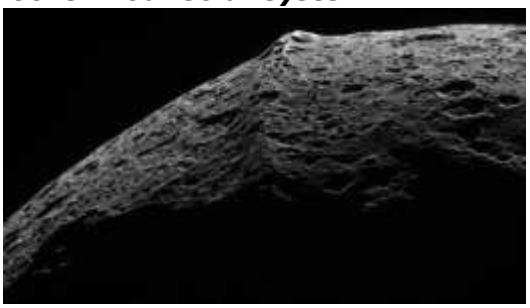
http://phys.org/news/2012-04-equatorial-ridge-saturn-moon-iapetus.html

How did the equatorial ridge on Saturn's moon Iapetus form?

Saturn's moon Iapetus is one of the most unusual moons in our solar system.



Perhaps the most bizarre feature of Iapetus is its equatorial ridge, a 20-km (12.4- mi) high, 200-km (124-mi) wide mountain range that runs exactly along the equator, circling more than 75 percent of the moon. No other body in the solar system exhibits such a feature, and as Dombard et al. show, previous models have been unable to adequately explain how the ridge formed.

The authors now propose that the ridge formed from an ancient giant impact that produced a subsatellite around Iapetus.

Raw image from Cassini space probe of the equatorial ridge on Saturn's moon Iapetus. Image: NASA

Tidal interactions with Iapetus ultimately led to orbital decay, eventually bringing the subsatellite close enough that the same forces tore it apart, forming a debris ring around Iapetus.

Material from this debris ring then rained down on Iapetus, creating the mountain ring along the equator.

More information: Delayed formation of the equatorial ridge on Iapetus from a subsatellite created in a giant impact, Journal of Geophysical Research-Planets, doi:10.1029/2011JE004010 , 2012.

http://www.eurekalert.org/pub_releases/2012-04/haoq-dcm040212.php

Death cap mushroom poison to arrest pancreatic cancer in mice

The mere thought of an identification error sends a chill down the spine of any mushroom lover:

The death cap mushroom (Amanita phalloides), which resembles the common white button mushroom, contains one of the most deadly poisons found in nature, α-amanitin. This substance kills any cell without exception, whether it be healthy or cancerous. At the German Cancer Research Center (Deutsches Krebsforschungszentrum, DKFZ) and the National Center for Tumor Diseases Heidelberg, immunologist Dr. Gerhard Moldenhauer, jointly with biochemist Professor Dr. Heinz Faulstich, Max Planck Institute for Medical Research, has now developed a method for destroying cancer cells using the dreaded fungal toxin without harming the body.

The trick to accomplish this is to deliver the poison directly to the right address in the body using something that virtually serves as a cab. In this case, the cab is an antibody whose highly specific arms attach to a cancer-typical cellular surface protein called EpCAM. The fungal toxin is linked to the antibody in a stable chemical conjugation.

In the culture dish, the poison-loaded antibody arrested the growth of pancreatic, colorectal, breast and bile duct cancer cell lines. In mice bearing transplanted human pancreatic cancer, a single antibody injection was sufficient to inhibit tumor growth. Two injections of higher doses of the antibody even caused complete tumor regression in 90 percent of the animals. Even the higher doses did not cause any poison-related damage to the liver or other organs of the animals.

EpCAM, the protein chosen by the Heidelberg immunologists as the tumor cell recognition structure, is a characteristic membrane protein of epithelial cells. This type of cells lines all inner and outer surfaces of the body. Most malignant tumors originate from such epithelial tissues. Many of these, such as pancreatic cancer, breast and ovarian cancers, bile duct carcinomas and tumors of the head and neck, produce too much EpCAM – and this is frequently associated with an extremely poor prognosis of the disease. EpCAM is therefore considered a suitable target structure for attacking tumor cells.

"Treatments with unconjugated antibodies against EpCAM have already been tested in clinical trials such as for breast cancer. They were intended to attack the cancer solely with the weapons of the immune system, but they turned out to be clinically ineffective," said Gerhard Moldenhauer. "However, our amanitin-conjugated antibody has a much greater potential for killing cancer cells."

