observation, and scientists concluded that it had disintegrated and vaporized. Then, a few hours later, it reappeared. A bright spot at the head of the comet suggested that part of its icy nucleus had survived.

But the resurrection was short-lived. ISON faded again over the weekend. By Sunday, it was already so dim that its debris will no longer be visible to the naked eye when it passes through Earth's region this month. "I really don't think there's a whole lot left," said Karl Battams, an astrophysicist at the Naval Research Laboratory who has spent a week observing ISON at the Kitt Peak National Observatory in Arizona. "I'm very disappointed for the public, because we're not going to see this beautiful object in the Northern Hemisphere skies." Dr. Battams said the comet had probably fallen largely apart before its closest approach to the sun. He said it was difficult to tell what was seen moving away from the sun. The comet may have possessed a small nucleus that has since vaporized. Or the ice that holds it together had already vaporized, leaving just a loose pile of rubble.

"It's also possible it was just a cloud of dust at that point," Dr. Battams said. The comet, discovered in September 2012 while still beyond Jupiter's orbit, originated from the Oort cloud, a region of icy debris over a light-year from the sun.

For scientists, the excitement of observation is over, but the work of analyzing data to understand what happened will take weeks and months. The complete disintegration of ISON could give better information about its composition — and hints of how the planets formed — than if it had remained intact. "Scientifically, I don't know if it gets much better than seeing the comet being ripped apart, falling apart right

"Scientifically, I don't know if it gets much better than seeing the comet being ripped apart, falling apart right before your eyes," Dr. Battams said.

http://www.bbc.co.uk/news/health-25156509

Caffeine energy drinks 'intensify heart contractions'

Energy drinks packed with caffeine can change the way the heart beats, researchers warn. The team from the University of Bonn in Germany imaged the hearts of 17 people an hour after they had an energy drink.

The study showed contractions were more forceful after the drink.

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The team told the annual meeting of the Radiological Society of North America that children and people with some health conditions should avoid the drinks.

Researcher Dr Jonas Dorner said: "Until now, we haven't known exactly what effect these energy drinks have on the function of the heart.

"The amount of caffeine is up to three times higher than in other caffeinated beverages like coffee or cola.

"There are many side effects known to be associated with a high intake of caffeine, including rapid heart rate, palpitations, rise in blood pressure and, in the most severe cases, seizures or sudden death."

The researchers gave the participants a drink containing 32mg per 100ml of caffeine and 400mg per 100ml of another chemical, taurine.

Short-term impact

They showed the chamber of the heart that pumps blood around the body, the left ventricle, was contracting harder an hour after the energy drink was taken than at the start of the study.

Dr Dorner added: "We've shown that energy drink consumption has a short-term impact on cardiac contractility. "We don't know exactly how or if this greater contractility of the heart impacts daily activities or athletic performance."

The impact on people with heart disease is also unknown.

However, the research team advises that children and people with an irregular heartbeat should avoid the drinks. The British Soft Drinks Association already says the drinks are not for children.