http://www.eurekalert.org/pub_releases/2011-06/uocp-idm062011.php

Informal daycare may harm kids' cognitive development, study finds

Formal daycare is better for a child's cognitive development than informal care by a grandparent, sibling, or family friend, according to a study of single mothers and their childcare choices published in the July issue of the Journal of Labor Economics.

According to the study, children who go to a formal preschool program or a licensed daycare center have essentially the same standardized test scores as those who stay home with mom. Conversely, each year of informal care reduces a child's test scores by 2.6 percent versus staying with mom.

"Extensive research has shown that a child's early achievement is a strong predictor of outcomes later in life," said Raquel Bernal of the Universidad de los Andes in Columbia, who performed the research with Michael Keane of the University of New South Wales in Sydney, Australia. "This research suggests that separation from the mother has a negative effect on a child's cognitive ability, but this can be offset by the appropriate choice of daycare."

The study took advantage of changes made in the 1990s to U.S. welfare laws that encouraged single mothers to enter the workforce. Before the changes, about 59 percent of single mothers worked outside the home. By 2001, that number increased to 72 percent. The researchers compared test scores for children born shortly before and after the law change to find out if increased employment had an effect on children's test scores, after controlling for outside factors such as socioeconomic status. The scores came from standardized tests the children took between the ages of 3 and 6.

The study found overall that use of childcare reduces a child's test scores significantly. But when the researchers divided the children in the sample into those who received formal and informal care, they found that the reduction in tests scores was driven solely by children in informal care. In other words, formal care was found to have no adverse effect on test scores. "The policy implication is that it would be desirable to provide financial support that would enable single mothers to spend more time with their children, or support to place children in formal care at early ages," Bernal said.

Raquel Bernal and Michael P. Keane, "Child Care Choices and Children's Cognitive Achievement: The Case of Single Mothers." Journal of Labor Economics 29:3 (July 2011).

http://www.eurekalert.org/pub_releases/2011-06/uow-acd061911.php

Atmospheric carbon dioxide buildup unlikely to spark abrupt climate change

There have been instances in Earth history when average temperatures have changed rapidly, as much as 10 degrees Celsius (18 degrees Fahrenheit) over a few decades, and some have speculated the same could happen again as the atmosphere becomes overloaded with carbon dioxide.

New research lends support to evidence from numerous recent studies that suggest abrupt climate change appears to be the result of alterations in ocean circulation uniquely associated with ice ages.

"There might be other mechanisms by which greenhouse gases may cause an abrupt climate change, but we know of no such mechanism from the geological record," said David Battisti, a University of Washington atmospheric sciences professor.

Battisti was part of a team that used a numerical climate model coupled with an oxygen-isotope model to determine what caused climate shifts in a computer-generated episode that mimicked Heinrich events during the last ice age, from 110,000 to 10,000 years ago. Heinrich events produced huge numbers of North Atlantic Ocean icebergs that had broken off from glaciers.

The simulations showed the sudden increase in North Atlantic sea ice cooled the Northern Hemisphere, including the surface of the Indian Ocean, which reduced rainfall over India and weakened the Indian monsoon.

Battisti noted that while carbon dioxide-induced climate change is unlikely to be abrupt, the impacts of changing climate could be.

"When you lose a keystone species, ecosystems can change very rapidly," he said. "Smoothly retreating sea ice will cause fast warming if you live within a thousand kilometers of the ice. If warming slowly dries already semi-arid places, fires are going to be more likely."

Previous studies of carbonate deposits from caves in China and India are believed to show the intensity of monsoon precipitation through the ratio of specific oxygen isotopes. The modeling the scientists' used in the current study reproduced those isotope ratios, and they determined that the Heinrich events were associated with changes in the intensity of monsoon rainfall in India rather than East Asia.

The research is published online June 19 by Nature Geoscience. The lead author is Franceso Pausata of the Bjerknes Centre for Climate Research in Norway. Besides Battisti, other co-authors are Kerim Nisancioglu of UNI Research in Norway and Cecilia Bitz of the UW.

The work was funded by the Norwegian Research Council and the U.S. National Science Foundation.

http://www.eurekalert.org/pub_releases/2011-06/nsf-fsr062011.php

Fastest sea-level rise in 2 millennia linked to increasing global temperatures

Rate is greater now than at any time during past 2,100 years

The rate of sea level rise along the U.S. Atlantic coast is greater now than at any time in the past 2,000 years - and has shown a consistent link between changes in global mean surface temperature and sea level.
The findings are published this week in the journal Proceedings of the National Academy of Sciences (PNAS).

The research, funded by the National Science Foundation (NSF), was conducted by Andrew Kemp, Yale University; Benjamin Horton, University of Pennsylvania; Jeffrey Donnelly, Woods Hole Oceanographic Institution; Michael Mann, Pennsylvania State University; Martin Vermeer, Aalto University School of Engineering, Finland; and Stefan Rahmstorf, Potsdam Institute for Climate Impact Research, Germany.

"Having a detailed picture of rates of sea level change over the past two millennia provides an important context for understanding current and potential future changes," says Paul Cutler, program director in NSF's Division of Earth Sciences.

"It's especially valuable for anticipating the evolution of coastal systems," he says, "in which more than half the world's population now lives."

Adds Kemp, "Scenarios of future rise are dependent on understanding the response of sea level to climate changes. Accurate estimates of past sea-level variability provide a context for such projections."

Kemp and colleagues developed the first continuous sea-level reconstruction for the past 2,000 years, and compared variations in global temperature to changes in sea level over that time period.

The team found that sea level was relatively stable from 200 BC to 1,000 AD.

Then in the 11th century, sea level rose by about half a millimeter each year for 400 years, linked with a warm climate period known as the Medieval Climate Anomaly. Then there was a second period of stable sea level during a cooler period called the Little Ice Age. It persisted until the late 19th century.

Since the late 19th century, sea level has risen by more than 2 millimeters per year on average, the steepest rate for more than 2,100 years. "Sea-level rise is a potentially disastrous outcome of climate change," says Horton, "as rising temperatures melt land-based ice, and warm ocean waters."

To reconstruct sea level, the scientists used microfossils called foraminifera preserved in sediment cores extracted from coastal salt marshes in North Carolina. The age of the cores was estimated using radiocarbon dating and other techniques.

To test the validity of their approach, the team compared its reconstructions with tide-gauge measurements from North Carolina for the past 80 years, and global tide-gauge records for the past 300 years.

A second reconstruction from Massachusetts confirmed their findings.

The records were corrected for contributions to sea-level rise made by vertical land movements.

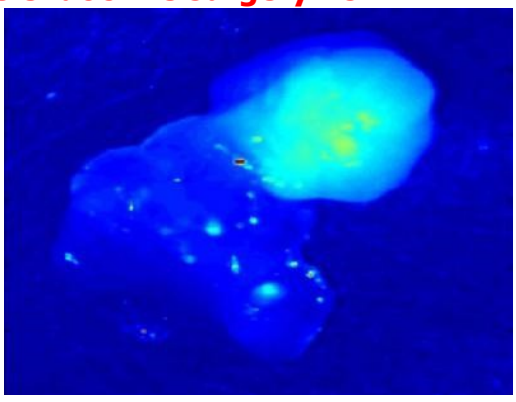
The reconstructed changes in sea level over the past millennium are consistent with past global temperatures, the researchers say, and can be determined using a model relating the rate of sea level rise to global temperature. "Data from the past helped calibrate our model, and will improve sea level rise projections under scenarios of future temperature increases," says Rahmstorf.

Support for the research also was provided by the National Oceanic and Atmospheric Administration, United States Geological Survey, the Academy of Finland, the European Science Foundation through European Cooperation in Science and Technology and the University of Pennsylvania.

http://www.eurekalert.org/pub_releases/2011-06/vu-dop062011.php

Discovery of parathyroid glow promises to reduce endocrine surgery risk

The parathyroid glands – four small organs the size of grains of rice located at the back of the throat – glow with a natural fluorescence in the near infrared region of the spectrum.



This unique fluorescent signature was discovered by a team of biomedical engineers and endocrine surgeons at Vanderbilt University, who have used it as the basis of a simple and reliable optical detector that can positively identify the parathyroid glands during endocrine surgery. The report of the discovery of parathyroid fluorescence and the design of the optical detector was published in the June issue of the Journal of Biomedical Optics.

Parathyroid tissue in the upper right emits a natural fluorescence in the infrared spectrum that is twice as strong as that of thyroid tissue in the lower left. The difference is strong enough that a simple optical system can distinguish between the two during endocrine surgeries. **Mahadevan-Jansen Laboratory**

Damage to these tiny organs can have deleterious, life-long effects on patients' health because they produce a hormone that controls critical calcium concentrations in bones, intestines and kidneys. However, the parathyroid glands are very difficult to identify with the naked eye. Not only are they small, but their location also varies widely from person to person and it takes a microscope to reliably tell the difference between parathyroid tissue and the thyroid and lymph tissue that surrounds it.

In 2004, more than 80,000 endocrine surgeries were performed in the United States and this number is projected to grow to more than 100,000 by 2020. Today, when a surgeon cuts into a patient's neck to remove a diseased thyroid, somewhere between 8 to 19 percent of the time the patient's parathyroid glands are also damaged or removed.

Parathyroid glow is surprisingly strong

"We have discovered that the parathyroid glands are two to 10 times more fluorescent in the near infrared than any other tissues found in the neck," said Professor of Biomedical Engineering Anita Mahadevan-Jansen, who directed the study. "We have taken measurements with more than 50 patients now and we have found this effect 100 percent of the time, even when the tissue is diseased. That is amazing. You almost never get 100 percent results in biological studies."

The fluorescence is so strong that it doesn't take expensive or sophisticated instruments to detect. The Vanderbilt researchers have assembled a detector from off-the-shelf hardware. It consists of a low-powered infrared laser connected to an optical fiber probe. As the fiber connected to the laser illuminates the tissue with invisible near infrared light, other fibers in the probe are connected to a detector that measures the strength of the fluorescence that the laser excites. The university has applied for an international patent that covers this application.

"I was certainly impressed with how accurate this method seems to be," said John Phay, an endocrine surgeon at the Ohio State University Medical Center, who collaborated in the study when he was at Vanderbilt. "The ability to detect the parathyroids would be a big help: The major problem in parathyroid surgery is finding them and it is very hard to avoid them in thyroid cancer surgery when you need to clear out lymph nodes."

Using the first generation of the device was "a bit burdensome, because you have to dim the lights," Phay commented. This will not be a problem with the next version, because it will include a filter that will block out visible light. According to the surgeon, the system will be the most useful with the planned addition of a camera that displays the fluorescence of all the tissues in the throat on a single display.

Project begins with curiosity of first-year surgery resident

The story of discovery began in 2007 when Lisa White, a first-year resident in the Vanderbilt surgery department, participated in her first neck surgery. "It was a very difficult case," White said. "We were looking for the parathyroid glands and they were very hard to find, although we finally did find them. After the surgery was over, I decided that we really need a better way of identifying parathyroid tissue."

This conclusion led White to conduct a literature search of the research that has been conducted on the basic physiology and biochemistry of the parathyroid. In the process she came across a paper written by Mahadevan-Jansen with another intern that described an optical technique that can detect liver cancer. "I thought that if such a technique could detect the difference between normal and cancerous liver tissue, surely it could tell two different types of tissue apart," White said. So she decided to pay Mahadevan-Jansen a visit.

"One afternoon there was a knock on my door. It was Lisa and she asked me about methods for detecting the parathyroid," said Mahadevan-Jansen, whose research centers on the use of optical techniques for the detection of diseased tissue. "I was interested, particularly because the maternal side of my family has a history of thyroid problems. So I asked her if she would like to check it out."

Initial attempts didn't work

White agreed and they began working together even though they didn't have any funding to support the project. They recruited Phay, who was an attending endocrine surgeon at the time and had access to animal tissue from other experiments being conducted on campus. They tried several different optical techniques, but none of the methods revealed anything distinctive about parathyroid tissue.

Finally, Mahadevan-Jansen decided to try a technique called Raman spectroscopy that fingerprints different organic molecules by subtle changes in the color of reflected light. The effect is very weak and difficult to measure.

When they put thyroid tissue in the instrument, they got a small signal. When they inserted parathyroid tissue, however, the detector saturated. This was totally unexpected because most biological fluorescence takes place in the ultraviolet and visible ranges. Biological molecules rarely fluoresce in the infrared region of the spectrum.

Possible light leak turned out to be a real effect

"At first, we thought it must be a light leak," Mahadevan-Jansen said. When they kept getting the strong signal with several different samples, however, they realized that the effect was real.

Based on this success, Mahadevan-Jansen brought biomedical engineering graduate students Constantine Paras and Matthew Keller on board.

The expanded research team submitted an experimental protocol to Vanderbilt's Institutional Review Board, which must approve all experiments involving animals or people. When it was approved, they gained access to human thyroid and parathyroid tissue.

"The parathyroid tissue from those first dozen patients kept saturating the Raman spectrometer so we had to keep reducing the laser's intensity," Mahadevan-Jansen recalled. "Finally, I realized that the instrument was saturating because the tissue was fluorescing."

When she confirmed that this was happening, the engineer realized that they didn't need the Raman spectroscope. All they needed was a light source in the near infrared and the right kind of near infrared detectors.

Cause of fluorescence remains a mystery

"We still haven't figured out the source of the fluorescence, but that doesn't stand in the way of using this effect to improve the effectiveness of parathyroid surgeries and reduce the damage done to the parathyroid in other endocrine surgeries," Mahadevan-Jansen said.

Meanwhile, White is finishing up her final year as a resident in general surgery. She intends to make endocrine surgery a major part of her practice, so she could be one of the first surgeons whose patients will benefit from the discovery that resulted from her curiosity and initiative as a first-year intern.

http://www.usatoday.com/tech/science/columnist/vergano/2011-06-19-bow-arrows-origin_n.htm

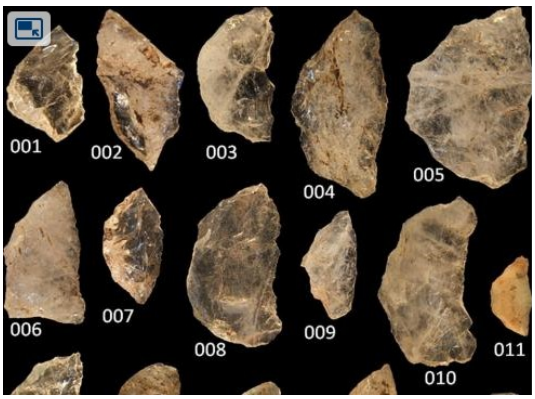
Arrow origins traced to Africa

Science Snapshot By Dan Vergano, USA TODAY

Back in 1991, archaeologists unearthed the frozen body of a man who died some 5,300 years ago in the Alps.

Nicknamed Otzi, for his resting place in the Ötztal Alps, the "Iceman" was outfitted with a copper ax, flint knife and bearskin hat, a surprise to archaeologists because they all were so well-crafted. His bow and 12 arrows, two of them nicely feathered and tipped with flint points, were likely less surprising, because they nicely fit with the then-current story of the bow and arrow's origins.

"The invention of the bow and arrow used to be closely linked to the late Upper Paleolithic (Stone Age) in Europe," less than 30,000 years ago, says anthropologist Marlize Lombard of South Africa's University of Johannesburg, in a study in the current Journal of Archaeological Science.



A collection of ancient arrowheads.

Last year, however, Lombard and her colleagues reported in the journal Antiquity, that arrows were around at least 64,000 years ago, and were first discovered not in Europe, but in South Africa. A single quartz arrowhead, bloodstained, had turned up at the Sibudu Cave site, dating to that time. In the new Journal of Archaeological Science study, Lombard reports more arrowheads and more evidence pushing back the age of the bow and arrow.

Why does it matter? Well, modern-looking humans turn up in fossils from as long as 195,000 years ago in Africa, but only spread worldwide starting about 60,000 years ago. Anthropologists have debated for decades about the innovations or changes, everything from language to genes to tools, that turned modern man loose on the world.

Arrows are one possibility for what helped people spread all over the world, either through hunting or fighting, as Lombard cautiously notes. "Although the existence of bow and arrow technology (more than 60,000 years ago) may have far-reaching consequences for hypotheses about human behavioural evolution and adaptation, it is by no means easy to establish," she says at the beginning of her study. In the study, she looks through the microscope at 16 quartz blades found in dirt layers as much as 65,000 years old at the South African site.

All but two of the ancient blades have blood traces on them and nine were deliberately hafted, or chipped, to fit onto a tool, she finds. More than half of the blades look like they were attached to arrows and eight carry

traces of blood stains, Lombard concludes. "It is therefore my reading that at least nine tools in this sample were probably used as transversely hafted arrowheads."

The others may have been blades used to butcher animals, she suggests, or fitted onto barbs or darts.

"I think the finding adds to growing evidence for the great antiquity of complex projectile weaponry in Africa," says paleoanthropologist John Shea of Stony Brook (N.Y.) University. "The real startling upshot of this finding is that it challenges longstanding archaeological beliefs that important changes in projectile technology only occurred very recently, less than 30,000 years ago, after humans dispersed into Europe."

In North America, Shea adds, "it also challenges the longstanding hypothesis that the bow and arrow were only invented a few thousand years ago and largely in conjunction with the origins of agriculture."

Even after prehistoric people invented arrows, they likely kept on using spears as well, Lombard suggests. Hunters in Africa still use spears to run down wildebeest and zebra, while using arrows only during part of the year to hunt for giraffe, eland, hartebeest and springbok. So, she concludes, archaeologists shouldn't be surprised when they find both heavier spear points and arrowheads mixed together at future archaeological digs.

" Complex projectile technology may have given our species a crucial ecological advantage in competition with other hominin (human) species as they dispersed from Africa," Shea says, by e-mail. That's one explanation for the disappearance of the Neanderthals, who have left only spear points behind at sites in Europe. Outgunned by modern humans and their arrows, the (literal) "killer app" of its day, the Neanderthals weren't able to compete for game and faded from the archaeological record (if not completely from our genes) about 30,000 years ago.

And the age of the bow and arrow may go further back, Shea says. "My own personal hunch is that the bow and arrow dates to at least 100,000 years ago based on stone tools found at sites in Ethiopia, Kenya and neighboring countries." No wonder Otzi had such nice arrows. Archery was an ancient technology in his day. Unfortunately for the Iceman.

After a puncture wound was discovered in Otzi's left shoulder a decade ago, researchers at Italy's South Tyrol Museum of Archaeology, X-rayed the wound and found what had killed the Iceman - a flint arrowhead that severed a major artery and likely paralyzed his arm. "The Iceman probably bled to death within a matter of minutes," the museum notes, because of the arrowhead.

http://www.eurekalert.org/pub_releases/2011-06/uoc-dai062011.php

Diagnosed autism is more common in an IT-rich region

Research provides important insight into 'systemizing' theory of autism

A new study from Cambridge University has for the first time found that autism diagnoses are more common in an IT-rich region.