Details are of vital importance

Each antibody is linked to between four and eight toxin molecules. Amanitin is regarded as very suitable for this purpose. It is small enough not to be recognized as foreign by immune cells, while it is also robust enough to lend itself to chemical conjugation. "When developing toxin-conjugated antibodies you have to take an awful lot of things into account," Moldenhauer explains. "The cancer cell has to regularly take the target molecule including the attached antibody into its interior, for this is the only place where the poison can act. In the cell's interior, the poison needs to detach from the antibody or else it will not be effective. At the same time it is absolutely vital that it does not get lost while it is being carried through the body, because this could cause severe adverse side effects."

The dosage of the amanitin antibody needs to be determined with the utmost care. One problem is that liver cells are extremely sensitive to the fungal toxin; another is that other healthy cells carry the EpCAM molecule as well and are therefore endangered. However, the results obtained in mice give reason to be optimistic, according to Gerhard Moldenhauer: "Even at high doses we have not detected any organ damage in the animals. We therefore expect that there is a sufficient therapeutic window for a dosage that kills cancer cells while leaving healthy tissue unaffected."

Moldenhauer, who has many years of experience in developing therapeutic antibodies, already has plans for amanitin-conjugated guided missiles against other cancers. In particular, certain types of leukemia and lymphoma cells also carry highly specific surface molecules which lend themselves as target structures for poison-loaded antibodies.

Gerhard Moldenhauer, Alexei V. Salnikov, Sandra Luttgau, Ingrid Herr, Jan Anderl and Heinz Faulstich: Therapeutic Potential of Amanitin-Conjugated Anti-Epithelial Cell Adhesion Molecule Monoclonal Antibody Against Pancreatic Carcinoma. JNCI Journal of the National Cancer Institute 2012; DOI: 10.1093/jnci/djs140

http://bit.ly/HGBb3J

Driverless cars ready to hit our roads

Sceptical about autonomous cars? Too late. They're already here – and they're smarter than ever

02 April 2012 by Paul Marks

LEAN back, let go of the steering wheel, ease your feet off the pedals and relax: your car is now in charge. The dream of a car that can drive itself has grown over the last decade as the necessary technologies have gradually proved their worth, but the idea has faced major legal hurdles.

Not for much longer. Politicians are now scrambling to make self-driving cars a reality. From Hawaii to Florida, and Oxford to Berlin, the race is on to get driverless cars onto our streets.

Promising improved safety, better fuel-efficiency and freedom from the boredom of long drives, autonomy has been coming piecemeal to our cars for some time - and it has always had its critics. In 1994, on a UK motorway, Jaguar and Lucas Industries demonstrated the safety of adaptive cruise control and automatic lane keeping; both technologies are now commonplace on our roads. The media were not impressed, describing the idea of cars that drive themselves as "madness".

But concerns about the safety of autonomous cars are misplaced in a world where 1.2 million people die every year in road accidents due to human error, says Paul Newman, a robotics engineer at the University of Oxford, whose team is developing autonomous cars.

"It's crazy to imagine that we are going to keep driving cars like we do now - that in 10 to 20 years we'll still have to sit behind a wheel, concentrating hard, not falling asleep and not running over people," he says.

This notion now has powerful backers - and barriers are beginning to fall. In an act that came into force on 1 March, the state of Nevada now allows driverless cars to ply the state's road network provided they sport a special red licence plate, and the owners pay a $1 to $3 million insurance bond. Similar legislation is being considered in California, Arizona, Florida, Hawaii and Oklahoma.

The phenomenon is not confined to the US either. In Germany, a driverless car research team led by Tinosch Ganjineh at the Free University of Berlin has permits to use the abandoned Templehof airport for autonomous tests. When necessary, team members get special permits to drive on Berlin's streets, and hope to drive on the autobahn soon. The Oxford team plan to approach the British government for similar permits.

The Berlin team are automating a VW Passat, patriotically named MadeInGermany, while Oxford is turning a BAE Systems WildCat military jeep into a self-driving machine. Nissan has just joined the Oxford project, so the Leaf all-electric car may end up driverless too.

Driverless cars first appeared in a meaningful way in the US Defence Advanced Research Project Agency's "grand challenges". Cars competed to drive fastest around desert courses in 2004 and 2005, and in an urban setting in 2007.

Mike Montemerlo and Sebastian Thrun of Stanford University, California, whose car won the 2005 prize, lead Google's self-driving car research programme. Their cars, based on the Toyota Prius and Audi TT, typify the approaches of the Oxford and Berlin teams. All the cars have laser rangefinders, radar and optical cameras to sense the vehicle's changing real-time environment with high accuracy. They know where the traffic lights and road signs are, and which moving objects are animals, people, bikes, motorbikes or trucks. Newman's team are studying how algorithms can make sense of data streaming from a 3D laser rangefinder and quickly decide whether an object is a car or pedestrian, for example. His team is also looking at how a robotic visual system can build up a picture of its world and adapt to changing conditions, varying light levels or even seasons. The commercial sensors and software to make this happen are still some way off, though.

"The Velodyne - the 64 spinning lasers on top of most driverless cars - give a quickly updated 360-degree, 3D view of the surroundings up to 40 metres away," says Newman. But cars of the future won't have unwieldy spinning lasers on them, he says.

Ganjineh agrees that driverless technology has to be refined. "The size and price of these systems needs to come down. Today, half a trunk of equipment is needed for autonomous driving," he says.

Another challenge, says Newman, is getting the cars to recognise the precursors to risky events - like sudden bright sun reflections on the road, truck spray, which may blind some sensors, or simply a burst tyre.

Google's cars, meanwhile, tell each other about the roads they have travelled, such as exchanging data on how to negotiate awkward junctions, says Vinton Cerf, a Google technology evangelist. Ganjineh wants similar technology to broadcast GPS map changes car-to-car, when there are roadworks ahead, for example.

However, driverless cars will not need to communicate wirelessly with expensive roadside technology as they need to be "independently smart" and aware of all risks around them at all times, says Newman.

"Automation of cars is going to happen," he says. "Computing has caused devastating change and transport is going to be its next target."

If the going's tough, the car gets cover

Driverless cars could reduce insurance costs, says Paul Newman of the University of Oxford, by allowing the car to add to its own insurance as road conditions change.

"On a dark icy night, when it is riskier to drive, the car could go online and bid for extra insurance cover until conditions change," he says. "If that proves too expensive, because conditions are tough for the autonomous system, the owner could take the wheel."

Meanwhile, clear standards for programmers and developers of such cars need to be drawn up, says Tinosch Ganjineh at the Free University of Berlin, Germany, as accident liability may fall more often on software or sensor-makers.

http://news.discovery.com/space/mars-dark-spots-120402.html

Martian Dark Spots Reveal Heart of Glass

Their finding represents the first detection of widespread surface weathering during the Amazonian epoch.

Content provided by Stuart Gary, ABC Science

Dark patches visible across much of the northern Martian hemisphere aren't canals or vegetation, as once thought, but volcanic glass according to a new study.

The discovery by Briony Horgan and James Bell from Arizona State University, provides evidence of the same sort of processes seen on Earth, also happening on the Red Planet.

Using near infrared spectroscopic data collected by the European Space Agency's Mars Express orbiter, Horgan and Bell found widespread weathered volcanic glass covering the surface of the Martian lowlands.

"The volcanic glass (probably basalt) was created when hot magma reacted explosively with ice or water," says Horgan. "The same sort of thing happens in Iceland, where volcanoes erupt under glaciers. The interaction with the ice and the water causes the magma to become super-explosive, creating tonnes of sand sized black particles. These form vast sand dunes covering about a quarter of Iceland's surface, which is exactly what we're seeing on Mars," says Horgan.

"We see these dark plains and enormous glassy sand dune fields up in the northern Martian polar regions."

Horgan and Bell also found evidence of dips in the spectrum consistent with weathering caused by the glass being exposed to acidic water.

Their finding, which appear in the journal Geology, represents the first detection of widespread surface weathering during the Amazonian epoch -- the most recent of the three Martian geologic periods.

"The deposits are young by Martian standards, just a few billion years old, but that's still well after the planet's warm and wet period ended," says Horgan.

According to Horgan, the evidence of weathering also supports the explosive magma hypothesis. "It works really well because we know there's lots of ice on Mars today," says Horgan. "In the past we think Mars went through multiple ice ages with huge icesheets, glaciers and snow packs covering the surface."