The Medical Research Council (MRC) funded study, published today in the Journal of Autism and Developmental Disorders, has important implications for service provision in different regions and for the 'hyper-systemizing' theory of autism.

Professor Simon Baron-Cohen, Director of the Autism Research Centre (ARC) at the University of Cambridge, led the study (which was conducted in the Netherlands) with Dr Rosa Hoekstra, a Dutch autism researcher based at ARC and The Open University.

The researchers predicted that autism spectrum conditions (ASC) would be more common in populations enriched for 'systemizing', which is the drive to analyse how systems work, and to predict, control and build systems. These skills are required in disciplines such as engineering, physics, computing and mathematics.

The team had previously discovered evidence for a familial association between a talent for systemizing and autism in that fathers and grandfathers of children with ASC are over-represented in the field of engineering. The team had also previously found that mathematicians more often have a sibling with ASC, and students in the natural and technological sciences, including mathematics, show a higher number of autistic traits.

The researchers tested for differences in the prevalence of ASC in school-aged children in three geographical regions in the Netherlands: Eindhoven, Haarlem, and Utrecht-city. The region Eindhoven was selected because it is rich in information-technology (IT) having the Eindhoven University of Technology there, as well as the High Tech Campus Eindhoven, where IT and technology companies such as Philips, ASML, IBM and ATOS Origin are based. (The Philips factory has been in Eindhoven since 1891. Since then, the region has attracted businesses in IT and technology.)

The growth of the High Tech Campus Eindhoven has led to Eindhoven becoming a major technology and industrial hub: 30% of jobs in Eindhoven are now in technology or ICT, in Haarlem and Utrecht this is respectively 16 and 17%.

The two control regions were selected because they have similar size populations and a similar socioeconomic class. Schools in each region were asked to provide the number of children enrolled, the number

having a clinical diagnosis of ASC and/or two control neurodevelopmental conditions (dyspraxia and ADHD). The participating schools in the three regions provided diagnostic information on a total of 62,505 children. The researchers found school-reported prevalence estimates of ASC in Eindhoven was 229 per 10,000, significantly higher than in Haarlem (84 per 10,000) and Utrecht (57 per 10,000), whilst the prevalence for the control conditions were similar in all regions.

Simon Baron-Cohen commented: "These results are in line with the idea that in regions where parents gravitate towards jobs that involve strong 'systemizing', such as the IT sector, there will be a higher rate of autism among their children, because the genes for autism may be expressed in first degree relatives as a talent in systemizing. The results also have implications for explaining how genes for autism may have persisted in the population gene pool, as some of these genes appear linked to adaptive, advantageous traits."

Rosa Hoekstra added: "We need to conduct a follow-up study to validate the diagnoses and to test the alternative explanations for the elevated rate of autism in Eindhoven, including the possibility that children with autism may more often remain undetected in the two other regions. These results are important findings in the field of autism epidemiology, since they suggest regional variation in autism prevalence. In our follow-up study we plan to study the causes of this variation in more detail. This will help local authorities plan services appropriately for the number of children with autism."

The Cambridge research team also included Martine Roelfsema (a Dutch graduate student), Sally Wheelwright and Dr Carrie Allison (experts in autism screening), and Professor Carol Brayne and Dr Fiona Matthews (experts in public health research and biostatistics).

Notes to editors:

1. Authors: Martine T. Roelfsema, Rosa A. Hoekstra, Carrie Allison, Sally Wheelwright, Carol Brayne, Fiona E. Matthews, Simon Baron-Cohen (2011). Are autism spectrum conditions more prevalent in an information-technology region? A school-based study of three regions in the Netherlands. Journal of Autism and Developmental Disorders, DOI 10.1007/s10803-011-1302-1

2. Author affiliations: Autism Research Centre, Department of Psychiatry, Cambridge University, Cambridge CB2 8AH, UK, (www.autismresearchcentre.com); Department of Life Sciences, The Open University, Milton Keynes, UK; Department of Public Health and Primary Care, Institute of Public Health, University of Cambridge, Cambridge, UK; MRC Biostatistics Unit, Institute of Public Health, Cambridge UK

3. Funding sources: Research grants from the Medical Research Council (UK); Target Autism Genome; the Nancy Lurie Marks Family Foundation; NIHR CLAHRC for Cambridgeshire and Peterborough NHS Foundation Trust; the Erasmus fund, the Bekker la Bastide fund and the University of Amsterdam and the Netherlands Organisation for Scientific Research (NWO Rubicon)

http://www.physorg.com/news/2011-06-japan-gadget-cellphone-campfire.html

Japan gadget charges cellphone over campfire

TES NewEnergy unveils a pot that can charge mobile phones while boiling water for use in other emergency situations

A Japanese company has come up with a new way to charge your mobile phone after a natural disaster or in the great outdoors - by heating a pot of water over a campfire.

The Hatsuden-Nabe thermo-electric cookpot turns heat from boiling water into electricity that feeds via a USB port into digital devices such as smartphones, music players and global positioning systems.

TES NewEnergy, based in the western city of Osaka, started selling the gadget in Japan this month for 24,150 yen ($299), and plans to market it later in developing countries with patchy power grids.

Chief executive Kazuhiro Fujita said the invention was inspired by Japan's March 11 earthquake and tsunami that left 23,000 people dead or missing, devastated the northeast region and left hundreds of thousands homeless.

A member of Japanese electronics venture TES NewEnergy unveils a pot that can charge mobile phones while boiling water for use in earthquake and other emergency situations, at a demonstration in Tsukuba City in Ibaraki prefecture on June 9. The Hatsuden-Nabe thermo-electric cookpot turns heat from boiling water into electricity that feeds via a USB port into digital devices such as smartphones.

"When I saw the TV footage of the quake victims making a fire to keep themselves warm, I came up with the idea of helping them to charge their mobile phones at the same time," Fujita said.

The pot features strips of ceramic thermoelectric material that generate electricity through temperature differentials between the 550 degrees Celsius at the bottom of the pot and the water boiling inside at 100 degrees.

The company says the device takes three to five hours to charge an iPhone and can heat up your lunch at the same time. "Unlike a solar power generator, our pot can be used regardless of time of day and weather while its small size allows people to easily carry it in a bag in case of evacuation," said director and co-developer Ryoji Funahashi.

TES NewEnergy was set up in 2010 to promote products based on technology developed at the National Institute of Advanced Industrial Science and Technology, Japan's largest public research organisation. It also makes and markets equipment to transform residual heat from industrial waste furnaces into electricity.

The company says the pot will be used mainly in emergency situations and for outdoor activities, but also has uses in developing countries. "There are many places around the world that lack the electric power supply for charging mobile phones," Fujita said. "In some African countries, for example, it's a bother for people to walk to places where they can charge mobile phones. We would like to offer our invention to those people."

http://www.physorg.com/news/2011-06-biologists-puzzling-parasite.html

Biologists shed light on a puzzling parasite

Toxoplasma gondii, a parasite that infects about one-third of the world's population, comes in several strains. Some can have severe consequences such as encephalitis, while others produce no noticeable symptoms.

Jeroen Saeij, an MIT biologist who has been studying Toxoplasma for several years, is trying to figure out the root of that discrepancy. In his latest work, he found that two of the three most common strains of Toxoplasma produce a protein that actually suppresses inflammation in the infected host - a discovery that could help researchers develop new ways to shut off inflammation in patients infected with the more threatening strains of Toxoplasma, or even in people with other inflammatory diseases such as Crohn's disease.

"There's a lot of these inflammatory diseases, and if there's a general pathway that's really good at quelling inflammation, there might be [drug] applications," says Saeij, an assistant professor of biology.

Saeij and his colleagues described the new discovery in the June 15 issue of Cell Host & Microbe. Lead author of the paper is postdoc Kirk Jensen.

Inflammation

Toxoplasma spores, which can infect nearly any warm-blooded animal, are often found in dirt; humans can be infected by eating undercooked meat or unwashed vegetables. The parasite can cause encephalitis in individuals with suppressed immune systems and is also harmful to fetuses whose mothers become infected during pregnancy. Some strains, primarily those found in South America, can lead to blindness.

There are about a dozen major types of strains of Toxoplasma; in North America and Europe, the most common is the type II strain, which is also the one most often isolated from patients with complications from Toxoplasma. In a previous paper, Saeij and colleagues showed that type II produces a form of the protein GRA15 that causes inflammation, an immune reaction that is meant to destroy invaders but can also damage the host's own tissues if unchecked.

In the new paper, Saeij showed that type II also has a variant form of another protein called ROP16. The researchers found that in types I and III, which are less often found in patients with Toxoplasma symptoms, ROP16 suppresses inflammation in the host. However, the form of ROP16 found in type II has no such effect.

When the researchers inserted type I ROP16 into the type II strain, inflammation was suppressed.

Why opposite results from strains of the same parasite? The researchers believe different forms of the parasite evolved to take advantage of different immune reactions from potential hosts. Toxoplasma wants to provoke some inflammation in the host, because inflammation helps the host survive acute infection. As a consequence, the parasite can form tissue cysts that spread to other victims through carnivorism. However, too much inflammation might kill the host and stop the parasite's life cycle. By employing different strategies, the parasite can be successful in hosts with varying inflammatory responses.

Jon Boyle, an assistant professor of biological sciences at the University of Pittsburgh who was not involved with this research, agrees with this assessment: "What the paper shows really well is that you have these different strains and even though they are quite similar, each type is adapted to different host environments."

A model for Crohn's disease

In the new paper, the researchers studied Toxoplasma infection in mice. The resulting inflammation of the mouse intestine strongly resembles the intestinal inflammation seen in Crohn's disease, which affects about half a million people in the United States.

It is unknown if any of these cases are actually caused by Toxoplasma, but Saeij says some studies have shown a higher incidence of Toxoplasma in Crohn's patients compared with the general population.

Saeij and Jensen are now trying to figure out how the ROP16 protein exerts its anti-inflammatory effects, which could help identify new approaches to developing anti-inflammatory drugs. However, that will likely require many more years of research, Saeij says.

“In reality, going from a discovery to a drug can take decades,” he says. “You hope that you contribute to something like that, but that’s always a long road.” *Provided by Massachusetts Institute of Technology*

http://medicalxpress.com/news/2011-06-context-short-words-year-old-language.html

Why context matters in the long and short of words: Researchers improve 75-year-old language theory

Why context matters in the long and short of words

In 1935, Harvard University linguist George Kingsley Zipf asserted that "the magnitude of words tends, on the whole, to stand in an inverse, not necessarily proportionate, relationship to the number of occurrences." In other words, short words are used more frequently than long ones. Now, cognitive scientists at the Massachusetts Institute of Technology demonstrated a substantial improvement to Zipf's law.

(Medical Xpress) - Do you ever wonder about the stuff that makes up words? Why is a word a word, what goes into forming it, what's its history or why is it long or short? Scientists at the Massachusetts Institute of Technology do.

Steven Piantadosi, Harry Tily and Edward Gibson study words for MIT's Department of Brain and Cognitive Sciences to understand how humans think and communicate.

Recently, they put a well-established, 75-year-old language theory to the test and found it had room for improvement. At issue was something called Zipf's law, an empirical scientific principle that says word length is primarily determined by frequency of use.

In 1935, Harvard University linguist George Kingsley Zipf asserted "the magnitude of words tends, on the whole, to stand in an inverse, not necessarily proportionate, relationship to the number of occurrences." In other words, short words are used more than long ones.

"One widely known and apparently universal property of human language is that frequent words tend to be short," the researchers write in their report. They note short words are used to make communication more efficient than what can be had with frequent use of longer words.

This is because of pressure for communicative efficiency, Zipf surmised. It would be impractical to ask everyone at a Thanksgiving dinner whether they would like a bowl of soup using a 15-letter word for "of," for example.

In the Brown University Standard Corpus of Present-Day American English, which contains about two million words of text, "of" is the fourth most commonly used word. Meanwhile, "the" is used more in writing than any other word in the English language. In fact, a list of the top 100 most frequently used words contains words such as "be," "on," "have," "with," "who," and "some," all very short words.

But the cognitive scientists at MIT demonstrated a substantial improvement to Zipf's law. They showed that across 10 languages the predictability of what a person says is a more important determinant of word length than how often he or she says it.

Word length actually comes down to the amount of information it contains

The goal of the research was to compare Zipf's word frequency theory to Piantadosi and colleagues' word predictability theory - the idea that the average amount of information a word conveys in context - its predictability - determines word length.

Using an Internet database, the researchers studied how often all possible sequences of two, three or four word combinations occur together in order to estimate how predictable any word is when it's typically written.

By knowing this, they could determine whether context and predictability were better determinants of word length than frequency of use.

"For instance, in a context like ‘Monday night ____' the word ‘football' is very predictable and therefore conveys very little information," said Piantadosi, a cognitive scientist in the Ph.D. program at MIT and lead author of the study. "But, in a context like ‘I ate ____,' the missing word is very unpredictable, but conveys a lot of information."

The hypothesis was that average information contained in two, three or four word sequences should in part determine the length of words, either in letters or syllables, since that's how an optimal code would behave. In this example, "football" and the two words preceding it demonstrated the effect.

"The only way these effects can get in to the lexicon is if our linguistic systems, and the mechanisms of language change, are sensitive to communicative pressures," said Piantadosi.

The sequences of words that people use are coded - their letters, syllables, sounds, etc. - for efficient communication and are better predictors of word length than frequency alone, he said.

"This means word sequences provide efficient codes for the meanings they convey, relative to the statistical regularities in language," he said. "That's our claim."

Context matters for love, amour, liebe, amor and kärlek,

Love, amour, liebe, amor and kärlek all mean the same thing across different languages and all are about the same length, which according to Zipf is what should be expected if they were similarly predictable or informative. But the MIT researchers stress it's the words before and after a particular word that determines how often the particular word is used, not length.

True, the word for strong fondness is very short, but how frequently do people say it, what are the circumstances when they do and how predictable is the information conveyed when it's said? Saying "I love you" is quite different from saying "I love chicken." For a word like "love," context matters.

The research results held across all but one of the languages studied: Czech, Dutch, English, French, German, Italian, Portuguese, Romanian, Spanish and Swedish, with German being the outlier.

"I was surprised that we found effects in so many languages," said Piantadosi. "I would have thought that differences in morphology, or word structure, might have swamped our effects in many languages, but this doesn't appear to be the case."

Why the most frequently used words are short

The research findings also provide an improved explanation as to why the most often used words are short - because they tend to be predictable, meaning many short words, on average, convey relatively little information. Of the top 100 words, many are "function words," whose main purpose is to join words together such as - "with," "from" and "over." By themselves, these words give the reader or listener a very small amount of data.

The researchers also found short words must be paired with other familiar words to derive context and convey information. This is because many times words occurring after well-known sequences of other words are the most predictable and contain the least information; for example "a ton of fun," is a well known sequence of words that conveys very little information. But words that have a little association to the words preceding them contain more information; for example, "a ton of butter."

A final word

The research revealed that people communicate through at least an approximately optimal code for meaning, said Piantadosi. "Lexicons are not arbitrary in the sense of being completely random. Instead, they are well-structured for communication, given the patterns of word sequences people typically use."

The problem with the traditional method of only looking at word frequency is that it merely involves counting words in isolation and does not consider the regular dependencies between words.

The research is published in the Proceeding of the National Academy of Sciences in an article titled "Word lengths are optimized for efficient communication." The National Science Foundation's Division of Behavioral and Cognitive Sciences funds the research.

More information: Word lengths are optimized for efficient communication: http://www.pnas.or … 108.abstract
Provided by National Science Foundation

http://medicalxpress.com/news/2011-06-natural-alzheimer-weapon-treatment.html

Natural Alzheimer's weapon suggests better treatment

Scientists have shown a molecular chaperone is working like a waste management company to collect and detoxify high levels of toxic amyloid beta peptide found in Alzheimer's disease.

It was known that the molecular chaperone, HspB1, was present in the hallmark plaque of Alzheimer's patients but its role remained a mystery.

"What we have found is HspB1 is a protective mechanism that tries to get rid of the toxic oligomers or aggregates of amyloid beta that occur in Alzheimer's," said Dr. Anil G. Cashikar, Biochemist at Georgia Health Sciences University's Center for Molecular Chaperones and Radiobiology. He is corresponding author of the study published in Molecular and Cellular Biology.

Amyloid beta peptide, or Abeta, is believed to start the cascade of events that leads to brain cell damage and death in Alzheimer's: as levels increase, the peptide starts clumping in the brain. In fact, high levels in the spinal fluid are a diagnostic marker for the disease. Molecular chaperones are known for their propensity to respond to disease-producing misfolded proteins, which is how the body views excessive Abeta.

While resulting plaques occupy prime real estate in the brain, it's still better than toxic Abeta killing neurons, Cashikar said. "We think maybe the system gets overwhelmed."

Acknowledging much work remains, the scientist is excited about identifying the protective mechanism and exploring its treatment potential.

Earlier this year, a paper Cashikar published in PLoS One showed deleting genes with a similar function from a mouse model of Alzheimer's worsened disease symptoms. The new study also showed neurons from HspB1-deficient mice were more sensitive to the toxic ravages of Abeta.

"HspB1 is present because its function is to protect cells. The implication is if we can elevate the levels of this molecular chaperone, we may be able to handle the situation a little better," Cashikar said.

He wants to exploit this natural system by developing a smaller version of the molecular chaperone that could be put into the bloodstream to leach excess Abeta from the brain. The brain has a natural protective mechanism that likely would prevent its direct application. However, the natural affinity of amyloid beta and HspB1 indicates a more distant approach could be effective. "We want to come up with smaller versions of HspB1 that can be put into the bloodstream so you can sop up the material from the brain into the blood where it can be cleared more efficiently." He also wants to explore a way to increase brain cells' natural production of protective HspB1.

Neurons actually also make the Abeta believed to attack them in Alzheimer's. The peptide's normal function in the brain is not clear, but early evidence suggests it could be involved in synaptic pruning, which is essential for memory formation. Synapses connect neurons and some existing connections must be cut for new connections and memories to be made. Why neurons start making too much Abeta and how its overproduction can be controlled are million-dollar questions, Cashikar said.

A related ongoing debate is whether the amyloid plaques and neurofibrillarly tangles, insoluble globs of protein also found in Alzheimer's, are a cause or result of the disease. Cashikar's work as well as new studies on the neurofibrillarly tangles, suggest both are protective mechanisms. Also, there is evidence of both in the brains of some healthy, elderly individuals.

GHSU Graduate Student Juhi Ojha is first author on the paper. Provided by Georgia Health Sciences University

http://www.scientificamerican.com/podcast/episode.cfm?id=rocking-increases-brain-activity-as-11-06-20

Rocking Increases Brain Activity Associated with Sleep

Volunteers were scanned when they napped in stationary and in rocking beds, revealing enhanced sleep brain activity when rocking. Cynthia Graber reports

You're lying in a hammock by a breezy shore. The hammock rocks softly back and forth. In no time… (snoring). It turns out that's not just the relaxation of being on vacation that's bringing on sleep. It's the rocking hammock. That might not be a huge surprise - babies get rocked to sleep. But researchers wanted to know how rocking works.