Horgan says the melt water would have included dissolved chemicals that weathered the surface of the glass deposits even under the arid conditions of Mars.

According to Horgan the creation of explosive volcanic glass also creates interesting habitats for study by astrobiologists. "If these things were created by magma ice interactions, they would have caused huge outflows of hot, chemically rich liquid water, which would have created a habitable environment which is one of the big drivers for the Mars Program today."

http://www.eurekalert.org/pub_releases/2012-04/uot-sfe_1032812.php

Scientists find evidence that human ancestors used fire one million years ago

300,000 years earlier than believed

An international team led by the University of Toronto and Hebrew University has identified the earliest known evidence of the use of fire by human ancestors. Microscopic traces of wood ash, alongside animal bones and stone tools, were found in a layer dated to one million years ago at the Wonderwerk Cave in South Africa.

"The analysis pushes the timing for the human use of fire back by 300,000 years, suggesting that human ancestors as early as Homo erectus may have begun using fire as part of their way of life," said U of T anthropologist Michael Chazan, co-director of the project and director of U of T's Archaeology Centre.

The research will be published in the Proceedings of the National Academy of Sciences on April 2.

Wonderwerk is a massive cave located near the edge of the Kalahari where earlier excavations by Peter Beaumont of the McGregor Museum in Kimberley, South Africa, had uncovered an extensive record of human occupation. A research project, co-directed by U of T's Chazan and Liora Kolska Horwitz of Hebrew University, has been doing detailed analysis of the material from Beaumont's excavation along with renewed field work on the Wonderwerk site. Analysis of sediment by lead authors Francesco Berna and Paul Goldberg of Boston University revealed ashed plant remains and burned bone fragments, both which appear to have been burned locally rather than carried into the cave by wind or water. The researchers also found extensive evidence of surface discoloration that is typical of burning.

"The control of fire would have been a major turning point in human evolution," says Chazan. "The impact of cooking food is well documented, but the impact of control over fire would have touched all elements of human society. Socializing around a camp fire might actually be an essential aspect of what makes us human."

The funding for the research was provided by the Social Sciences and Humanities Research Council of Canada, the National Science Foundation and the Wenner Gren Foundation. Other team members include James Brink and Sharon Holt of the National Museum, Bloemfontein, Marion Bamford of the University of Witswatersrand, and Ari Matmon and Hagai Ron of Hebrew University. Research at Wonderwerk Cave is carried out in collaboration with the McGregor Museum, Kimberley and under permit for the South African Heritage Resources Agency.

http://www.eurekalert.org/pub_releases/2012-04/gumc-bc040212.php

Breast cancer resistance linked to timing of soy consumption

Could women with breast cancer who began eating soy as an adult develop a tumor more resistant to treatment?

CHICAGO - Studies exploring the relationship between soy consumption and breast cancer have been mixed, but new research introduces a new thought: Could women with breast cancer who began eating soy as an adult develop a tumor more resistant to treatment?

That's the suggestion of a new study in animal models that could provide important hints for women with breast cancer who eat soy. The research from Georgetown Lombardi Comprehensive Cancer Center was reported today at the American Association for Cancer Research (AACR) Annual Meeting 2012.

For tumors that are sensitive to hormonal treatment (estrogen receptor and/or progesterone receptor positive), tamoxifen is often given after primary treatment to keep the cancer at bay. Unfortunately, many tumors become resistant to the tamoxifen --meaning the drug stops working and the cancer grows again.

In the new research conducted in the laboratories of Leena Hilakivi-Clarke, PhD and Robert Clarke, PhD, DSc, both professors of oncology at Georgetown Lombardi, researchers looked at the impact soy consumption might have on breast tumors.

For the study, female rats were fed soy isoflavone genistein (a estrogen-like compound in soy) at various points in their lifetime. At adulthood, all rats were exposed to a substance that triggered mammary tumors to develop and then given tamoxifen. The study groups were as follows: one group was never fed genistein until tamoxifen was started; a second group was fed genistein only in youth and not again until tamoxifen was started; a third group was fed genistein only as adults and continued after tamoxifen was given; and finally, a fourth group was fed genistein during youth and adulthood and continued after tamoxifen was given.