They recruited 12 healthy males, all good sleepers. Each volunteer twice took an afternoon nap in a dark room on a custom-made bed that could rock. For one nap, the bed was still. For the other, it rocked gently.

All the men fell asleep faster when they swayed. And the scientists monitored the men's brain activity during all the naps. They found that rocking increased the duration of what's called N2, a non-REM stage that accounts for about half of a good night's sleep.

Rocking also increased deep-sleep-associated brain activity - so-called slow oscillations as well as bursts of action called sleep spindles. The research was published in the journal Current Biology. [Laurence Bayer et al., "Rocking Synchronizes Brain Waves During a Short Nap"]

The next step is to find out whether rocking can help treat sleep disorders. Meanwhile, insomniacs can always try a hammock. - *Cynthia Graber*

http://www.eurekalert.org/pub_releases/2011-06/uomm-chs062111.php

Can humans sense the Earth's magnetism?

New research shows that the human retina protein, CRY2, has the molecular capability to function as a light-sensitive magnetic sensor

WORCESTER, Mass. – For migratory birds and sea turtles, the ability to sense the Earth's magnetic field is crucial to navigating the long-distance voyages these animals undertake during migration. Humans, however, are widely assumed not to have an innate magnetic sense. Research published in Nature Communications this week by faculty at the University of Massachusetts Medical School shows that a protein expressed in the human retina can sense magnetic fields when implanted into Drosophila, reopening an area of sensory biology in humans for further exploration.

In many migratory animals, the light-sensitive chemical reactions involving the flavoprotein cryptochrome (CRY) are thought to play an important role in the ability to sense the Earth's magnetic field. In the case of Drosophila, previous studies from the Reppert laboratory (http://reppertlab.org/) have shown that the cryptochrome protein found in these flies can function as a light-dependent magnetic sensor.

To test whether the human cryptochrome 2 protein (hCRY2) has a similar magnetic sensory ability, Steven Reppert, MD, the Higgins Family Professor of Neuroscience and chair and professor of neurobiology, graduate

student Lauren Foley, and Robert Gegear, PhD, a post doctoral fellow in the Reppert lab now an assistant professor of biology and biotechnology at Worcester Polytechnic Institute, created a transgenic Drosophila model lacking its native cryptochrome protein but expressing hCRY2 instead. Using a behavioral system Reppert's group previously developed, they showed that these transgenic flies were able to sense and respond to an electric-coil-generated magnetic field and do so in a light-dependent manner.

These findings demonstrate that hCRY2 has the molecular capability to function in a magnetic sensing system and may pave the way for further investigation into human magnetoreception. "Additional research on magneto sensitivity in humans at the behavioral level, with particular emphasis on the influence of magnetic field on visual function, rather than non-visual navigation, would be informative," wrote Reppert and his colleagues in the study.

http://www.eurekalert.org/pub_releases/2011-06/uosf-mii062011.php

Mystery ingredient in coffee boosts protection against Alzheimer's disease

Tampa, FL – *A yet unidentified component of coffee interacts with the beverage's caffeine, which could be a surprising reason why daily coffee intake protects against Alzheimer's disease.*

A new Alzheimer's mouse study by researchers at the University of South Florida found that this interaction boosts blood levels of a critical growth factor that seems to fight off the Alzheimer's disease process.

The findings appear in the early online version of an article to be published June 28 in the Journal of Alzheimer's Disease. Using mice bred to develop symptoms mimicking Alzheimer's disease, the USF team presents the first evidence that caffeinated coffee offers protection against the memory-robbing disease that is not possible with other caffeine-containing drinks or decaffeinated coffee.

Previous observational studies in humans reported that daily coffee/caffeine intake during mid-life and in older age decreases the risk of Alzheimer's disease. The USF researchers' earlier studies in Alzheimer's mice indicated that caffeine was likely the ingredient in coffee that provides this protection because it decreases brain production of the abnormal protein beta-amyloid, which is thought to cause the disease.

The new study does not diminish the importance of caffeine to protect against Alzheimer's. Rather it shows that caffeinated coffee induces an increase in blood levels of a growth factor called GCSF (granulocyte colony stimulating factor). GCSF is a substance greatly decreased in patients with Alzheimer's disease and demonstrated to improve memory in Alzheimer's mice. A just-completed clinical trial at the USF Health Byrd Alzheimer's Institute is investigating GCSF treatment to prevent full-blown Alzheimer's in patients with mild cognitive impairment, a condition preceding the disease. The results of that trial are currently being evaluated and should be known soon.

"Caffeinated coffee provides a natural increase in blood GCSF levels," said USF neuroscientist Dr. Chuanhai Cao, lead author of the study. "The exact way that this occurs is not understood. There is a synergistic interaction between caffeine and some mystery component of coffee that provides this beneficial increase in blood GCSF levels." The researchers would like to identify this yet unknown component so that coffee and other beverages could be enriched with it to provide long-term protection against Alzheimer's.

In their study, the researchers compared the effects of caffeinated and decaffeinated coffee to those of caffeine alone. In both Alzheimer's mice and normal mice, treatment with caffeinated coffee greatly increased blood levels of GCSF; neither caffeine alone or decaffeinated coffee provided this effect. The researchers caution that, since they used only "drip" coffee in their studies, they do not know whether "instant" caffeinated coffee would provide the same GCSF response.

The boost in GCSF levels is important, because the researchers also reported that long-term treatment with coffee (but not decaffeinated coffee) enhances memory in Alzheimer's mice. Higher blood GCSF levels due to coffee intake were associated with better memory. The researchers identified three ways that GCSF seems to improve memory performance in the Alzheimer's mice. First, GCSF recruits stem cells from bone marrow to enter the brain and remove the harmful beta-amyloid protein that initiates the disease. GCSF also creates new connections between brain cells and increases the birth of new neurons in the brain.

"All three mechanisms could complement caffeine's ability to suppress beta amyloid production in the brain" Dr. Cao said, "Together these actions appear to give coffee an amazing potential to protect against Alzheimer's - but only if you drink moderate amounts of caffeinated coffee."

Although the present study was performed in Alzheimer's mice, the researchers indicated that they've gathered clinical evidence of caffeine/coffee's ability to protect humans against Alzheimer's and will soon publish those findings.

Coffee is safe for most Americans to consume in the moderate amounts (4 to 5 cups a day) that appear necessary to protect against Alzheimer's disease. The USF researchers previously reported this level of coffee/caffeine intake was needed to counteract the brain pathology and memory impairment in Alzheimer's

mice. The average American drinks 1½ to 2 cups of coffee a day, considerably less than the amount the researchers believe protects against Alzheimer's.

"No synthetic drugs have yet been developed to treat the underlying Alzheimer's disease process" said Dr. Gary Arendash, the study's other lead author. "We see no reason why an inherently natural product such as coffee cannot be more beneficial and safer than medications, especially to protect against a disease that takes decades to become apparent after it starts in the brain."

The researchers believe that moderate daily coffee intake starting at least by middle age (30s – 50s) is optimal for providing protection against Alzheimer's disease, although starting even in older age appears protective from their studies. "We are not saying that daily moderate coffee consumption will completely protect people from getting Alzheimer's disease," Dr. Cao said. "However, we do believe that moderate coffee consumption can appreciably reduce your risk of this dreaded disease or delay its onset."

The researchers conclude that coffee is the best source of caffeine to counteract the cognitive decline of Alzheimer's because its yet unidentified component synergizes with caffeine to increase blood GCSF levels. Other sources of caffeine, such as carbonated drinks, energy drinks, and tea, would not provide the same level of protection against Alzheimer's as coffee, they said.

Coffee also contains many ingredients other than caffeine that potentially offer cognitive benefits against Alzheimer's disease. "The average American gets most of their daily antioxidants intake through coffee," Dr. Cao said. "Coffee is high in anti-inflammatory compounds that also may provide protective benefits against Alzheimer's disease."

An increasing body of scientific literature indicates that moderate consumption of coffee decreases the risk of several diseases of aging, including Parkinson's disease, Type II diabetes and stroke. Just within the last few months, new studies have reported that drinking coffee in moderation may also significantly reduce the risk of breast and prostate cancers.

"Now is the time to aggressively pursue the protective benefits of coffee against Alzheimer's disease," Dr. Arendash said. "Hopefully, the coffee industry will soon become an active partner with Alzheimer's researchers to find the protective ingredient in coffee and concentrate it in dietary sources."

New Alzheimer's diagnostic guidelines, now encompassing the full continuum of the disease from no overt symptoms to mild impairment to clear cognitive decline, could double the number of Americans with some form of the disease to more than 10 million. With the baby-boomer generation entering older age, these numbers will climb even more unless an effective preventive measure is identified.

"Because Alzheimer's starts in the brain several decades before it is diagnosed, any protective therapy would obviously need to be taken for decades," Dr. Cao said. "We believe moderate daily consumption of caffeinated coffee is the best current option for long-term protection against Alzheimer's memory loss. Coffee is inexpensive, readily available, easily gets into the brain, appears to directly attack the disease process, and has few side-effects for most of us."

According to the researchers, no other Alzheimer's therapy being developed comes close to meeting all these criteria.

"Aside from coffee, two other lifestyle choices - physical and cognitive activity - appear to reduce the risk of dementia. Combining regular physical and mental exercise with moderate coffee consumption would seem to be an excellent multi-faceted approach to reducing risk or delaying Alzheimer's," Dr. Arendash said. "With pharmaceutical companies spending millions of dollars trying to develop drugs against Alzheimer's disease, there may very well be an effective preventive right under our noses every morning – caffeinated coffee."

This USF study was funded by the NIH-designated Florida Alzheimer's Disease Research Center and the State of Florida.

Article citation:

Caffeine Synergizes with Another Coffee Component to Increase Plasma GCSF: Linkage to Cognitive Benefits in Alzheimer's Mice; *Chuanhai Cao, Li Wang, Xiaoyang Lin, Malgorzata Mamcarz, Chi Zhang, Ge Bai, Jasson Nong, Sam Sussman and Gary Arendash; Journal of Alzheimer's Disease, 25(2), June 28, 2011.*

http://www.eurekalert.org/pub_releases/2011-06/uof-uro062111.php

UF review of resveratrol studies confirms potential health boost

GAINESVILLE, Fla. - ***A University of Florida review of research finds the polyphenol compound known as resveratrol found in red wine, grapes and other fruits may not prevent old age, but it might make it more tolerable.***

News stories have long touted resveratrol as a cure for various diseases and a preventative against aging.

"We're all looking for an anti-aging cure in a pill, but it doesn't exist. But what does exist shows promise of lessening many of the scourges and infirmities of old age," said UF exercise psychologist Heather Hausenblas, one of the researchers involved in the study.

A comprehensive review of human clinical research on resveratrol has found it has "anti-aging, anti-carcinogenic, anti-inflammatory and antioxidant properties," but more research of its benefits is needed, she said.

The study, which appeared online this week in Molecular Nutrition and Food Research, examined results gleaned from thousands of laboratory studies with enzymes, cultured cells and laboratory animals. It was conducted by Hausenblas and fellow researchers James Smoliga of Marywood University and Joseph Baur of the University of Pennsylvania School of Medicine. Their review aimed to examine the current state of knowledge of the effects of resveratrol on humans and to use this information to guide much needed future human clinical trials.

Despite numerous clinical studies on resveratrol's tonic effects on animals, there is little evidence that it benefits human health. That's because "there haven't been many studies on humans," Hausenblas said.

However, she points out, for years many scientists have thought that a link between resveratrol and human health exists. The French people, for example, enjoy low levels of cardiovascular disease, even though their diets are rich in saturated fats and oils. Some researchers think the reason for this paradox lies in France's national drink - red wine, which is the most important dietary source of resveratrol. The UF review, said Hausenblas, shows that the resveratrol has considerable potential to improve health and prevent chronic disease in humans. However, further research examining the long-term health effects of resveratrol is much needed.

Exactly how resveratrol works isn't yet fully understood. Correlating factors such as metabolism, the chemical interplay of molecules, genetics, exercise, age, dosage, and many others all play a role.

Among resveratrol's most intriguing aspects is how it functions as an antioxidant. Oxidation is a natural chemical process in living tissues that results in a transfer of electrons. When this happens, groups of atoms are formed called "free radicals" that can cause cell damage which in turn provides a pathway for diseases. Antioxidants, however, suppress free radicals. "It's not so easy to say resveratrol is the main factor," Hausenblas said. "It's one piece of the overall puzzle that reduces the free radicals."

The UF study also reveals that resveratrol's contribution to good health promises to be widespread. Various clinical trials, for example, indicate that this polyphenol - an antibiotic substance produced by plants as a defense against microorganisms - prevents the growth of some cancers in mice, inhibits enzymes that cause inflammation, shrinks tumors and increases blood flow, thus reducing cardiovascular diseases. In many cases, it also extends the life of obese animals. Some evidence also shows that resveratrol could one day be used to help regulate insulin sensitivity in diabetic patients.

Hausenblas and her colleagues think research that explores resveratrol's potential to alleviate human infirmities will become increasingly more important as the nation's 76 million baby boomers undergo the aging process. One trial under way at UF's College of Medicine in the Institute on Aging examines the effect resveratrol may have on the physical and cognitive skills on older people. ***Credits*** *Writer John Dunn*

http://www.bbc.co.uk/news/health-13858115

Scarlet fever hits Hong Kong

Officials are warning of an outbreak of scarlet fever among children in Hong Kong.

There have been more than 400 cases of the disease this year, including the death of a six-year-old last month. Initial tests suggest a five-year-old boy may also have died from the bacterial infection, which is spread by coughing and sneezing.

Scientists in Hong Kong believe the bacteria may be spreading more quickly than usual due to a genetic mutation. Cases have also been seen in mainland China and Macau.

Dr Thomas Tsang, controller of Hong Kong's Centre for Health Protection, said: "If the genetic mutation is responsible for the increased transmissibility of the bacteria, the outbreak may be sustained for some time."

Scarlet fever happens every year in the region, but this year there have been more cases than usual.

Hong Kong has had 419 cases, already the highest annual total in the city.

Most are in children under 10, with clusters in kindergartens, primary schools and childcare centres.

Scarlet fever

Scarlet fever is due to a throat infection caused by a bacterium called streptococcus

A strain called group A streptococcus causes scarlet fever

Scarlet fever causes a sore throat, high temperature and a rash

It usually occurs in children

The tongue may become pale but coated with red spots (strawberry tongue)

Treatment is with antibiotics like penicillin

Source: Patient UK

Scarlet fever infections have gone up around fivefold in China, and threefold in Macau, about an hour by ferry from Hong Kong.

According to a government statement, there may be regional outbreak of scarlet fever.

http://medicalxpress.com/news/2011-06-reveals-pigs-human.html

New study reveals pigs could grow human organs

(PhysOrg.com) - *At the annual European Society of Human Genetics conference, a group of researchers presented their newly discovered technique that may soon enable pigs to grow human organs for transplant.*

Lead researcher and the director of the Center for Stem Cell Biology and Regenerative Medicine at the University of Tokyo Professor Hiromitsu Nakauchi described the new technique called blastocyst complementation.

Using mice and rats the researchers injected rat's stem cells into mice which had been genetically altered so they were unable to produce their own organs. The mice instead grew rat organs.

The stem cells used are called pluripotent stem cells and are adult stem cells that can be taken from tissue and grow in any kind of cell within the body. These cells were injected into the mice embryos that were unable to grow a pancreas, an organ responsible for producing insulin. When the mice grew into adulthood, they displayed no signs of diabetes and the rat stem cells had developed into a pancreas.

The ultimate goal of the researchers is to take this technique and grow human organs inside pigs. If this technique works it would be able to minimize the risk of human transplant rejection because the organs could be grown using the patient's own stem cells. This technique would also work to create a plentiful supply or organs for transplantation.

Using the mice as an example, human stem cells could be used to create a new pancreas to be transplanted into diabetic patients.

Nakauchi is currently looking for approval to use human stem cells for further research. This is the first time that blastocyst complementation has been shown to work, so the idea of growing human organs is promising. Ethically, researchers are not able to make an human embryo organ deficient, so in order to test the idea of growing organs, another animal needed to be used.

http://www.newscientist.com/article/mg21028175.500-botclouds-a-cyberattackers-dream.html

Botclouds: a cyberattacker's dream

*** 21 June 2011 by Jacob Aron**

Editorial: "The cloud that hangs over cloud computing"

OFFLOADING your software and data to a cloud computing service has never been easier.

Apple last week became the latest tech company - after Google and Amazon - to offer cheap online storage, with its new iCloud service allowing users to access music, documents and other files from any Apple device. But cloud services could also be used to launch attacks, send spam and commit fraud.

"Right now it's just a few attacks, most aren't well publicised and a lot can go undetected," says Kassidy Clark of the Delft University of Technology in the Netherlands. "As long as cloud service providers are not taking proactive steps to prevent these things, I think this trend will increase."

As well as basic online storage, firms such as Amazon, which provides the largest cloud service, also offer virtual computing. This allows people to rent as many "virtual computers" as they need.

Now Clark and colleagues have investigated how the cloud could be used to build a botnet, a network of infected computers under an attacker's control. Traditional botnets are built over time by taking control of ordinary people's computers without their knowledge, but a cloud botnet - or botcloud - can be put together in a couple of minutes just by purchasing space in the cloud with stolen credit card details. "It makes deployment much faster," says Clark, who presented his findings at the CLOSER cloud computing conference in Noordwijkerhout, the Netherlands, last month. "You don't have to wait months for millions of machines around the world to get infected."

To find out just how easy it is to construct a botcloud, Clark and colleagues hired 20 virtual computers from a leading cloud service provider for around €100 and used them to carry out attacks on their own web server. They first attempted a distributed denial of service (DDoS) attack, which floods a target with massive amounts of traffic. The botcloud pumped out 20,000 page requests per second and brought the server down in just 10 seconds.

Clark also built a larger botcloud and used it to simulate "click fraud" - clicking links in pay-per-click adverts in order to generate fraudulent revenue. Advertising companies normally stop this by tracking the internet protocol (IP) address of each individual computer and blocking one if it clicks a link too many times. The researchers circumvented this defence by setting up a botcloud of 1000 virtual computers, each with its own address. Neither botcloud attack was detected or shut down by the cloud provider.

So are botclouds being used? There were certainly rumours that the recent attack on Sony's PlayStation Network was carried out via Amazon servers rented using stolen credit cards, but these have not been substantiated. "We have seen spam coming from some of these environments, but not on a massive scale," says Paul Wood, a senior analyst at Symantec.cloud, which provides cloud-based security services. He says that it is even possible for a virtual computer in the cloud to become infected by an ordinary botnet, because cloud users don't normally run anti-virus software.

Thomas Roth, a security researcher in Cologne, Germany, who recently showed how to use Amazon's servers to crack Wi-Fi passwords, agrees the lack of anti-virus protection in the cloud is a problem. "I think that Amazon should provide infrastructure for doing vulnerability assessments and virus scans," he says.

"Amazon Web Services employs a number of mitigation techniques, both manual and automated, to prevent the misuse of the services," Amazon told New Scientist. "We have automatic systems in place that detect and block many attacks before they leave our infrastructure."

But Wood warns that attacks from the cloud could easily take off in countries with more lax web policing. "It's only a matter of time before a Russian or Chinese equivalent of Amazon offers similar services," agrees Clark. "You put malicious or illegal software there, it doesn't matter, they will never take you offline."

The cloud that hangs over cloud computing

AT THE launch of Apple's iCloud service last week, Steve Jobs promised to "demote" those workhorses of modern life, the PC and Mac. That's because cloud computing can liberate those devices, along with smartphones and others, offloading the burden of number-crunching and storage onto distant data centres.

But the cloud is vulnerable. Within its nebulous frame, it appears to be relatively easy to mount cyberattacks and commit fraud.

The cloud is a welcome development, but if these loopholes exist, what others might there be? If cloud providers like Google, Amazon and Apple want us to trust them with our digital worldly possessions they must monitor more closely what is happening on their servers; measures such as closing criminals' accounts after the event are not enough.

Greater collaboration with credit card providers and other companies targeted by cloud criminals could help ensure a safe and reliable service. Cloud computing will provide great advantages, potentially so great that we must not allow it to be subverted.

http://www.eurekalert.org/pub_releases/2011-06/uoc - usa062011.php

UCLA scientists accurately predict age with saliva sample

Patented test could offer new tool for crime-scene investigation, personalized medicine

Self-conscious about your age? Careful where you spit. UCLA geneticists now can use saliva to reveal how old you are.

The June 22 advance online edition of the Public Library of Science (PLoS) ONE publishes the findings, which offer a myriad of potential applications. A newly patented test based on the research, for example, could offer crime-scene investigators a new forensic tool for pinpointing a suspect's age.

"Our approach supplies one answer to the enduring quest for reliable markers of aging," said principal investigator Dr. Eric Vilain, a professor of human genetics, pediatrics and urology at the David Geffen School of Medicine at UCLA. "With just a saliva sample, we can accurately predict a person's age without knowing anything else about them." Vilain and his colleagues looked at a process called methylation – a chemical modification of one of the four building blocks that make up our DNA.

"While genes partly shape how our body ages, environmental influences also can change our DNA as we age," explained Vilain. "Methylation patterns shift as we grow older and contribute to aging-related disease."

Using saliva samples contributed by 34 pairs of identical male twins ages 21 to 55, UCLA researchers scoured the men's genomes and identified 88 sites on the DNA that strongly correlated methylation to age. They replicated their findings in a general population of 31 men and 29 women aged 18 to 70.

Next, the scientists built a predictive model using two of the three genes with the strongest age-related linkage to methylation. When they plugged in the data from the twins' and the other group's saliva samples, they were able to correctly predict a person's age within five years – an unprecedented level of accuracy.

"Methylation's relationship with age is so strong that we can identify how old someone is by examining just two of the 3 billion building blocks that make up our genome," said first author Sven Bocklandt, a former UCLA geneticist now at Bioline. Vilain and his team envision the test becoming a forensic tool in crime-scene investigations. By analyzing the traces of saliva left in a tooth bite or on a coffee cup, lab experts could narrow the age of a criminal suspect to a five-year range.

In a minority of the population, methylation does not correlate with chronological age. Using this data, scientists may one day be able to calculate a person's "bio-age" - the measurement of a person's biological age versus their chronological age.

Physicians could evaluate the risk of age-related diseases in routine medical screenings and tailor interventions based on the patient's bio-age rather than their chronological age. Instead of requiring everyone to undergo a colonoscopy at age 50, for example, physicians would recommend preventive tests according to a person's bio-age.

"Doctors could predict your medical risk for a particular disease and customize treatment based on your DNA's true biological age, as opposed to how old you are," noted Vilain. "By eliminating costly and unnecessary tests, we could target those patients who really need them."

The UCLA team is currently exploring whether people with lower bio-age live longer and suffer less disease. They also are examining if the reverse is true - whether higher bio-age is linked to a greater rate of disease and early death.

The study was internally funded by UCLA. Vilain's coauthors included Bocklandt, Wen Lin, Mary Sehl, Francisco Sáncheza, Janet Sinsheimer and Steve Horvath, all of UCLA.

http://www.eurekalert.org/pub_releases/2011-06/uoc - btc062211.php

Breaking the chain: 'Molecular cap' blocks processes that lead to Alzheimer's, HIV

A new advance by UCLA biochemists has brought scientists one step closer to developing treatments that could delay the onset of Alzheimer's disease and prevent the sexual transmission of HIV.

The researchers report that they have designed molecular inhibitors that target specific proteins associated with Alzheimer's disease and HIV to prevent them from forming amyloid fibers, the elongated chains of interlocking proteins that play a key role in more than two dozen degenerative and often fatal diseases.

"By studying the structures of two key proteins that form amyloids, we were able to identify the small chain of amino acids responsible for amyloid fiber formation and engineer a 'molecular cap' that attaches to the end of the fibers to inhibit their growth," said research leader David Eisenberg, director of the UCLA–Department of Energy Institute of Genomics and Proteomics and a Howard Hughes Medical Institute investigator.

"This research is an important first step toward the development of structure-based drugs designed against amyloid diseases," said Eisenberg, who is a UCLA professor of chemistry, biochemistry and biological chemistry and a member of the California NanoSystems Institute at UCLA. "Our results have opened up an avenue so that universities and industry can start creating therapeutics that could not have been produced 10 years ago." The study was published online June 15 in the journal Nature and will be available in an upcoming print edition.

Toward delaying Alzheimer's disease

Amyloid fibers are elongated, water-tight structures formed from two linked protein sheets. Proteins from each sheet contribute side chains, causing them to interlock like the teeth of a zipper, Eisenberg said.

The fibers are found not only Alzheimer's disease but in a variety of conditions, including Lou Gehrig's disease, Parkinson's disease, type II diabetes and a family of disorders related to mad cow disease, among others. In Alzheimer's and other neurodegenerative diseases, the tau protein forms amyloid fibers inside brain cells, destroying them through a mechanism that is still being investigated.

Though many serious diseases are characterized by amyloid fibers, Alzheimer's is the most prevalent, Eisenberg said. Today there are 5 million patients in the U.S. who suffer from Alzheimer's, with 500,000 new cases every year. Alzheimer's health care cost this year alone have been estimated at $178 billion, including the value of unpaid care for Alzheimer's patients provided by nearly 10 million family members and friends.

"By the year 2050, it is projected that there will be 19 million Alzheimer's patients," Eisenberg said. "The care of so many patients with this debilitating illness could be a substantial fraction of the gross domestic product of the United States."

Eisenberg and his research team found that of the entire tau protein, a small chain of just six amino acids - abbreviated VQIVYK - was responsible for the formation of amyloid fibers. By studying the structure of the fibers using microcrystallography, a method developed at UCLA for this research, the team was able to use the fibers as a template to design an inhibitor that could 'cap' the fiber and stop it from growing.

The results were dramatic. The introduction of the inhibitor into a tau protein solution completely prevented amyloid fiber formation, validating the idea that the structure-based design of therapeutics for amyloid diseases is a plausible option.

Despite this success, there is still a long road ahead before a viable therapeutic can be developed to combat the onset of Alzheimer's in human patients, Eisenberg said. The inhibitor, a chain of amino acids, is far too large to penetrate deep into the brain where the tau proteins form amyloid fibers.

"This research is an important step toward identifying smaller molecules that can be utilized to develop a therapeutic," Eisenberg said. "Our goal is to be able to delay the onset of Alzheimer's disease."

Preventing the transmission of HIV

Unlike the tau protein, the SEVI (semen-derived enhancer of viral infection) protein is a far more accessible target for a molecular blocker because it builds amyloid fibers in a vaginal environment, a key process in the sexual transmission of HIV, Eisenberg said.

"The presence of SEVI makes the rate of HIV infection through sexual transmission up to 100,000 times more likely," he said. "By blocking SEVI, we have a method for inhibiting the sexual transmission of HIV."

Though the tau and SEVI proteins have different structures and unrelated functions, they both form amyloid fibers with similar morphology, making it possible to design two separate inhibitors using the same process, according to Eisenberg. The SEVI blocker proved to be equally effective in preventing fiber growth, bolstering the idea that blockers can be designed for other diseases associated with amyloid fibers as well.

"Though many tests remain, it seems we could be on the way to developing a therapeutic," Eisenberg said. "Our hope is that we could make a blocker that could be applied with a vaginal gel or spray that would help to prevent HIV infection."

The tau and SEVI protein inhibitors were designed using synthetic amino acids, similar to the standard protein building blocks of the human body. But these synthetic amino acids were flipped, as if viewed in a mirror, or had added side chains not normally found in nature. Enzymes in the human body that are programmed to break apart protein-like chains are, in principle, unable to recognize the non-natural amino acids, keeping the blockers safe to latch on to the target proteins.

This research was federally funded by the National Institutes of Health, the National Science Foundation and the U.S. Department of Energy, as well as by the Howard Hughes Medical Institute and the Joint Center for Translational Medicine. Other co-authors of this study included UCLA postdoctoral scholars Stuart Sievers and Lin Jiang; UCLA graduate students Howard Chang and Anni Zhao; John Karanicolas, an assistant professor at the University of Kansas; Jason Stevens, an undergraduate at the University of Kansas; David Baker, a professor at the University of Washington; and professor Jan Münch and researcher Onofrio Zirafi, of the University of Ulm in Germany. This study was published June 14 in PLoS Biology, an online journal of the Public Library of Science.

Small molecules, big job

A second research team also led by Eisenberg recently announced that it had identified four small molecules that bind to amyloid fibers, including a promising candidate called 'orange-G' that wedges into the zipper-like fiber and may be able to break it apart.

"These are the first small molecules visualized as they bind to amyloid-like fibers," Eisenberg said. "These small molecules are less likely to be broken up in the body and can potentially be modified to force apart amyloid fibers or serve as diagnostic tools to identify infected areas of the body."

Eisenberg and his research team found that orange-G was uniquely able to pierce the impenetrable "steric zippers" that seal the water-tight amyloid fibers of the amyloid-beta protein that is responsible for forming senile plaques in Alzheimer's disease.

"In 10 years we have gotten to the point where we are starting to understand the structural biology of amyloid fibers and how to inhibit them and how to interfere with them," Eisenberg said. "The next step is to make practical molecules that inhibit and break amyloid fibers - that is the ultimate goal."

Co-authors on this UCLA research included Kym Faull, professor of psychiatry and biobehavioral sciences; Jorge Barrio, professor of molecular and medical pharmacology; researchers Michael Sawaya and Jie Liu; postdoctoral scholars Meytal Landau, Lin Jiang and Stuart Sievers; and graduate student Arthur Laganowsky.

http://www.eurekalert.org/pub_releases/2011-06/uoca-sey062211.php

Strongest evidence yet indicates icy Saturn moon hiding saltwater ocean

Samples of icy spray shooting from Saturn's moon Enceladus collected during Cassini spacecraft flybys show the strongest evidence yet for the existence of a large-scale, subterranean saltwater ocean, says a new international study led by the University of Heidelberg and involving the University of Colorado Boulder.

The new discovery was made during the Cassini-Huygens mission to Saturn, a collaboration of NASA, the European Space Agency and the Italian Space Agency. Launched in 1997, the mission spacecraft arrived at the Saturn system in 2004 and has been touring the giant ringed planet and its vast moon system ever since.

The plumes shooting water vapor and tiny grains of ice into space were originally discovered emanating from Enceladus - one of 19 known moons of Saturn - by the Cassini spacecraft in 2005. The plumes were originating from the so-called "tiger stripe" surface fractures at the moon's south pole and apparently have created the material for the faint E Ring that traces the orbit of Enceladus around Saturn.

During three of Cassini's passes through the plume in 2008 and 2009, the Cosmic Dust Analyser, or CDA, on board measured the composition of freshly ejected plume grains. The icy particles hit the detector's target at speeds of up to 11 miles per second, instantly vaporizing them. The CDA separated the constituents of the resulting vapor clouds, allowing scientists to analyze them. The study shows the ice grains found further out from Enceladus are relatively small and mostly ice-poor, closely matching the composition of the E Ring. Closer to the moon, however, the Cassini observations indicate that relatively large, salt-rich grains dominate.

"There currently is no plausible way to produce a steady outflow of salt-rich grains from solid ice across all the tiger stripes other than the salt water under Enceladus' icy surface," said Frank Postberg of the University of Germany, lead author of a study being published in Nature on June 23. Other co-authors include Jürgen Schmidt from the University of Potsdam, Jonathan Hillier from Open University headquartered in Milton Keynes, England, and Ralf Srama from the University of Stuttgart.

"The study indicates that 'salt-poor' particles are being ejected from the underground ocean through cracks in the moon at a much higher speed than the larger, salt-rich particles," said CU-Boulder faculty member and study co-author Sascha Kempf of the Laboratory for Atmospheric and Space Physics.

"The E Ring is made up predominately of such salt-poor grains, although we discovered that 99 percent of the mass of the particles ejected by the plumes was made up of salt-rich grains, which was an unexpected finding," said Kempf. "Since the salt-rich particles were ejected at a lower speed than the salt-poor particles, they fell back onto the moon's icy surface rather than making it to the E Ring."

According to the researchers, the salt-rich particles have an "ocean-like" composition that indicates most, if not all, of the expelled ice comes from the evaporation of liquid salt water rather than from the icy surface of the moon. When salt water freezes slowly the salt is "squeezed out," leaving pure water ice behind. If the plumes were coming from the surface ice, there should be very little salt in them, which was not the case, according to the research team.

The researchers believe that perhaps 50 miles beneath the surface crust of Enceladus a layer of water exists between the rocky core and the icy mantle that is kept in a liquid state by gravitationally driven tidal forces created by Saturn and several neighboring moons, as well as by heat generated by radioactive decay. According to the scientists, roughly 440 pounds of water vapor is lost every second from the plumes, along with smaller amounts of ice grains. Calculations show the liquid ocean must have a sizable evaporating surface or it would easily freeze over, halting the formation of the plumes. "This study implies that nearly all of the matter in the Enceladus plumes originates from a saltwater ocean that has a very large evaporating surface," said Kempf.

Salt in the rock dissolves into the water, which accumulates in a liquid ocean beneath the icy crust, according to the Nature authors. When the outermost layer of the Enceladus crust cracks open, the reservoir is exposed to space. The drop in pressure causes the liquid to evaporate into a vapor, with some of it "flash-freezing" into salty ice grains, which subsequently creates the plumes, the science team believes.

"Enceladus is a tiny, icy moon located in a region of the outer Solar System where no liquid water was expected to exist because of its large distance from the sun," said Nicolas Altobelli, ESA's project scientist for the Cassini-Huygens mission. "This finding is therefore a crucial new piece of evidence showing that environmental conditions favorable to the emergence of life may be sustainable on icy bodies orbiting gas giant planets."

The Huygens probe was released from the main spacecraft and parachuted through the atmosphere to the surface of Saturn's largest moon, Titan, in 2005. The Cassini spacecraft is carrying 12 science instruments, including a $12.5 million CU-Boulder ultraviolet imaging spectrograph designed and built by a LASP team led by Professor Larry Esposito.

http://news.discovery.com/history/earliest-american-art-mammoth-110622.html

Earliest American Art: Mammoth on Mammoth

The first known art object from America is a 13,000-year-old carving of a mammoth on mammoth bone.

By Jennifer Viegas

The first known depiction of an animal from the Americas is an image of a mammoth engraved on a mammoth bone, suggests a paper accepted for publication in the Journal of Archaeological Science.

Researchers believe the object, found in Florida, dates to at least 13,000 years ago when most large Pleistocene animals went extinct in the eastern United States. The artifact may even be up to 20,000 years old.

"The engraving was done by a group of people that we would refer to as Paleoindian or Paleoamericans," co-author Jeff Speakman, head of technical studies at the Smithsonian Museum Conservation Institute, told Discovery News. The word "Paleoamerican" does not necessarily point to a cultural group, he added, but instead is a "general term that refers to the earliest inhabitants of the Americas."

The animal engraving has made headlines since fossil hunter James Kennedy first collected it from a location in North Vero Beach sometime in 2006 or 2007. In February of 2009, Kennedy discovered the engraving while cleaning the incised bone.

To determine the piece's authenticity, Speakman and his colleagues recently put it through a barrage of scientific tests. Scanning electron microscopy, forensic analysis, high powered X-rays and other tests all suggest the mammoth engraving is not a forgery.

Compared to the rough crosshatched, checkered lines associated with Paleoindian art from early sites such as Gault in Texas, this animal image from Florida looks quite sophisticated. Speakman, however, points out that 120,000-year-old decorative ornaments have been found in Africa and the Near East. Venus figurines date to at least 35,000 years ago, and paint materials in the human archaeological record could date to more than 300,000 years ago.

This sketch of a mammoth inscribed on mammoth bone may be the oldest known American art. **Chip Clark, Smithsonian National Museum of Natural History**

Twenty thousand years ago is therefore a drop in the proverbial art bucket from a global perspective, but it is extremely old for the Americas. The researchers indicate the object may strengthen the controversial theory that people associated with the Solutrean culture of Europe migrated to North America via the North Atlantic Ice Sheet. In other words, some of America's first inhabitants could have been Europeans that settled in what is now Florida and at other locations. "The hypothesis rests upon similarities between Solutrean and Clovis tool technologies that have no known counterparts in Eastern Asia, Siberia, or Beringia - areas that early people are known to have migrated through," Speakman explained.

Two of the strongest arguments against this "Solutrean hypothesis" have been a lack of art and other archaeological finds from the proposed period in North America. The mammoth engraving could address the art issue. As to the second argument, Speakman said that over the past decade, "numerous archaeological sites in the eastern U.S. have been identified" dating to 20,000 plus years ago, possibly even pre-dating the Clovis culture.

In terms of the engraving's subject, "Mammoths, mastodons and dozens of other now extinct species were present and plentiful in Florida 13,000 years ago," he said. "Certainly these animals were hunted by Paleoindians. People tend to focus on the significance of the larger mammals, but that's kind of what preserves best, other than stone tools, in many parts of North America."

Bruce Bradley, an associate professor of experimental archaeology at the University of Exeter, told Discovery News that he believes the new paper "makes a compelling case for the authenticity of the engraving."

"As to who and when the bone was inscribed," Bradley continued, "the only issue is really whether it was done on fresh or mineralized bone. Since there is no means of directly dating the bone, the probability is that it is at least as old as the extinction of the animal species it came from."

Bradley agrees that the image likely "would have been made by Clovis contemporaries or earlier."