"Genistein intake in adult life which continues during tamoxifen treatment appears to make the tumors resistant to tamoxifen," explains Hilakivi-Clarke, the study's senior author. "However, if animals were fed genistein during childhood, and intake continues before and after tumors develop, the tumors are highly sensitive to the tamoxifen," she explains. "These results suggest that western women who started soy intake as adults, should stop if diagnosed with breast cancer," Clarke concludes.

The research was presented by the abstract's lead author Xiyuan Zhang, M.S. Other authors include Anni Warri, Ph.D., Idalia M. Cruz, and Katherine Cook, Ph.D.

The research was funded by the National Cancer Institute. The authors report having no personal financial interests related to the study.

http://www.sciencedaily.com/releases/2012/04/120402144738.htm

How Do I Love Me? Let Me Count the Ways, and Also Ace That Interview

The secret to excelling in a job interview may not hinge on how much your interviewers like you, but in how much you like yourself.

ScienceDaily - Narcissism, a trait considered obnoxious in most circumstances, actually pays off big-time in the short-term context of a job interview, according to a new study to be published in the Journal of Applied Social Psychology. Narcissists scored much higher in simulated job interviews than non-narcissists, researchers found. They pointed to narcissists' innate tendency to promote themselves, in part by engaging and speaking at length, which implied confidence and expertise even when they were held to account by expert interviewers.

"This is one setting where it's OK to say nice things about yourself and there are no ramifications. In fact, it's expected," said Peter Harms, assistant professor of management at the University of Nebraska-Lincoln and a co-author of the study. "Simply put, those who are comfortable doing this tend to do much better than those who aren't."

The two-part study examined the effectiveness of the types of behaviors that narcissists exhibit - which would be typically seen as maladjusted - in the narrow context of an interview. In the first part, 72 participants were videotaped in a simulated job-applicant setting. As expected narcissists were more likely to self-promote; however, it was when expert interviewers challenged applicants that narcissists started behaving in unexpected ways, Harms said.

While normal individuals backed off of their self-promotion tactics when held accountable, narcissists actually increased their attempts to make themselves look better.

"When feeling challenged, they tend to double down," Harms said. "It's as if they say 'Oh, you're going to challenge me? Then I'm not just great, I'm fantastic.' And in this setting, it tended to work."

In the study's second part, 222 raters evaluated videos of applicants with similar job skills and varying levels of narcissism. The raters consistently awarded chronic self-promoters -- who spoke quickly and at length and who used ingratiation tactics such as smiling, gesturing and complimenting others -- far more positive evaluations.

Meanwhile, equally qualified applicants who tended to rely on tactical modesty scored lower, according to the study. "This shows that what is getting (narcissists) the win is the delivery," Harms said. "These results show just how hard it is to effectively interview, and how fallible we can be when making interview judgments. We don't necessarily want to hire narcissists, but might end up doing so because they come off as being self-confident and capable."

For interviewers, the study's findings mean they must become aware of the tactics used by narcissists, Harms said -- and, if necessary, avoid selecting people who chronically use self-promotion and ingratiation, unless those behaviors are appropriate for the position.

"On the whole, we find very little evidence that narcissists are more or less effective workers. But what we do know is that they can be very disruptive and destructive when dealing with other people on a regular basis. If everything else is equal, it probably is best to avoid hiring them."

In addition to UNL's Harms, the study was authored by Delroy L. Paulhus, Bryce G. Westlake and Stryker S. Calvez of the University of British Columbia.

http://nyti.ms/HKJmJN

Solazyme and Bunge Plan Factory to Make Oil From Algae

The promise of using algae to make biofuels - a dream scientists have chased for decades - might seem particularly welcome in a time of stubbornly high gasoline prices.

By DIANE CARDWELL

But the path to commercial-scale production has been circuitous.

Young companies have attracted investors and interest, but as they struggle to make large quantities of ethanol and biodiesel at a competitive cost, some have branched out into making oils for products with higher profit margins, like cosmetics, food and soap.

Now, one of these companies, Solazyme, is about to take a step toward large-scale fuel production. On Tuesday, it will announce an agreement with Bunge Global Innovation to build a factory in Brazil that would make triglyceride oils for both chemical and fuel products.