In the future, a full excavation of the North Vero Beach site could be possible, allowing researchers to learn more about what could be the first human inhabitants of the Americas.

http://www.newscientist.com/article/mg21028185.300-red-wines-heart-health-chemical-unlocked-at-last.html

Red wine's heart health chemical unlocked at last

FANCY receiving the heart protecting abilities of red wine without having to drink a glass every day?

Soon you may be able to, thanks to the synthesis of chemicals derived from resveratrol, the molecule believed to give wine its protective powers. The chemicals have the potential to fight many diseases, including cancer.

Plants make a huge variety of chemicals, called polyphenols, from resveratrol to protect themselves against invaders, particularly fungi. But they only make tiny amounts of each chemical, making it extremely difficult for scientists to isolate and utilise them. The unstable nature of resveratrol has also hindered attempts at building new compounds from the chemical itself.

Scott Snyder at Columbia University in New York and his team have found a way around this: building polyphenols from compounds that resemble, but are subtly different to, resveratrol. These differences make the

process much easier. Using these alternative starting materials, they have made dozens of natural polyphenols, including vaticanol C, which is known to kill cancer cells (Nature, DOI: 10.1038/nature10197).

"It's like a recipe book for the whole resveratrol family," says Snyder. "We've opened up a whole casket of nature's goodies."

http://www.bbc.co.uk/news/uk-13859049

Nazis 'planned gas attack' during UK war invasion

Germany planned to use chemical and biological attacks during a wartime invasion of Britain, according to documents from the National Archives.

Aircraft adapted to spray gas or foot-and-mouth disease, and even anthrax shells, were seen as possibilities.

The Nazis were "rapidly preparing" by March 1941 and would not hesitate to attack, say the papers.

Gas attacks on troops were viewed as most likely, but attacks on the public were also predicted.

The newly-released documents - from the Ministry of Home Security and other bodies - say the aim would be to cause panic and start an evacuation that would block roads and prevent defending forces reaching the coast. Intelligence also revealed that the Germans were planning to falsely accuse Britain of using gas in order to justify its own attacks.

The documents detail the ominous movement of large amounts of chemicals from factories to "areas occupied by troops likely to take part in an invasion". It also lists evidence from various secret witnesses and "observers". They include a Swedish army officer who saw gas canisters ready to be loaded at German air bases, and a prisoner of war who "had volunteered information that his squadron had carried out tests near Vienna of gas spray".

'Cloud attack'

Intelligence chiefs were also mindful of a possible seaborne gas cloud threatening the country.

A document from August 1940 states that such an attack, using phosgene or chlorine, although "operationally difficult", could not be disregarded and could threaten an area up to 10 miles in width.

"Whenever an on-shore wind at night is forecast, a warning will be issued to all troops within five miles of the coast", reads the document. The "warning zone" stretched from the east coast of Scotland all the way round the south coast of England to the Bristol Channel. Alerting the public to a potential gas cloud "would be undesirable and should not be done" adds the secret memo.

'Biological warfare'

The possibility of anthrax and biological warfare is also briefly mentioned in the newly released papers, which date from 1939 to 1941. "The germs of foot-and-mouth disease are reputed to have been sprayed, experimentally, from aircraft," says one report. It continues: "Tests are said to have been carried out with shells infected with Anthrax... they are said to result in 95% mortality, death occurring in 10-12 days."

The archives also reveal that experts expected an initial surprise bombardment of up to 2,500 airborne gas sorties, delivered by bomb or spray, during the first few days of a Nazi invasion.

Boy in gas mask The British had more gas masks than the Germans say the documents

One document states: "The German High Command may think that, if gas is used on the first day... the resulting casualties and panic will enable them to get a firm foothold on these shores."

The planned German invasion, codenamed Operation Sea Lion, was postponed in September 1940 after the Luftwaffe's defeat by the RAF in the Battle of Britain. Air superiority was considered vital for the Germans' amphibious assault on Britain. Hitler eventually cancelled the invasion plans in January 1941.

http://news.nationalgeographic.com/news/2011/06/110623-iceman-mummy-otzi-meal-goat-stomach-science/

Iceman's Stomach Sampled - Filled With Goat Meat

Missing until 2009, mummy's stomach found to contain lumps of last meal.

Ker Than for National Geographic News

Hours before he died, "Ötzi" the Iceman gorged on the fatty meat of a wild goat, according to a new analysis of the famous mummy's stomach contents. The frozen body of the Copper Age hunter was discovered in 1991 in the Alps of northern Italy, where he died some 5,000 years ago. The circumstances surrounding Ötzi's death are not fully known, but the most popular theory - based in part on the discovery of an arrowhead in his back - is that he was murdered by other hunters while fleeing through the mountains.

Scientists previously analyzed the contents of Ötzi's lower intestine and determined that he ate a meal of grains along with possibly cooked red deer and goat meat up to 30 hours before his death. But attempts using an endoscopic tool to sample Ötzi's stomach were unsuccessful.

The reason for the failure became clear in 2009, when scientists studying CAT scans of Ötzi discovered that the Iceman's stomach had shifted upward after death, to where the lower part of his lungs would normally be.

"Why it moved upward, we don't know," said Frank Maixner, a microbiologist at the Institute for Mummies and the Iceman in Bolzano, Italy, who was involved in the new investigation.

Iceman's Last Supper

The team found the stomach by examining other associated organs, which had maintained their relative positions to one another when they shifted. The team found gallstones in the gall bladder, for instance, and from there could identify the stomach.

As a result of the natural mummification process, Ötzi's stomach had shrunk considerably. But the researchers were able to get sample of its contents, which - like the intestines - contained evidence of meat and wheat grains. What's more, the state of the partially digested food suggests the Iceman ate a substantial meal less than two hours before his death. "The stomach content is yellowish to brownish colored and mushy, with some bigger pieces of meat and grain," Maixner said.

DNA analysis of the meat showed that it came from an ibex, a wild goat species whose males have large, backward-curving horns. Ibex would have been much more common in Ötzi's day and would have been a good source of meat for hunters. The animals are usually skittish around humans and will flee at the first opportunity, but a skilled hunter can creep up on one under the right circumstances.

For example, "during certain periods when the males are fighting each other, you can get as close as 20 to 50 meters [65 to 160 feet]," Maixner said. According to past studies, such a distance would have been just within range of the bow and arrows that were found with Ötzi, he added.

It's unclear if the ibex meat was cooked, but it's possible that it was, especially since ash particles associated with other meals, possibly from cooking fires, were found in Ötzi's lower intestine, Maixner said.

Still, strands of animal hair and fly parts also found in Ötzi's stomach suggest the Iceman wasn't overly concerned with cleaning the meat before he ate it. "It wasn't the most hygienic of meals," Maixner said.

The new Iceman research was presented at the 7th World Congress on Mummy Studies in San Diego, California, earlier this month.

http://www.eurekalert.org/pub_releases/2011-06/uob-atf062211.php

A thermometer for dinosaurs

Researchers identify body temperature of these long-extinct giant saurians

Small heads, large bodies, and a slow metabolism - these are the characteristics that make us think of dinosaurs as dull, lethargic and cold-blooded giants. However, this image seems to be deceiving. These giant saurians that have been extinct for 65 million years may have been high-performance models of evolution. In cooperation with colleagues from the US, researchers from the University of Bonn have just determined that the body temperature of some large herbivorous dinosaurs was between 36 and 38 degrees Celsius.

"Originally, dinosaurs were considered to have been cold-blooded animals because they are reptiles, just like salamanders or crocodiles," explained Dr. Thomas Tütken, a biochemist from the Steinmann Institut at the University of Bonn. Their body temperature depends on the ambient temperature. "This is why after a cold night, the mobility of today's reptiles is very limited, and so is their activity," he added. Warm-blooded animals such as mammals or birds, in contrast, are able to keep their body temperature constant by combusting food. Did dinosaurs also have such an active "heating system"? A homeothermic organism could be compared to a race car that, while it is a high-performance system, uses a lot of "fuel" too. In contrast, a cold-blooded animal will start up more slowly during operation in the cold, but it will also need only about one tenth of the energy a warm-blooded animal will require.

With their colleagues in the U.S., the Bonn researchers developed a method that allows determining the absolute body temperature of dinosaurs with the accuracy of a thermometer by analyzing their dental enamel. "The original chemical composition of their dental enamel has been much better preserved than that of dinosaur bones," said Tütken. Enamel contains a certain percentage of carbonate, a carbon/oxygen compound. Both elements have a heavier and a lighter variant called isotopes. "The mineral formation temperature determines how frequently the two heavy carbon isotopes (13C) and the two heavy oxygen isotopes (18O) will enter a 13C-18O bond within a dinosaur's tooth," explained the geochemist. The warmer it was while the dental enamel was formed, the more infrequently will the two heavy isotopes enter such a bond. "We used this correlation as a thermometer that allowed us to determine the body temperature accurately to within two degrees," explained Tütken.

Using this chemical thermometer, the scientists analyzed teeth from different dinosaurs - on the Camarasaurus, which reached a length of up to 20 meters and a weight of up to 15 tonnes, and the Brachiosaurus, which even topped out at 23 meters and about 40 tonnes. These two giant saurians from the sub-order of herbivorous sauropods lived during the Jurassic Era, about 150 million years ago. "We analyzed

thirteen teeth from a Camarasaurus, and three from a Brachiosaurus," the Bonn researcher reported. However, only seven of them were preserved well enough for the analyses to be deemed conclusive.

The heavier carbon and oxygen isotope variants occur in enamel only in minute concentrations – on average, only in 45 ppm (parts per million.) "Consequently, we needed just a little more than a tenth of a gram of tooth enamel, which can be much thinner than a millimeter on dinosaur teeth," explained Tütken. Dinosaurs actually had bigger teeth measuring up to several centimeters. "Since they did not chew, but only cut off their food with their teeth, their teeth were replaced permanently - often as frequently as once a month, as still happens with some reptiles today," he added.

Dinosaurs had a body temperature of 36 to 38 degrees Celsius

For the Camarasaurus from the U.S., dental enamel analyses resulted in a body temperature of about 36 degrees Celsius, and for the Brachiosaurus from Tanzania, in approximately 38 degrees. "Our method enabled us to determine the body temperature of giant saurians for the first time," declared the researcher from Bonn. In a previous study, the scientists had already successfully applied the chemical thermometer to fossil teeth from 30,000 year-old mammoths. Tütken added, "Our dinosaur tooth analyses have now expanded the time scale to 150 million years."

The question whether dinosaurs were warm-blooded has not been finally resolved yet. "Our data provides clear indications that their body temperature was clearly higher and more stable than ambient temperatures," said Tütken. This could, however, also be a function of the giant saurians' sheer size since a large body mass is also very good at keeping its temperature constant. The scientists now want to study the body temperatures of smaller dinosaurs because they were not able to store heat as well due to the fact that their body surface was large compared to their body volume. If they showed similarly high body temperatures as warm-blooded animals, it would be a clear indication that they were also warm-blooded.

Publication: Robert A. Eagle, Thomas Tütken, Taylor S. Martin, Aradhna K. Tripati, Henry C. Fricke, Melissa Connely, Richard L. Cifelli and John M. Eiler: "Dinosaur Body Temperatures Determined from Isotopic (13C-18O) Ordering in Fossil Biominerals," Science, 23 June 2011 (10.1126/science.1206196)

http://www.eurekalert.org/pub_releases/2011-06/vph-ctf062211.php

Cautionary tale for people with diabetes: Dog consumed part of a sleeping patient's toe

Valley Presbyterian specialist co-authors case study to highlight the danger of pets in the presence of open wounds

Van Nuys, CA – In a case study that illustrates the need for people with diabetes to be cautious of foot injuries and to protect themselves from pets, a woman with numbness in her feet caused by diabetic neuropathy slept through a traumatic episode in which her Jack Russell terrier chewed off part of her slightly infected big toe, according to an article published in this month's issue of the Journal of the American Podiatric Medical Association. The patient's wound required surgery, and it ultimately led the amputation of her leg, leaving her a double amputee.

The case study, co-authored by Valley Presbyterian Hospital specialist Lee C. Rogers, D.P.M., is only the second of its kind to be published in the medical literature, although more cases like it have been reported in the media. This case highlights the need for diabetic patients with neuropathy to avoid having their feet or wounds exposed when sleeping with their pets.

"Pets have a tendency to lick wounds, and that simple lick can turn into a bite, if there is no response from the owner. There have also been reports of dogs' saliva infecting diabetic patients with the antibiotic-resistant Superbug, MRSA, which can be deadly," Dr. Rogers said. "This case illustrates the perils of pet ownership in diabetic patients who have numbness in their hands or feet caused by neuropathy."

The Centers for Disease Control estimate diabetes affects 25.8 million Americans - or 8.3 percent of the population - and report that it is the leading cause of non-traumatic amputations. In this case, the 48-year-old woman didn't feel any pain and only realized part of her toe was missing when she awakened in the morning and found blood in her bed and on the floor.

She was sleeping with her 2-year-old Jack Russell terrier and realized it must have chewed off part of the toe during the night because the dog had blood in its facial fur. Doctors amputated part of her toe and, later, the lower portion of her leg because she developed other infections and neuropathic ulcerations, skin lesions that are common in diabetics who suffer from numbness. "People with diabetes and neuropathy must take special precautions to protect their feet from infections to avoid amputations and other complications," said Dr. Rogers.

Dr. Rogers is the associate director of Valley Presbyterian Hospital's Amputation Prevention Center, an integrated limb-preservation center that is one of the nation's only facilities of its kind. Since its January 2010 opening, the Amputation Prevention Center's specialized multidisciplinary team of highly skilled professionals has treated more than 350 patients with leading-edge technology and achieved a limb salvage rate of 96 percent.

"With its exemplary record of success, the Amputation Prevention Center is truly a community asset and an extraordinary benefit to patients in danger of losing a limb," said Gustavo Valdespino, President and CEO of Valley Presbyterian Hospital. "The Center is leading the way in patient care and treatment with its cutting-edge technology and innovative team approach pairing podiatrists with vascular surgeons."

George Andros, M.D., the Center's Medical Director, notes the center recorded an average wound-healing rate of 52 days – less than half the national average of 120 days, in its first year.

"At Valley Presbyterian Hospital, we are proud to be part of this pioneering effort to employ new technology to bring expertise to patients wherever they may be," he said. "The Amputation Prevention Center is on the leading edge of advancing the pace of medicine and improving the care of patients."

http://www.eurekalert.org/pub_releases/2011-06/hms-nii062311.php

New insights into origin of deadly cancer

Barrett's esophagus, often a precursor to esophageal cancer, results from residual, embryonic cells

Boston, MA - Researchers have discovered a new mechanism for the origin of Barrett's esophagus, an intestine-like growth in the esophagus that is triggered by chronic acid reflux and often progresses to esophageal cancer. Studying mice, the researchers found that Barrett's esophagus arises not from mutant cells in the esophagus but rather a small group of previously overlooked cells present in all adults that can rapidly expand to cancer precursors when the normal esophagus is damaged by acid. Decades of cancer research tells us that most of the common cancers begin with genetic changes that occur over a period of 15 to 20 years, in some cases leading to aggressive cancers. However, for a subset of cancers that appear to be linked to chronic inflammation, this model might not hold. This research will be published online in the June 24th issue of Cell.

Barrett's esophagus, which was first described by the Australian surgeon Norman Barrett in 1950, affects two to four million Americans. In this condition, tissue forms in the esophagus that resembles the intestinal tissue normally located much farther down the digestive tract. As a result, a person's chances of developing a deadly esophageal adenocarcinoma increase by 50- to 150-fold. Late stage treatment is largely palliative, so it is important to understand how acid reflux triggers it in the first place.

Research from the laboratory of Frank McKeon, Harvard Medical School professor of cell biology, together with Wa Xian, a postdoctoral researcher at Brigham and Women's Hospital and the Institute of Medical Biology, Singapore, along with an international consortium including Christopher Crum, director of Women's and Perinatal Pathology at Brigham and Women's Hospital, has shown that Barrett's esophagus originates from a minor population of non-esophageal cells left over from early development.

For the past decade, McKeon and his laboratory have been using mouse models to investigate the role of p63, a gene involved in the self-renewal of epithelial stem cells including those of the esophagus. McKeon joined forces two years ago with Wa Xian, an expert in signal transduction in cancer cells, to tackle the vexing problem of the origin of Barrett's esophagus.

At that time, the dominant hypothesis for Barrett's was that acid reflux triggers the esophageal stem cells to make intestine cells rather than normal esophageal tissue. However, McKeon and Xian felt the support for this concept was weak. Taking a different track, they studied a mouse mutant lacking the p63 gene and mimicked the symptoms of acid reflux. As a result, the entire esophagus was covered with a Barrett's-like tissue that proved to be a near exact match with human Barrett's at the gene expression level.

The researchers were particularly surprised by the sheer speed with which this Barrett's esophagus appeared in the mice. "From the speed alone we knew we were dealing with something different here," said Xia Wang, postdoctoral fellow at Harvard Medical School and co-first author of this work.

Yusuke Yamamoto, a postdoctoral fellow at the Genome Institute of Singapore and also co-first author, added that, "we just had to track the origins of the Barrett's cells back through embryogenesis using our markers from extensive bioinformatics."

In essence, the investigators tracked the precancerous growth to a discrete group of leftover embryonic cells wedged between the junction of the esophagus and the stomach - precisely where endoscopists have argued Barrett's esophagus begins. As predicted by the mouse studies, the researchers identified a group of embryonic cells exactly at the junction between the esophagus and the stomach in all normal humans.

"Barrett's arises from this discrete group of pre-existing, residual embryonic cells present in all adults that seemingly lie-in-wait for a chance to take over when the esophagus is damaged," said McKeon. Added Xian, "We know these embryonic cells have different gene expression patterns from all normal tissues and this makes them inviting targets for therapies to destroy Barrett's before it progresses to cancer."

The therapeutic opportunities of this work are potentially immense.

"We are directing monoclonal antibodies to cell surface markers that can identify these precursor cells, so we may have a new opportunity to intervene therapeutically and prevent Barrett's esophagus in at-risk patients," said Wa Xian.

"Additionally," noted McKeon, "we are cloning the stem cells for both these precursors and for Barrett's esophagus itself, and these should represent critical targets for both monoclonal antibodies and small molecule inhibitors." Finally, there is reason to believe that this unusual mechanism might apply to a subset of other lethal cancers with unsure origins.

Crum noted that "some very aggressive cancers arise at junctions of two tissues and these deserve closer scrutiny to get at their origins if we are to surmount these diseases."

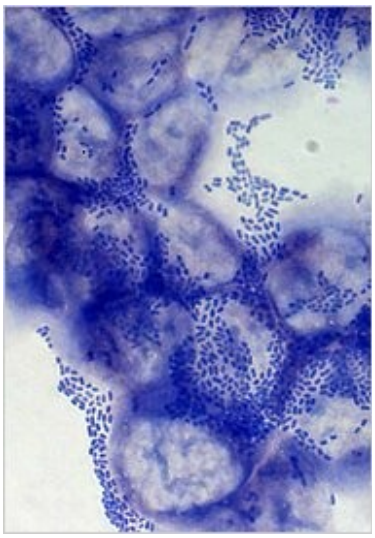
This work was supported by the National Institutes of Health.

http://www.nytimes.com/2011/06/23/health/research/23ecoli.html?_r=1&partner=rss&emc=rss

Unusual Traits Blended in Germany E. Coli Strain

By GINA KOLATA

The E. coli bacteria that killed dozens of people in Germany over the past month have a highly unusual combination of two traits and that may be what made the outbreak among the deadliest in recent history, scientists there are reporting.



One trait was a toxin, called Shiga, that causes severe illness, including bloody diarrhea and, in some patients, kidney failure. The other is the ability of this strain to gather on the surface of an intestinal wall in a dense pattern that looks like a stack of bricks, possibly enhancing the bacteria's ability to pump the toxin into the body.

The thought is that the bacteria started out being able to aggregate with the brick pattern and then were infected with a bacterial virus that gave them the Shiga toxin, said Dr. Matthew K. Waldor, an infectious-disease expert at Harvard Medical School who was not connected with the new research.

With the two traits combined in one strain of E. coli bacteria, "now they are highly virulent," Dr. Waldor said. The new findings, by a team led by Dr. Helge Karch of the University of Münster, were published Wednesday in the journal Lancet Infectious Diseases. They result from two days of fevered work to characterize the bacteria causing the illness that raced through Germany in May.

The E. coli O104:H4 strain has a pattern that looks like a stack of bricks on cultured intestinal epithelial cells.
University Hospital M*nster/Institute for Hygiene

Experts in the United States praised the German scientists' work. The work and the entire outbreak are "a real game-changer," said Dr. Philip I. Tarr, a professor of pediatrics and an expert in gut infections at the Washington University School of Medicine in St. Louis. Dr. John Mekalanos of Harvard called the paper "extremely important."

Other Shiga-producing bacteria adhere to the lining of the gut much less avidly, in diffuse clumps, not bricklike walls, Dr. Tarr said. And other strains of E. coli that do attach tightly to the gut do not make Shiga toxins. The combination of the two traits in one E. coli strain may be what makes this one so lethal.

Microbiologists knew, of course, that E. coli can be deadly; outbreaks in the United States involving tainted hamburger or vegetables have led to kidney failure in 5 to 10 percent of victims. And they knew that the most vulnerable were the very young and the very old.

But the recent outbreak, which has been traced to contaminated bean sprouts grown on a German farm, was different. As of June 20, it had sickened 2,684 people with diarrhea and 810 with kidney failure. Thirty-nine people died. The proportion with kidney failure - 25 percent - was "extraordinary," Dr. Waldor said.

Moreover, the victims tended to be young and middle-aged women.

Still, Dr. Michael T. Osterholm, an epidemiologist at the University of Minnesota who has investigated food-borne disease outbreaks in the United States, said that while there was no doubt that the German outbreak was horrendous, he questioned whether as many as 25 percent had kidney failure. The percentage depends on the denominator - the total number of people infected. And many, especially at the beginning of the outbreak when the numbers were highest, were not tested, Dr. Osterholm said. Labs had to look for Shiga toxin in stool, he said, and that "is hardly ever done."

But whether it was 25 percent of the infected or something less, the number of victims was sobering. The hospital in Münster was only mildly affected, compared with others in northern Germany. Yet Dr. Karch said that even there, "within a few weeks, 20 patients had to be dialyzed." Now, he added, although the epidemic is

dying out, at least 100 people will need kidney transplants or will have to undergo dialysis for the rest of their lives.

Dr. Karch, an expert in E. coli, infections, got the first stool samples on May 23. Over the next few days, more and more samples flooded his lab, 50 to 100 a day. “You can’t imagine,” he said.

He isolated the strain that was causing the illness and analyzed it to determine that it was strain O104:H4. Then he began investigating the bacteria’s DNA. First he determined what kind of Shiga toxin it made. Then he did adherence tests and found that the bacteria stuck to surfaces in the bricklike pattern. It is an unmistakable phenomenon: “Once you see it you will never forget it,” Dr. Karch said.

He posted the results and provided detailed information so most labs that had a suspicious stool sample could analyze it immediately and see if the stool contained O104:H4 bacteria. Until he posted that information, most labs would be at a loss. The strain is so rare that there are no standard tests to find it.

Dr. Karch also realized that the O104:H4 strain had been seen before in bloody diarrhea and kidney failure, but only on rare occasions - first in Germany in 2001, then sporadically in a few other countries. And in each outbreak, at most a few people were ill.

Why, then, was the German outbreak so widespread, and where did the bacteria go between outbreaks?

Many experts assumed the bacteria lived in animals, probably cattle. That is where the strain that usually causes severe illness, E. coli O157:H7, is found. And that is why it has spread all over the world as animals, and their meat, transmit it to humans. In fact, Dr. Karch said, E.coli O157:H7 is thought to have traveled to Europe from America in 1610, spread by cattle.

But the strain that caused the German outbreak does not seem to live in animals. “I think it is human-specific,” Dr. Karch said. And that increases the mystery of where it goes between outbreaks.

Dr. Karch thinks it smoldered in human populations, causing mild illnesses in most and occasionally causing severe disease. Then, somehow, it was passed to the bean sprouts by someone who harbored the bacteria. And since sprouts are eaten raw, they were highly infectious.

The strain is so rare, Dr. Karch said, that those infected had no immunity. An epidemic caught fire.

Women may have been the primary victims, Dr. Karch speculates, because they are more likely to eat sprouts. He himself does not like sprouts, he said, though his wife does. Aware that sprouts have always been “a high-risk food” for bacterial illnesses, he will not touch them unless they have been cooked.

http://www.newscientist.com/article/mg21028184.300-lab-yeast-make-evolutionary-leap-to-multicellularity.html

Lab yeast make evolutionary leap to multicellularity

*** 23 June 2011 by Bob Holmes**

IN JUST a few weeks single-celled yeast have evolved into a multicellular organism, complete with division of labour between cells. This suggests that the evolutionary leap to multicellularity may be a surprisingly small hurdle.

Multicellularity has evolved at least 20 times since life began, but the last time was about 200 million years ago, leaving few clues to the precise sequence of events. To understand the process better, William Ratcliff and colleagues at the University of Minnesota in St Paul set out to evolve multicellularity in a common unicellular lab organism, brewer's yeast.

Their approach was simple: they grew the yeast in a liquid and once each day gently centrifuged each culture, inoculating the next batch with the yeast that settled out on the bottom of each tube. Just as large sand particles settle faster than tiny silt, groups of cells settle faster than single ones, so the team effectively selected for yeast that clumped together.

One giant leap for yeastkind **Eye of Science/SPL**

Sure enough, within 60 days - about 350 generations - every one of their 10 culture lines had evolved a clumped, "snowflake" form. Crucially, the snowflakes formed not from unrelated cells banding together but from cells that remained connected to one another after division, so that all the cells in a snowflake were genetically identical relatives. This relatedness provides the conditions necessary for individual cells to cooperate for the good of the whole snowflake.

"The key step in the evolution of multicellularity is a shift in the level of selection from unicells to groups. Once that occurs, you can consider the clumps to be primitive multicellular organisms," says Ratcliff.

In some ways, the snowflakes do behave as if they are multicellular. They grow bigger by cell division and when the snowflakes reach a certain size a portion breaks off to form a daughter cell. This "life cycle" is much like the juvenile and adult stages of many multicellular organisms.

After a few hundred further generations of selection, the snowflakes also began to show a rudimentary division of labour. As the snowflakes reach their "adult" size, some cells undergo programmed cell death, providing weak points where daughters can break off. This lets the snowflakes make more offspring while leaving the parent large enough to sink quickly to the base of the tube, ensuring its survival. Snowflake lineages exposed to different evolutionary pressures evolved different levels of cell death. Since it is rarely to the advantage of an individual cell to die, this is a clear case of cooperation for the good of the larger organism. This is a key sign that the snowflakes are evolving as a unit, Ratcliff reported last week at a meeting of the Society for the Study of Evolution in Norman, Oklahoma.

Other researchers familiar with the work were generally enthusiastic. "It really seemed to me to have the elements of the unfolding in real time of a major transition," says Ben Kerr, an evolutionary biologist at the University of Washington in Seattle. "The fact that it happened so quickly was really exciting."

Sceptics, however, point out that many yeast strains naturally form colonies, and that their ancestors were multicellular tens or hundreds of millions of years ago. As a result, they may have retained some evolved mechanisms for cell adhesion and programmed cell death, effectively stacking the deck in favour of Ratcliff's experiment.

"I bet that yeast, having once been multicellular, never lost it completely," says Neil Blackstone, an evolutionary biologist at Northern Illinois University in DeKalb. "I don't think if you took something that had never been multicellular you would get it so quickly."

Even so, much of evolution proceeds by co-opting existing traits for new uses - and that's exactly what Ratcliff's yeast do. "I wouldn't expect these things to all pop up de novo, but for the cell to have many of the elements already present for other reasons," says Kerr.

Ratcliff and his colleagues are planning to address that objection head-on, by doing similar experiments with Chlamydomonas, a single-celled alga that has no multicellular ancestors. They are also continuing their yeast experiments to see whether further division of labour will evolve within the snowflakes. Both approaches offer an unprecedented opportunity to bring experimental rigour to the study of one of the most important leaps in our distant evolutionary past.

http://news.discovery.com/human/driving-may-influence-left-sided-skin-cancers.html

Driving Triggering Left-Sided Skin Cancers?

By Marianne English | Thu Jun 23, 2011 02:08 PM ET

Since the days of Driver's Ed, we've been conditioned to buckle up and check our mirrors before driving a car.

But should applying sunscreen on a sunny day be added to our pre-departure regimen? One recent study supports the idea in certain situations, especially for those who hit the road with the windows down or often drive when sunlight beams through side windows. After examining roughly 85,000 cases of malignant melanoma and Merkel cell carcinoma in U.S. patients from 1986 to 2006, dermatologists and scientists found these potentially fatal cancers occurred more often on the left side of patients' faces and arms than the right.

With driver's seats positioned in the left side of cars, the research suggests the trend can be traced back to drivers receiving more exposure to UV radiation on the left sides of their bodies while driving.

In the sample, 52 percent of melanoma patients developed the cancer on the left side of their bodies, while almost 48 percent had the condition on the right side. For Merkel cell carcinoma, approximately 53 percent of cases developed on the left side compared to around 47 percent on the right.

Though the difference seems small, it was statistically significant, meaning left-sided cancers occurred more frequently than by chance. Left-side trends were also seen more in men, but the results were less pronounced.

Though the findings are intriguing, researchers were unable to track patients' exposure to sun over the years. It's also difficult to be sure that driving was the primary culprit in all cases. In addition, the team only used cases in which skin cancers were attributable to one side of the body. In other words, a person with cancer in the middle of his chest was not included in the sample.

Weaknesses aside, the findings could hold true for many patients. With this knowledge, people working in travel-intensive professions such as truck driving might benefit from the added protection of sunscreen, especially if they enjoy driving with the window down. Also, since car windows do not shield drivers from all UV rays - some UVA rays still penetrate through, it's not a bad idea to have sunscreen handy, notably if you feel you're getting burned. Other research tackled whether skin cancer asymmetry exists, but the evidence wasn't as strong.

Similar results have popped up abroad, too. Researchers in Australia, where driver seats are positioned on the right side of vehicles, posed the same question and discovered similar results on the right side of patients' bodies.

http://www.bbc.co.uk/news/health-13887909

Type 2 diabetes in newly diagnosed 'can be reversed'

An extreme eight-week diet of 600 calories a day can reverse Type 2 diabetes in people newly diagnosed with the disease, says a Diabetologia study.

Newcastle University researchers found the low-calorie diet reduced fat levels in the pancreas and liver, which helped insulin production return to normal. Seven out of 11 people studied were free of diabetes three months later, say findings published in the journal. More research is needed to see whether the reversal is permanent, say experts.

Type 2 diabetes affects 2.5m people in the UK. It develops when not enough insulin is produced in the body or the insulin that is made by the body doesn't work properly. When this happens, glucose - a type of sugar - builds up in the blood instead of being broken down into energy or fuel which the body needs.

The 11 participants in the study were all diagnosed with Type 2 diabetes within the previous four years. They cut their food intake drastically for two months, eating only liquid diet drinks and non-starchy vegetables.

Fat loss

After one week of the diet, researchers found that the pre-breakfast blood sugar levels of all participants had returned to normal. MRI scans of their pancreases also revealed that the fat levels in the organ had decreased from around 8% - an elevated level - to a more normal 6%.

Three months after the end of the diet, when participants had returned to eating normally and received advice on healthy eating and portion size, most no longer suffered from the condition.

Professor Roy Taylor, director of Newcastle Magnetic Resonance Centre at Newcastle University and lead study author, said he was not suggesting that people should follow the diet. "This diet was only used to test the hypothesis that if people lose substantial weight they will lose their diabetes. "Although this study involved people diagnosed with diabetes within the last four years, there is potential for people with longer-standing diabetes to turn things around too."

Susceptibility question

Dr Ee Lin Lim, also from Newcastle University's research team, said that although dietary factors were already known to have an impact on Type 2 diabetes, the research showed that the disease did not have to be a life sentence. "It's easy to take a pill, but harder to change lifestyle for good. Asking people to shift weight does actually work," she said.

However, not everyone in the study managed to stay free of diabetes. "It all depends on how much individuals are susceptible to diabetes. We need to find out why some people are more susceptible than others, then target these obese people. We can't know the reasons for that in this study," Dr Lim said.

Professor Edwin Gale, a diabetes expert from the University of Bristol, said the study did not reveal anything new. "We have known that starvation is a good cure for diabetes. If we introduced rationing tomorrow, then we could get rid of diabetes in this country.

"If you can catch people with diabetes in the early stages while beta cells are still functioning, then you can delay its onset for years, but you will get it sooner or later because it's in the system."

But Keith Frayn, professor of human metabolism at the University of Oxford, said the Newcastle study was important. "People who lose large amounts of weight following surgery to alter their stomach size or the plumbing of their intestines often lose their diabetes and no longer need treatment.

"This study shows that a period of marked weight loss can produce the same reversal of Type 2 diabetes.

"It offers great hope for many people with diabetes, although it must be said that not everyone will find it possible to stick to the extremely low-calorie diet used in this study."

Dr Iain Frame, director of research at Diabetes UK, which funded the study, said the diet was not an easy fix.

"Such a drastic diet should only be undertaken under medical supervision. Despite being a very small trial, we look forward to future results particularly to see whether the reversal would remain in the long term."

http://www.eurekalert.org/pub_releases/2011-06/bifa-lpp062011.php

Lithium profoundly prevents brain damage associated with Parkinson's disease

Buck Institute research in mice moves into preclinical stage; working toward human trials

Lithium profoundly prevents the aggregation of toxic proteins and cell loss associated with Parkinson's disease (PD) in a mouse model of the condition.

Preclinical research is now underway at the Buck Institute for Research on Aging to determine correct dosages for a drug that continues to be the gold standard for the treatment of bipolar disorder. The Buck is

currently working toward initiating a Phase IIa clinical studies of lithium in humans in conjunction with standard PD drug therapy. The research appears in the June 24 online edition of the Journal of Neuroscience Research.

"This is the first time lithium has been tested in an animal model of PD," said lead author and Buck Professor Julie Andersen, PhD. "The fact that lithium's safety profile in humans is well understood greatly reduces trial risk and lowers a significant hurdle to getting it into the clinic."

According to Andersen, lithium has recently been suggested to be neuroprotective in relation to several neurodegenerative conditions including Alzheimer's disease, Huntington's disease and amyotrophic lateral sclerosis and has been touted for its anti-aging properties in simple animals. "We fed our mice levels of lithium that were at the low end of the therapeutic range," said Andersen. "The possibility that lithium could be effective in PD patients at subclinical levels is exciting, because it would avoid many side effects associated at the higher dose range." Overuse of lithium has been linked to hyperthyroidism and kidney toxicity.

PD is a progressive, incurable neurodegenerative disorder that affects 1 million Americans and results in tremor, slowness of movement and rigidity. It is the second most common neurodegenerative disease after Alzheimer's. Between 50,000 and 60,000 new cases are diagnosed each year. Age is the largest risk factor for the PD. Onset usually begins between the ages of 45 and 70 years.

Andersen's research focuses on lithium as a potential treatment for PD as well as its efficacy in combination with drugs currently used to control the symptoms of the disease. An internet search reveals stories from PD patients who are using lithium "off label" as part of their treatment regime; others report benefits from low dose lithium salts which are available as a supplement in some health food stores. "This finding gives us an opportunity to explore lithium as a recognized therapeutic for PD, in doses that are safe and effective" said Andersen.

Contributors to the work: *Other Buck Institute researchers involved in the study include Yong-Hwan Kim, Anand Rane and Stephanie Lussier. The research was supported by a grant from the National Institutes of Health.*

Citation: *Lithium protects against oxidative stress-mediated cell death in alpha-synuclein over-expressing in vitro and in vivo models of Parkinson's disease. JNR: 852471-744204*

http://www.eurekalert.org/pub_releases/2011-06/iuso-cdl062411.php

Common drugs linked to cognitive impairment and possibly to increased risk of death

INDIANAPOLIS – *A large, long-term study confirms that medications with anticholinergic activity, which include many drugs frequently taken by older adults, cause cognitive impairment.*

The research is also the first to identify a possible link between these drugs – which include over-the-counter and prescription sleep aids and incontinence treatments – and risk of death.

The two-year study of the impact of these medications on 13,000 men and women aged 65 and older is part of the Medical Research Council (UK) Cognitive Function and Ageing Studies (CFAS), a large UK-based longitudinal multi-center study initiative looking at health and cognitive function in older adults. Results of the study of anticholinergics appear June 24, 2011 in an advanced online publication of the Journal of the American Geriatrics Society.

Anticholinergics affect the brain by blocking acetylcholine, a nervous system neurotransmitter. Over-the-counter products containing diphenhydramine, sold under various brand names such as Benadryl®, Dramamine®, Excedrin PM®, Nytol®, Sominex®, Tylenol PM®, and Unisom®, have anticolinergic activity. Other anticholinergic drugs, such as Paxil®, Detrol®, Demerol® and Elavil® are available by prescription.

"Our findings make it clear that clinicians need to review the cumulative anticholinergic burden in people presenting with cognitive impairment to determine if the drugs are causing decline in mental status," said co-author Malaz Boustani, M.D., Regenstrief Institute investigator, Indiana University School of Medicine associate professor of medicine, and research scientist with the IU Center for Aging Research.

"Physicians should review with older patients all the over-the-counter and prescription drugs they are taking to determine exposure," said Dr. Boustani a geriatrician who sees patients at Wishard Health Services' Healthy Aging Brain Center in Indianapolis.

The researchers, led by Chris Fox, M.D., of the University of East Anglia and Carol Brayne, M.D. of the University of Cambridge, used the Anticholinergic Cognitive Burden Scale developed by Dr. Boustani and colleagues at the Regenstrief Institute, Indiana University and in the United Kingdom to evaluate the link between anticholinergic activity and cognitive decline.