Under the joint venture, whose financial terms were not disclosed, the factory would rise next to Bunge's Moema sugar cane mill and have an annual capacity of 100,000 metric tons, or about 110,250 short tons, of oil.

It would start production in the second half of next year, making oils for fuel as well as additives for soaps, detergents and plastics.

Ben Pearcy, a managing director of Bunge Ltd., a global agribusiness and food company, said in a prepared statement that the partnership would "enable us to link our sugar and vegetable oil value chains," and expand the company's reach in fuels and chemicals. For Solazyme, which has skin care and nutritional supplement lines, the arrangement provides a boost in production capacity "to meet the strong demand we're seeing in our initial target markets," said Jonathan Wolfson, the company's chief executive.

So far, the company has produced only limited quantities of biofuels, including several hundred thousand liters to the United States military, which is seeking to promote alternatives to conventional fuels.

Solazyme, which is based in South San Francisco, Calif., bioengineers its algae so that it can convert sugars directly into oils without photosynthesis. This allows the organisms to be grown in fermentation tanks, which reduces the costs of a still-expensive process.

A competitor, Sapphire Energy, announced on Monday that it had secured $144 million from investors for its demonstration plant in New Mexico. Sapphire, which does use photosynthesis in its production process, has also received a $50 million grant from the Energy Department and a $54.4 million loan guarantee from the Agriculture Department, according to the company.

http://www.eurekalert.org/pub_releases/2012-04/plos-iha032912.php

Increasing height and body mass index are risk factors for ovarian cancer

Study suggests increasing height and increased body mass index are risk factors for developing ovarian cancer.

A study in this week's PLoS Medicine suggests that increasing height and, among women who have never taken menopausal hormone therapy, increased body mass index are risk factors for developing ovarian cancer.

These findings are important as in high income countries, the average height and average body mass index of women have increased by about 1 cm and 1 kg/m2 respectively per decade. These findings suggest that if all other factors that affect ovarian cancer risk had remained constant, the increases in height and weight among women would have resulted in ovarian cancer incidence increasing by 3% per decade.

The Collaborative Group on Epidemiological Studies of Ovarian Cancer, based in the University of Oxford and involving over 100 researchers internationally, reached these conclusions by analyzing individual-patient data from 47 epidemiological studies from 14 countries which comprised a total of 25,157 women with ovarian cancer and 81,311 women without ovarian cancer – virtually all the relevant data on the topic worldwide.

The researchers found a significant increase in the risk of developing ovarian cancer for every 5cm increase in height which did not vary when other factors such as age, menopausal status, smoking, alcohol consumption, having first degree relatives with ovarian or breast cancer, use of oral contraceptives, or use of menopausal hormone therapy, were taken into account. However, for body mass index, the risks depended on whether women had ever taken menopausal hormone therapy but not on 11 other factors examined.

The collaborators say: "This collaboration has brought together and re-analysed individual data from 47 studies on ovarian cancer risk associated with adult height, weight, and body mass index, that is, most of the available epidemiological information worldwide. Collectively, the findings show a highly significant increase in the risk of ovarian cancer with increasing values for each of the anthropometric variables examined."

They continue: "The increase in ovarian cancer risk with increasing height and with increasing body mass index did not vary materially by women's age, year of birth, ethnicity, education, age at menarche, parity, family history of ovarian or breast cancer, use of oral contraceptives, menopausal status, hysterectomy, or consumption of alcohol and tobacco. However use of hormone therapy for the menopause attenuated the relationship with body mass index, since an increase in ovarian cancer risk with increasing adiposity was found only in never-users of such therapy."

They add: "An advantage of seeking to review all epidemiological studies of ovarian cancer with relevant information on body size, published and unpublished, is that this helps avoid unduly selective emphasis on published results or just on some studies."

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Competing Interests: The authors have declared that no competing interests exist.