Medications with anticholinergic effects are used for many diseases including hypertension and congestive heart failure. The study found that older age, lower income, and greater number of health conditions increased use of medications with anticholinergic activity. Women were more likely to report taking anticholinergic

medications, due to the greater number of health conditions reported by women than by men. Participants living in institutions were more likely to report taking anticholinergic medications.

"We looked at drugs with either moderate and severe anticholinergic activity. After adjusting for age, sex, baseline mental status, education, income level, number of non-anticholinergic medications and health conditions, we found that taking anticholinergic medications was linked to cognitive impairment and for the first time to death," said study corresponding author Dr. Fox, a psychiatrist. "We need follow-up to determine the degree to which anticholinergics are being prescribed for diseases with significant risk of death and the impact of that on our findings."

Authors of the study are Chris Fox, M.D., University of East Anglia; Carol Brayne, M.D., Kathryn Richardson, M.Sc. and George M. Savva, Ph.D, University of Cambridge; Ian D. Maidment, M.A., Kent and Medway NHS and Social Care Partnership Trust; Fiona E. Matthews, Ph.D., Medical Research Council Biostatistics Unit; David Smithard, M.D., Kent Community Health NHS Trust; Simon Coulton M.Sc., University of Kent; Cornelius Katona, M.D., University College London and Malaz Boustani, M.D., M.P.H., Regenstrief Institute, Indiana University School of Medicine and IU Center for Aging Research.

"The Anticholinergic Medication Use and Cognitive Impairment in the Older Population: The Medical Research Council Cognitive Function and Ageing Study (CFAS)" *was funded by the Medical Research Council.*

"The Medical Research Council invests in cohort studies like CFAS because they provide vital clinical information through observation. Such projects require long-term commitment to fulfill their potential but having supported cohort studies for well over half a century, MRC funding and collaborations have made us an international leader in this field," said Chris Kennard, MBBS, Ph.D., chairman of the MRC's Neuroscience and Mental Health Board.

Dr. Boustani's development of the Anticholinergic Burden Scale was supported by the U.S. National Institute on Aging.

http://www.physorg.com/news/2011-06-states-circumcisions-funds-crisis.html

States stop circumcisions funds amid budget crisis

(AP) - ***A nationwide debate about circumcisions for newborn boys, combined with cash-strapped public health budgets, has Colorado taking sides with 17 other states that no longer fund Medicaid coverage of the once widely accepted procedure.***

For years, Colorado lawmakers considered doing away with funding for circumcisions under Medicaid - a move that would save the state $186,500 a year. Now facing a seismic budget shortfall estimated to be $1 billion at the beginning of this year, lawmakers finally approved the change, which takes effect July 1.

"We were just looking at virtually every option and trying to decide what was absolutely urgent now," said Republican Sen. Kent Lambert, a member of the budget-writing Joint Budget Committee. "I think 99 percent of it was completely economic."

The matter of circumcisions has gotten contentious in California, where San Francisco will be the first city to hold a public vote in November on whether to ban the practice. Jewish and Muslim families are challenging that proposal in court, claiming it violates their right to practice their religion and decide what's best for their children. Supporters of the ballot initiative say male circumcision is a form of genital mutilation that parents should not be able to force on their children.

Matthew Hess, the president of the group behind the San Francisco proposal, called the Male Genital Mutilation Bill, applauded Colorado's move and said he hopes it will lead to a drop in the circumcision rate.

"That's a good thing, because paying someone to amputate a healthy functional body part from an unconsenting minor is not just a waste of taxpayer money - it's also a violation of human rights," he said.

South Carolina is one of the most recent states to eliminate Medicaid payments for circumcisions amid budget concerns. The change, which went into effect in February, was expected to save the state about $114,800 a year. States that also no longer fund circumcisions through Medicaid include Arizona, California, Florida, Maine and Louisiana.

Scott Levin, the regional director of the Mountain States office of the Anti-Defamation League, said Jews are unlikely to be affected by the defunding of Medicaid payments for circumcisions. For them, the procedure is not performed by a hospital physician, but a mohel - a specialist trained in Jewish ritual circumcision.

Levin said his group is more concerned about places like San Francisco that are trying to ban the procedure because Jewish people see the ritual as one of their religion's most sacred.

The World Health Organization reported that circumcisions are one of the most common procedures performed on newborn males in the United States, but the practice is not as common in the rest of the world. About 75 percent of baby boys in the U.S. are circumcised, compared to 30 percent elsewhere, the organization said. The figures refer to non-religious circumcisions.

Joanne Zahora, spokeswoman for the Colorado Department of Health Care Policy and Financing, which administers health care programs for low-income families, said the research her organization has seen shows that circumcisions are not medically necessary. But the procedure retains its supporters. Although the topic

never became heated during the Colorado budgetary debate, some lawmakers spoke in favor of keeping the Medicaid funding. Among them was Democratic Sen. Irene Aguilar, a primary care doctor at Denver Health.

"It's really a pretty inexpensive procedure to perform, and so it's just a little penny-wise and dollar-poor," she said.

Aguilar argued that circumcisions reduce the rates of urinary tract infections, penile cancer, and also lower the rates of cervical cancer for men's sexual partners. She also said she worried that doing away with funding for circumcisions would be discriminatory for Jewish and Muslim people on Medicaid.

Lambert, from Colorado's JBC, said the topic is sensitive for most, but the question lawmakers faced really was whether the government has the money to pay for the procedure.

"I think the general answer was no," Lambert said.

http://medicalxpress.com/news/2011-06-diet-reverses-diabetes.html

Diet reverses type 2 diabetes

(Medical Xpress) - *A Newcastle University team has discovered that Type 2 diabetes can be reversed by an extreme low calorie diet alone.*

Affecting two and half million people in the UK – and on the increase – Type 2 diabetes is a long-term condition caused by too much glucose, a type of sugar, in the blood.

In an early stage clinical trial of 11 people, funded by Diabetes UK, all reversed their diabetes by drastically cutting their food intake to just 600 calories a day for two months. And three months later, seven remained free of diabetes.

Professor Roy Taylor of Newcastle University who led the study and is also Director of the Newcastle Magnetic Resonance Centre said: "To have people free of diabetes after years with the condition is remarkable - and all because of an eight week diet.

"This is a radical change in understanding Type 2 diabetes. It will change how we can explain it to people newly diagnosed with the condition. While it has long been believed that someone with Type 2 diabetes will always have the disease, and that it will steadily get worse, we have shown that we can reverse the condition."

Research revealed today at the American Diabetes Association conference and published in Diabetologia transforms thinking on diabetes. It demonstrates that people who go on a very low calorie diet can remove fat which is clogging up the pancreas allowing normal insulin secretion to be restored.

Traditionally, it has been thought that as a progressive condition, Type 2 diabetes can be controlled by diet initially then tablets, but may eventually require insulin injections.

Type 2 diabetes, which was once known as adult-onset diabetes, is now found in young adults and children. It is caused by too much glucose in the blood due to the pancreas not producing enough insulin - a hormone which breaks down glucose into energy in the cells – or due to the body not reacting to it, known as insulin sensitivity.

The results of the diet

Under close supervision of a medical team, 11 people who had developed diabetes later in life were put on an extreme diet of just 600 calories a day consisting of liquid diet drinks and non-starchy vegetables. They were matched to a control group of people without diabetes and then monitored over eight weeks. Insulin production from their pancreas and fat content in the liver and pancreas were studied.

After just one week, the Newcastle University team found that their pre-breakfast blood sugar levels had returned to normal.

A special MRI scan of their pancreas revealed that the fat levels in the pancreas had returned from an elevated level to normal (from around 8% to 6%). In step with this, the pancreas regained the normal ability to make insulin and as a result, blood sugar after meals steadily improved.

The volunteers were then followed-up three months later. During this time they had returned to eating normally but had received advice on portion size and healthy eating. Of the ten people re-tested, seven remained free of diabetes.

"We believe this shows that Type 2 diabetes is all about energy balance in the body," explained Professor Taylor, "if you are eating more than you burn, then the excess is stored in the liver and pancreas as fat which can lead to Type 2 diabetes in some people. What we need to examine further is why some people are more susceptible to developing diabetes than others."

Dr Iain Frame, Director of Research at Diabetes UK, said: "We welcome the results of this research because it shows that Type 2 diabetes can be reversed, on a par with successful surgery without the side effects. However, this diet is not an easy fix and Diabetes UK strongly recommends that such a drastic diet should only be undertaken under medical supervision. Despite being a very small trial, we look forward to future results particularly to see whether the reversal would remain in the long term."

"I no longer needed my diabetes tablets"

Gordon Parmley, 67, from Stocksfield in Northumberland took part in the trial. He said: ""I love playing golf but I was finding that when I was out on the course sometimes my vision would go fuzzy and I would have trouble focussing. It was after this that I was diagnosed with Type 2 diabetes. That was about six years ago and from then on, I had to control the diabetes with a daily combination of tablets - the diabetes drug, gliclazide and tablets for my cholesterol.

"When my doctor mentioned the trial I thought I would give it a go as it might help me and other diabetics. I came off my tablets and had three diet shakes a day and some salad or vegetables but it was very, very difficult and I'm not sure I'd have done it without the support of my wife who went on a diet alongside me.

"At first the hunger was quite severe and I had to distract myself with something else – walking the dog, playing golf – or doing anything to occupy myself and take my mind off food but I lost an astounding amount of weight in a short space of time.

"At the end of the trial, I was told my insulin levels were normal and after six years, I no longer needed my diabetes tablets. Still today, 18 months on, I don't take them. It's astonishing really that a diet – hard as it was – could change my health so drastically. After six years of having diabetes I can tell the difference - I feel better, even walking round the golf course is easier." *Provided by Newcastle University*

http://www.scientificamerican.com/article.cfm?id=special-report-japans-throwaw

Special Report: Japan's "Throwaway" Nuclear Workers

A decade and a half before it blew apart in a hydrogen blast that punctuated the worst nuclear accident since Chernobyl, the No. 3 reactor at the Fukushima nuclear power plant was the scene of an earlier safety crisis.

By Kevin Krolicki and Chisa Fujioka

FUKUSHIMA, Japan (Reuters) - A decade and a half before it blew apart in a hydrogen blast that punctuated the worst nuclear accident since Chernobyl, the No. 3 reactor at the Fukushima nuclear power plant was the scene of an earlier safety crisis.

Then, as now, a small army of transient workers was put to work to try to stem the damage at the oldest nuclear reactor run by Japan's largest utility.

At the time, workers were racing to finish an unprecedented repair to address a dangerous defect: cracks in the drum-like steel assembly known as the "shroud" surrounding the radioactive core of the reactor.

But in 1997, the effort to save the 21-year-old reactor from being scrapped at a large loss to its operator, Tokyo Electric, also included a quiet effort to skirt Japan's safety rules: foreign workers were brought in for the most dangerous jobs, a manager of the project said.

"It's not well known, but I know what happened," Kazunori Fujii, who managed part of the shroud replacement in 1997, told Reuters. "What we did would not have been allowed under Japanese safety standards."

The previously undisclosed hiring of welders from the United States and Southeast Asia underscores the way Tokyo Electric, a powerful monopoly with deep political connections in Japan, outsourced its riskiest work and developed a lax safety culture in the years leading to the Fukushima disaster, experts say.

Story Continues . . .

http://www.physorg.com/news/2011-06-world-aircraft-serial-hybrid-electric.html

World's first aircraft with serial hybrid electric drive

Electromobility is now making inroads into the world of aircraft.

The technology of the series hybrid electric drive is scalable and will be used in small and medium-sized aircraft and, in the medium term, larger planes. It will make aviation greener.

Together with partners, Siemens has built the world's first aircraft with a serial hybrid electric drive system. The two-seater motor glider DA36 E-Star is presented by Siemens, Diamond Aircraft and EADS at the Paris Air Show Le Bourget 2011 (until June 26th) in daily flight shows. The aircraft was built to test the hybrid electric drive concept. In the future, the technology, which is intended for later use also in large-scale aircraft, will cut fuel consumption and emissions by 25 percent, compared to today's most efficient aircraft drives.

The motor glider, which is based on Diamond Aircraft's HK36 Super Dimona, is the only aircraft of its kind in the world. It is the first to use a so-called serial hybrid electric drive, which has been utilized to date only in cars, as an integrated drive train. The plane's propeller is powered by a 70 kW electric motor from Siemens. Electricity is supplied by a small Wankel engine from Austro Engine with a generator that functions solely as a

power source. A Siemens converter supplies the electric motor with power from the battery and the generator. Fuel consumption is very low since the combustion engine always runs with a constant low output of 30kW. A battery system from EADS provides the increased power required during takeoff and climb. The accumulator is recharged during the cruising phase. The plane is able to start noiseless with the electric drive. It can fly over long distances. During test flights it was airborne for about two hours.

The next development step will be to further optimize the entire drive train. Siemens scientists of the global research department Corporate Technology are currently working on a new electric motor that is expected to be five times lighter than conventional drives. In two years, another aircraft is expected to be equipped with an ultra-light electric drive. Siemens' Drive Technologies Division has already used integrated drive trains in other applications like marine drives. The knowhow gained in these areas has now been applied in the aviation industry as well. Combined with the corresponding product portfolio, the components of the drive train can be optimally adjusted to one another.

Air traffic accounts for some 2.2 percent of CO2 emissions worldwide. For this reason, aircraft, too, must become more efficient. In the long term, however, the drive system will also be used in large-scale aircraft.

Provided by Siemens

http://www.bbc.co.uk/news/health-13905331

Breast cancer prostate drug hope

By James Gallagher Health reporter, BBC News

Drugs used to treat prostate cancer in men may also be useful for difficult-to-treat breast cancers in some women, a Cancer Research UK study suggests.

Hormone treatments like tamoxifen and aromatase inhibitors are ineffective against up to 30% of breast cancers. But laboratory research in Cambridge, reported in The EMBO Journal, suggests some of these tumours may respond to drugs for male cancers. Cancer Research UK said the findings were a "great surprise".

Hormones can switch on genes which lead to cells dividing uncontrollably and developing into tumours. In women, breast cancers can be driven by the female sex hormone oestrogen. In men, prostate cancer can be driven by male sex hormones - androgens.

Breakthroughs have been made in treatments for breast cancer by developing drugs which interfere with the oestrogen's action, halting the tumour's progress. However, tumours which are not driven by the hormone have been harder to treat.

Prostate to breast

Researchers at the Cancer Research UK Cambridge Research Institute found that some of these oestrogen negative tumours were instead influenced by male hormones. The same genes which were switched on by female sex hormones in oestrogen responsive tumours were activated by the male sex hormones. It raises the prospect that drugs already developed for prostate cancer could help some women. While androgens, such as testosterone, are typically associated with male development, they are also present in women.

The lead researcher Dr Ian Mills said: "This important discovery suggests that patients with a type of oestrogen-receptor-negative breast cancer may potentially benefit from therapies given to prostate cancer patients, which could transform treatment for this patient group in the future. "But at the moment this laboratory research is still at an early stage." Researchers said this could apply to up to 5% of all breast cancers.

Dr Lesley Walker, from Cancer Research UK, said: "Prostate cancer depends on the androgen receptor for growth so it's a great surprise that a type of breast cancer might also be fuelled by this protein."

Dr Caitlin Palframan, policy manager at Breakthrough Breast Cancer, said: "This fascinating research opens the door to personalised treatment for a small group of breast cancer patients. "Women with oestrogen receptor negative disease have fewer treatment options and new ways to tackle it are urgently needed."

http://www.nytimes.com/2011/06/26/us/26cable.html?partner=rss&emc=rss

Atop TV Sets, a Power Drain Runs Nonstop

By ELISABETH ROSENTHAL

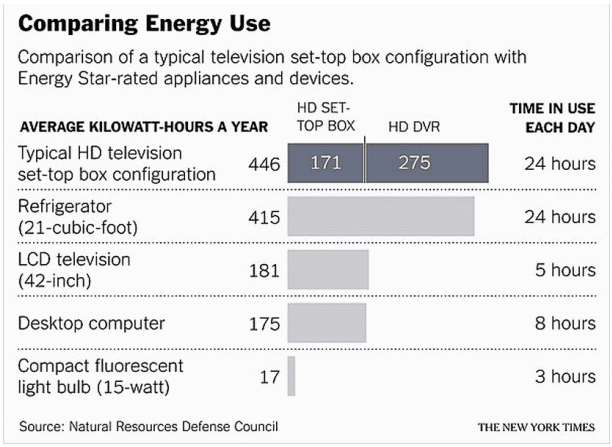
Those little boxes that usher cable signals and digital recording capacity into televisions have become the single largest electricity drain in many American homes, with some typical home entertainment configurations eating more power than a new refrigerator and even some central air-conditioning systems.

There are 160 million so-called set-top boxes in the United States, one for every two people, and that number is rising. Many homes now have one or more basic cable boxes as well as add-on DVRs, or digital video recorders, which use 40 percent more power than the set-top box.

One high-definition DVR and one high-definition cable box use an average of 446 kilowatt hours a year, about 10 percent more than a 21-cubic-foot energy-efficient refrigerator, a recent study found.

These set-top boxes are energy hogs mostly because their drives, tuners and other components are generally running full tilt, or nearly so, 24 hours a day, even when not in active use. The recent study, by the Natural Resources Defense Council, concluded that the boxes consumed $3 billion in electricity per year in the United States - and that 66 percent of that power is wasted when no one is watching and shows are not being recorded. That is more power than the state of Maryland uses over 12 months.

"People in the energy efficiency community worry a lot about these boxes, since they will make it more difficult to lower home energy use," said John Wilson, a former member of the California Energy Commission who is now with the San Francisco-based Energy Foundation. "Companies say it can't be done or it's too expensive. But in my experience, neither one is true. It can be done, and it often doesn't cost much, if anything."



The perpetually "powered on" state is largely a function of design and programming choices made by electronics companies and cable and Internet providers, which are related to the way cable networks function in the United States. Fixes exist, but they are not currently being mandated or deployed in the United States, critics say. Similar devices in some European countries, for example, can automatically go into standby mode when not in use, cutting power drawn by half. They can also go into an optional "deep sleep," which can reduce energy consumption by about 95 percent compared with when the machine is active.

One British company, Pace, sells such boxes to American providers, who do not take advantage of the reduced energy options because of worries that the lowest energy states could disrupt service. Cable companies say customers will not tolerate the time it takes to reboot the system once the system has been shut down or put to sleep.

"The issue of having more efficient equipment is of interest to us," said Justin Venech, a spokesman for Time Warner Cable. But, he added, "when we purchase the equipment, functionality and cost are the primary considerations." But energy efficiency experts say that technical fixes could eliminate or minimize the waiting time and inconvenience, some at little expense. Low-energy European systems reboot from deep sleep in one to two minutes. Alan Meier, a scientist at Lawrence Berkeley National Laboratory, said of the industry in the United States, "I don't want to use the word 'lazy,' but they have had different priorities, and saving energy is not one of them."