Citation: *Collaborative Group on Epidemiological Studies of Ovarian Cancer (2012) Ovarian Cancer and Body Size: Individual Participant Meta-Analysis Including 25,157 Women with Ovarian Cancer from 47 Epidemiological Studies. PLoS Med 9(4): e1001200. doi:10.1371/journal.pmed.1001200*

http://www.eurekalert.org/pub_releases/2012-04/obi-hhv040312.php

Harmless human virus may be able to boost the effects of chemotherapy

Trials show promise of human virus to treat head and neck cancer patients

A naturally-occurring harmless human virus may be able to boost the effects of two standard chemotherapy drugs in some cancer patients, according to early stage trial data published in Clinical Cancer Research.

The paper titled: Phase I/II trial of carboplatin and paclitaxel chemotherapy in combination with intravenous oncolytic reovirus in patients with advanced malignancies with first author Eleni M. Karapanagiotou from the ICR and The Royal Marsden was published in the print edition of Clinical Cancer Research on April 1.

RT3D, trade name Reolysin, is a new drug developed by Oncolytics Biotech Inc with preclinical and clinical studies conducted at The Institute of Cancer Research (ICR) and The Royal Marsden Hospital. It is based on a virus (reovirus type 3 Dearing) that is found in almost all adults' respiratory and gastrointestinal tracts without causing any symptoms.

RT3D has the ability to grow in and kill certain types of cancer cells, but does not grow in normal cells.

Previous trials injecting patients with the virus on its own showed limited effectiveness, but the team found that RT3D appeared to magnify the effects of platin and taxane-based chemotherapy on tumour cells.

Dr Kevin Harrington and colleagues in Leeds therefore started a clinical trial testing intravenous RT3D in combination with chemotherapeutics carboplatin and paclitaxel in 31 patients with advanced cancers who had stopped responding to standard treatments.

An initial Phase I study was carried out in patients with a range of advanced cancers, which showed the drug combination was safe. Side-effects were found to be generally mild, and consistent with chemotherapy alone.

Patients with head and neck cancers were found to have the best responses, so a Phase II expansion study at The Royal Marsden Hospital, London, and St James's Hospital, Leeds, was therefore targeted to patients with these types of cancers.

Cancers shrank for about one third of the patients who could be evaluated, and disease stabilised for a further third. For one patient, all signs of their cancer disappeared.

"We saw really very impressive response rates in these patients. These are patients whose cancers had grown despite a great deal of previous treatment, including platinum-based chemotherapy for many," Dr Harrington, Leader of the ICR's Targeted Therapy Team and Consultant Oncologist at The Royal Marsden, said. "Under those circumstances, we'd expect that the average response rate to chemotherapy alone might be as low as single digits figures and the average survival would be somewhere between three to four months. In our Phase I/II study we show this had been prolonged to an average of seven months, albeit not in a randomised trial."

"Based on the results of this study we've now started recruiting patients with advanced head and neck cancer to a randomised Phase III trial, in which all patients will receive chemotherapy and half will receive Reolysin as well. We are extremely excited about this progress."

The study also found the virus was not shed after treatment. This means people could be given the drug as outpatients as no risk was found that they could transmit the virus to others.

http://www.eurekalert.org/pub_releases/2012-04/gumc-sts032312.php

Stopping the spread of a deadly childhood bone cancer

Many children with the bone cancer, osteosarcoma, die after the tumor spreads to their lungs

CHICAGO -. In a critical step toward finding a way to stop metastasis, researchers at Georgetown Lombardi Comprehensive Cancer Center say they have discovered an agent that prevents this type of cancer from spreading to the lungs in mice with the disease.

The new agent stops or inhibits "ezrin," a protein vital to the spread of osteosarcoma, say the researchers who presented their findings today at the American Association for Cancer Research (AACR) Annual Meeting 2012. If proven effective in human studies, their ezrin inhibitor might potentially treat adults whose cancers are fueled by over-expression of this protein, and could be a life-saver for children with bone tumors.

"If we can prevent metastatic disease in osteosarcoma, we will significantly improve survival and quality of life for these patients," says the study's senior investigator, Aykut Üren, M.D., an associate professor of oncology, and of biochemistry and molecular & cellular biology at Georgetown Lombardi Comprehensive Cancer Center.

The molecule they discovered represents the first-in-class ezrin inhibitor, he says. "In addition to its potential clinical application, an ezrin inhibitor will be an extremely valuable tool in the laboratory as we work to better understand how ezrin works."