The Environmental Protection Agency has established Energy Star standards for set-top boxes and has plans to tighten them significantly by 2013, said Ann Bailey, director of Energy Star product labeling, in an e-mail. The voluntary seal indicates products that use energy efficiently. But today, there are many boxes on the list of products that meet the Energy Star standard that do not offer an automatic standby or sleep mode. "If you hit the on/off button it only dims the clock, it doesn't significantly reduce power use," said Noah Horowitz, senior scientist at the natural resources council.

Energy efficiency is a function of hardware, software, the cable network and how a customer uses the service, said Robert Turner, an engineer at Pace, which makes set-top boxes that can operate using less power while not in active use. Sometimes energy efficiency can be vastly improved by remotely adjusting software over a cable, Mr. Turner said. In this way, Pace reduced the energy consumption of some of its older boxes by half.

Cable boxes are not designed to be turned completely off, and even when in deep sleep mode, it takes time to reconnect and "talk" with their cable or satellite network, though that time is highly variable depending on the technology. Mr. Wilson said he routinely unplugged his set-top boxes at night and waited only 45 seconds for television in the morning. But Dr. Meier said that when he tried to power down his home system at night, it took "hours" to reboot because the provider "downloaded the programming guide in a very inefficient way."

Cable providers and box manufacturers like Cisco Systems, Samsung and Motorola currently do not feel consumer pressure to improve box efficiency. Customers are generally unaware of the problem - they do not know to blame the unobtrusive little device for the rise in their electricity bills, and do not choose their boxes anyway.

Those devices may cause an increase of as little as a few dollars a month or well over $10 for a home with many devices. In Europe, electricity rates are often double those in the United States, providing greater financial motivation to conserve.

Cisco Systems, one of the largest makers of set-top boxes, said in an e-mail that they would offer some new models this year that would cut consumption by 25 percent "through reduced power used in 'on' and standby states." There will be no deep sleep or fully "off" setting.

But Cisco said that taking advantage of the potential energy savings for a box would also depend on "how it is operated by the service provider." Cable and satellite providers will have to decide whether the boxes can automatically go to standby, for example, and whether customers will be able to adjust their own settings. Currently, providers often do system maintenance and download information at night over the cable, so an ever-at-the ready cable box is more convenient for them.

Cable companies can become Energy Star "partners" if they agree to install or upgrade boxes so that 25 percent to 50 percent of the homes they serve have "energy star qualified" equipment. The E.P.A. merely encourages providers to use units that can automatically power down at least partly when not in use.

But as of Sept. 1, typical electricity consumption of Energy Star qualified products would drop to 97 kilowatt hours a year from an average of 138; and then by the middle of 2013, they must drop again to 29 kilowatt hours a year. Companies have fought the placement of the "Energy Star" seal on products and the new ambitious requirements, which may still be modified before enacted.

Mr. Wilson recalled that when he was on the California Energy Commission, he asked box makers why the hard drives were on all the time, using so much power. The answer: "Nobody asked us to use less."

The biggest challenge in reducing energy use is maintaining the rapid response time now expected of home entertainment systems, Mr. Turner said. "People are used to the idea that computers take some time to boot up," he said, "but they expect the TV to turn on instantly."

http://www.eurekalert.org/pub_releases/2011-06/qmuo-rbc062311.php

Rogue blood cells may contribute to post-surgery organ damage

A study from scientists at Queen Mary, University of London, sheds new light on why people who experience serious trauma or go through major surgery, can suffer organ damage in parts of the body which are seemingly unconnected to the injury.

The study, published today in Nature Immunology*, examines the way certain white blood cells, called neutrophils move out of blood vessels to defend damaged organs against injury or infection.

This is normally a one-way journey but researchers were surprised to find that, in some cases, this process can go into reverse, with rogue super-activated neutrophils, re-entering the blood stream and causing damage to other parts of the body.

The researchers used a cutting edge imaging technique which allowed them to watch the movement of neutrophils, in three dimensions and in real time in mice. As they expected the neutrophils moved out of blood vessels and into tissues to tackle injury or infection and they showed that his process was being controlled by a protein on the surface of the blood vessels called JAM-C.

However, when they temporarily blocked the blood vessels, mimicking the trauma experienced by patients undergoing major surgery, JAM-C was lost from the blood vessels. When this happened the neutrophils seemed to loose their way. Cells that had already exited blood vessels returned to the blood stream and damaged other parts of the body. In particular, the researchers found that these confused but highly activated neutrophils lodged into blood vessels in the lungs where they appeared to cause inflammation and damage to lungs.

Further research on the JAM-C molecule and the properties of these rogue neutrophils could lead to the development of drugs aimed at reducing life threatening complications following major surgeries such as inflammation of the lungs.

Professor Sussan Nourshargh who led the study said: "This is a really exciting piece of research as we have been able to watch how white blood cells move out of blood vessels to enter parts of the body that need their help. But with the advanced imaging technique that we have developed we could also for the first time see neutrophils move back into blood vessels following trauma. The neutrophils that behave this way are very different from normal blood neutrophils in that they are highly activated and fully capable of causing damage to other organs."

"Neutrophils are usually our first line of defence against infection but they have the ability to cause many diseases. As we learn more about the complex processes that protect us against infections we also find ways of tackling inflammatory diseases where white blood cells are inappropriately switched on."

** 'The junctional adhesion molecule JAM-C regulates the polarized transendothelial migration of neutrophils in vivo', Woodfin, et al*

http://www.eurekalert.org/pub_releases/2011-06/hsif-gss062311.php

Genetic study shows that low body fat may not lower risk for heart disease and diabetes

BOSTON—*Having a lower percentage of body fat may not always lower your risk for heart disease and diabetes, according to a study by an international consortium of investigators.*

Having a lower percentage of body fat may not always lower your risk for heart disease and diabetes, according to a study by an international consortium of investigators, including two scientists from the Institute for Aging Research of Hebrew SeniorLife, an affiliate of Harvard Medical School (HMS).

The Institute researchers, Douglas P. Kiel, M.D., M.P.H., and David Karasik, Ph.D., who are working with the Framingham Heart Study, identified a gene that is linked with having less body fat, but also with an increased risk of type 2 diabetes and heart disease, examples of so-called "metabolic diseases."

"We've uncovered a truly fascinating genetic story and, when we found the effect of this gene, we were very intrigued by the unexpected finding," says Dr. Kiel, a senior scientist at the Institute for Aging Research and a professor of medicine at HMS. "People, particularly men, with a specific form of the gene are both more likely to have lower percent body fat, but also to develop heart disease and type 2 diabetes. In simple terms, it is not only overweight individuals who can be predisposed for these metabolic diseases."

Reported online in the journal Nature Genetics on June 26, 2011, the investigators examined the genomes of more than 75,000 people to look for the genes that determine body fat percentage. They found strong evidence for a gene, called IRS1, to be linked with having less body fat. On further study, they found that this gene also leads to having unhealthy levels of cholesterol and blood glucose.

To understand why a gene that lowers body fat can be harmful, the scientists in the international consortium found that the gene lowers only the "subcutaneous" fat under the skin, but not the more harmful "visceral" fat that surrounds organs. The study authors speculate that people with this gene variant are less able to store fat safely under the skin and may, therefore, store fat elsewhere in the body, where it may interfere with normal organ function. All observations were more pronounced in men than in women and, indeed, many apparently lean men still carry too much abdominal fat.

"Genetic variants may not only determine the amount of total fat in your body," says Dr. Kiel, "but also what kind of fat you have. Some collections of fat, such as the kind located just under the skin, may actually be less harmful than the type located in the abdominal cavity, which may increase the risk of developing metabolic disease."

The effect, the researchers add, may be more pronounced in men due to the different body fat distributions between the sexes. Men store less fat than women, so they are more sensitive to changes in its distribution.

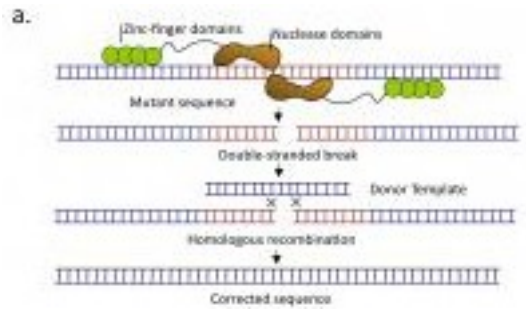
Headed by the Medical Research Council in the United Kingdom, the research consortium included scientists at 72 institutions in 10 countries, and used data from 26 different genetic studies.

http://www.eurekalert.org/pub_releases/2011-06/chop-gea062411.php

Genome editing, a next step in genetic therapy, corrects hemophilia in animals

Children's Hospital of Philadelphia study advances new strategy for gene therapy

Using an innovative gene therapy technique called genome editing that hones in on the precise location of mutated DNA, scientists have treated the blood clotting disorder hemophilia in mice. This is the first time that genome editing, which precisely targets and repairs a genetic defect, has been done in a living animal and achieved clinically meaningful results. The study appeared online today in Nature.



As such, it represents an important step forward in the decades-long scientific progression of gene therapy—developing treatments by correcting a disease-causing DNA sequence. In this new study, researchers used two versions of a genetically engineered virus (adeno-associated virus, or AAV)—one carrying enzymes that cut DNA in an exact spot and one carrying a replacement gene to be copied into the DNA sequence. All of this occurred in the liver cells of living mice.

From top, ZFNs on both sides of the DNA strand target a site upstream of the gene where most hemophilia-causing mutations reside. The ZFNs then cut both strands of the DNA double helix. As homologous recombination repairs the break in the strands, a normal copy of the clotting-factor gene (made from the donor template) is inserted into the DNA sequence, resulting in a corrected gene capable of expressing clotting factor. The mutated, nonfunctional gene remains in the DNA but is now harmless. **The Children's Hospital of Philadelphia**

"Our research raises the possibility that genome editing can correct a genetic defect at a clinically meaningful level after in vivo delivery of the zinc finger nucleases," said the study leader, Katherine A. High, M.D., a hematologist and gene therapy expert at The Children's Hospital of Philadelphia. High, a Howard Hughes Medical Institute Investigator, directs the Center for Cellular and Molecular Therapeutics at Children's Hospital, and has investigated gene therapy for hemophilia for more than a decade.

High's research, a collaboration with scientists at Sangamo BioSciences, Inc., makes use of genetically engineered enzymes called zinc finger nucleases (ZFNs) that act as molecular word processors, editing mutated sequences of DNA. Scientists have learned how to design ZFNs custom-matched to a specific gene location. ZFNs specific for the factor 9 gene (F9) were designed and used in conjunction with a DNA sequence that restored normal gene function lost in hemophilia. By precisely targeting a specific site along a chromosome, ZFNs have an advantage over conventional gene therapy techniques that may randomly deliver a replacement gene into an unfavorable location, bypassing normal biological regulatory components controlling the gene. This imprecise targeting carries a risk of "insertional mutagenesis," in which the corrective gene causes an unexpected alteration, such as triggering leukemia.

In hemophilia, an inherited single-gene mutation impairs a patient's ability to produce a blood-clotting protein, leading to spontaneous, sometimes life-threatening bleeding episodes. The two major forms of the disease, which occurs almost solely in males, are hemophilia A and hemophilia B, caused respectively by a lack of clotting factor VIII and clotting factor IX. Patients are treated with frequent infusions of clotting proteins, which are expensive and sometimes stimulate the body to produce antibodies that negate the benefits of treatment.

In the current study, the researchers used genetic engineering to produce mice with hemophilia B, modeling the disease in people. Before treatment, the mice had no detectable levels of clotting factor IX.

Previous studies by other researchers had shown that ZFNs could accomplish genome editing in cultured stem cells that were then injected into mice to treat sickle cell disease. However, this ex vivo approach is not feasible for many human genetic diseases, which affect whole organ systems. Therefore the current study tested whether genome editing was effective when directly performed in vivo (in a living animal).

High and colleagues designed two versions of a vector, or gene delivery vehicle, using adeno-associated virus (AAV). One AAV vector carried ZFNs to perform the editing, the other delivered a correctly functioning version of the F9 gene. Because different mutations in the same gene may cause hemophilia, the process replaced seven different coding sequences, covering 95 percent of the disease-carrying mutations in hemophilia B. The researchers injected mice with the gene therapy vector, which was designed to travel to the liver—where clotting factors are produced. The mice that received the ZFN/gene combination then produced enough clotting factor to reduce blood clotting times to nearly normal levels. Control mice receiving vectors lacking the ZFNs or the F9 minigene had no significant improvements in circulating factor or in clotting times.

The improvements persisted over the eight months of the study, and showed no toxic effects on growth, weight gain or liver function, clues that the treatment was well-tolerated.

"We established a proof of concept that we can perform genome editing in vivo, to produce stable and clinically meaningful results," said High. "We need to perform further studies to translate this finding into safe, effective treatments for hemophilia and other single-gene diseases in humans, but this is a promising strategy for gene therapy." She continued, "The clinical translation of genetic therapies from mouse models to humans has been a lengthy process, nearly two decades, but we are now seeing positive results in a range of diseases from inherited retinal disorders to hemophilia. In vivo genome editing will require time to mature as a therapeutic, but it represents the next goal in the development of genetic therapies."

Support for this work came from the National Institutes of Health and the Howard Hughes Medical Institute. High's co-authors were from The Children's Hospital of Philadelphia, the University of Pennsylvania, and Sangamo BioSciences, Inc. of Richmond, Calif.

"In vivo genome editing restores hemostasis in a mouse model of hemophilia," Nature, published online June 26, 2011. doi: 10.1038/nature10177

http://www.newscientist.com/article/dn20615-first-evidence-that-birds-tweet-using-grammar.html

First evidence that birds tweet using grammar

*** 18:00 26 June 2011 by Andy Coghlan**

They may not have verbs, nouns or past participles, but birds challenge the notion that humans alone have evolved grammatical rules.

Bengal finches have their own versions of such rules – known as syntax – says Kentaro Abe of Kyoto University, Japan. "Songbirds have a spontaneous ability to process syntactic structures in their songs," he says.

To show a sense of syntax in the animals, Abe's team played jumbled "ungrammatical" remixes of finch songs to the birds and measured the response calls.

Although many animals, including dogs, parrots and apes are known to interpret and construct "sentences", and recognise human words for individual objects, Abe says that only his finches have been shown to have a form of grammar in their utterances. Similar claims have been made for whale song, however.

In the wild, Bengal finches call back vigorously whenever they hear unfamiliar songs, usually from intruding finches. In the lab, Abe and colleague Dai Watanabe of the Japan Science and Technology Agency in Saitama exploited these reactions to gauge whether finches could notice "ungrammatical" songs.

The rules

First, they played finches unfamiliar songs repeatedly until the birds got used to them and stopped overreacting. Then they jumbled up syllables within each song and replayed these versions to the birds. "What we found was unexpected," says Abe. The birds reacted to only one of the four jumbled versions, called SEQ2, as if they noticed it violated some rule of grammar, whereas the other three remixes didn't. Almost 90 per cent of the birds tested responded in this way. "This indicates the existence of a specific rule in the sequential orderings of syllables in their songs, shared within the social community," Abe told New Scientist.

In subsequent experiments Abe showed that the rules were not innate – they had to be learned. Birds raised in isolation failed to react to SEQ2 until they had spent two weeks with other birds. He also taught birds unnatural grammatical rules by habituating them to one of his jumbled versions, then gauging their reactions to remixed versions that violated the "artificial" rules.

Finally, Abe chemically destroyed an area of the brain called the anterior nidopallium in some birds, and was thereby able to demonstrate that it is vital for registering faulty grammar. In humans, a region called Broca's area is activated when we hear ungrammatical sentences, so Abe suggests that studying the counterpart region in finches might throw new light on the origins of human grammar.

Bird words?

Constance Scharff, who works on birdsong at the Free University of Berlin, Germany, says the work is important because it is often claimed that humans are the only species that uses grammar.

"It's an ingenious experiment showing that birds are sensitive to changes in song that are consistent with different grammars," she says. "More and more, we are seeing similarities between humans and animals, and that makes some people uneasy." *Journal reference: Nature Neuroscience, DOI: 10.1038/nn.2869*

http://www.scientificamerican.com/article.cfm?id=hope-for-premature-babies-with-brain-injuries

Discovery Suggests Drugs Can Prevent Brain Injuries Common in Premature Babies

Identification of key protein for nerve repair opens avenue for potential therapy.

By Erica Check Hayden of Nature magazine

Scientists have identified the molecular players central to an incurable brain injury common in premature babies, and have shown how such injuries might one day be treated, sparing people from lifelong conditions such as cerebral palsy. In babies born before their lungs are fully developed, lack of oxygen can disrupt nerve cells' ability to make a protective coating, called myelin, that makes up the brain's 'white matter'. Without myelin, brain cells die, leaving children vulnerable to neurological deficits such as cerebral palsy. Some 20% of babies born before 6.5 months gestation experience lasting brain damage (see 'The most vulnerable brains').

"We have become very good at keeping these premature babies alive, but we have no strategy to prevent the long-term neurological consequences that can occur in them," says Vittorio Gallo, a neuroscientist at the Children's National Medical Center in Washington DC.

Writing in Nature Neuroscience, David Rowitch at the University of California, San Francisco, and his colleagues point the way AXIN2 was expressed in infants with white-matter brain injuries.

They also found AXIN2 in the damaged nerve cells of adults with multiple sclerosis, a disease in which the immune system attacks myelin. The AXIN2 protein interacts with proteins in the Wnt signalling pathway, which is involved in controlling many cellular processes, including development.

Repair kit

The authors went on to study young mice with white-matter nerve damage similar to that seen in premature babies. When the researchers injected myelin-deficient regions in the mice with a drug that prevents destruction of the AXIN2 protein, the mice grew myelin sheaths faster than untreated mice, repairing the damage.

"There's a lot of work needed before we want to seriously propose that this is going to be a therapeutic avenue," says Rowitch. "But this is the first evidence that this pathway can be manipulated therapeutically.

The study is an important demonstration that a drug might be used to repair brain damage, Gallo says. "One of the strengths of this paper is that it shows that a small molecule that affects the Wnt pathway promotes regeneration, so this shows one pharmaceutical way to treat this type of injury."

However, Stephen Back, a neurologist at the Oregon Health and Science University in Portland, points out that there is not yet proof that myelin-producing cells are stuck in arrested development in infants with brain injuries, although this has been shown both in mice and in adults with multiple sclerosis. So it is not entirely clear that a drug to speed their development would remedy such injuries.

"This work is certainly relevant to multiple sclerosis, but I would strike a cautionary note that it remains to be seen whether these myelin-producing cells are also arrested in newborn brain injury," Back says.

First author Stephen Fancy, a postdoc working with Rowitch, says that the work could be very important for multiple sclerosis. Although treatments are available for the disease, they do not repair the damage to nerve cells.

"This is going to be important in the future, both for multiple sclerosis and different types of newborn white-matter injury," Fancy says.