Ezrin is present in many types of cells in the body including cancer cells. It controls how the cell interacts with its environment, how the cell moves and how it survives in new locations.

In osteosarcoma, the tumor cells that produce high levels of ezrin are more aggressively invasive, Üren says. "Ezrin also helps cancer cells survive when they reach the lungs. If an osteosarcoma cell with no ezrin spreads to the lung, it can't grow there. Having too much ezrin makes it easier for cancer cells to move to the lungs and, once there, it gives these cells a growth advantage."

Osteosarcoma most commonly develops around knee and shoulder joints in children and is "relatively easy to treat the tumor on the limbs, but when the lungs are involved, patients usually die due to pulmonary insufficiency," he says.

If successfully developed, an ezrin inhibitor may be useful in preventing the spread of other tumors, too. Breast cancers, colon cancers, melanoma, ovarian carcinoma, brain tumors, and soft tissue sarcomas all show evidence that too much ezrin may mean poor survival outcomes for the patient, says Üren.

Üren and his research team are currently testing several novel compounds in other disease models, including rhabdomyorsarcoma (tumors of the skeletal muscles). They also are making derivatives of several compounds to further increase effectiveness. Some of these new derivatives are also presented the same annual meeting of the AACR today.

"Although we feel we have made a great discovery towards establishing a novel targeted therapy, we are far from our ultimate goal of using this in humans," Üren says.

Other investigators on this work include Jared T. Murdoch, Sung-Hyeok Hong, Gulay Bulut, George W. Kosturko, Lauren E. Drebing, and Jeffrey A. Toretsky, all of Georgetown Lombardi. The authors also wish to thank Milton Brown and Mikell A. Paige for their contributions.

The study has been funded by a peer reviewed grant support from The Children's Cancer Foundation® of Baltimore, Md. and the Department of Defense.

Üren, Bulut, Kosturko, Toretsky, Brown and Paige are named as co- inventors on a patent application that has been filed by Georgetown University related to technology described in this abstract.

http://www.bbc.co.uk/news/world-africa-17589445

Uganda's nodding disease: 'I've lost hope'

In the north of Uganda, thousands of children have fallen ill with a fatal, incurable disease known as nodding disease.

Communities are starting to panic and some people are losing hope as the medical community struggles to either find a cause or a cure, as the BBC's Will Ross reports.

Driving through villages of Uganda's northern Kitgum district it is staggering how many families have been affected by the debilitating nodding syndrome. In Tumangu village it is like a plague.

Francis Anywar sits in front of the grass thatched home. The 15-year-old is naked and appears to be in another world - never speaking. The scars on his head point to the violent seizures that strike several times a day. Francis's health started deteriorating eight years ago and both his physical and mental development have steadily been eroded.

Tied to a tree

A few hundred metres further down the dirt road a mother tells me something I will never forget.

"I've lost hope. I'm just taking care of Sarah and Moses like flowers in the home knowing they are of no use in the future," Betty Olana says. Because of the neurological syndrome, her 14-year-old daughter and 12-year-old son look much younger and need round-the-clock care. They cannot wash themselves or get dressed without help. "When I go off to farm I tie them to the tree so they don't get injured.

"If they walk off they don't know where they are going they just keep walking and get lost."

One nurse told me that when the children develop psychosis and wander off into the bush they often do not return home. She said hunger can then kill them and mentioned incidents of dogs carrying the bodies of dead children back to a village.

When Ms Olana brings out a tray of millet porridge and stew, Sarah and Moses wash their hands and tuck in. But within minutes Moses's head starts to drop, his eyes close and as if in a trance he appears to be drifting off into another world. Food, heat and cold weather are the triggers.

There are more than 3,000 reported cases in northern Uganda and the government has now set up nodding syndrome screening centres at health centres. However, due to deep poverty, many families cannot afford to pay for the transport. Others like Ms Olana have simply given up on their children.

Ojok Samuel sits at the clinic staring into space. He is nine but looks three or four. "He started nodding his head at the age of two whenever we gave him food," his father John Oboda says, adding that the stress on the family has affected his own health. Unable to farm, because he always has to care for the children, the family is slipping deeper and deeper into poverty.

Stigma

He has also brought his 10-year-old daughter Jaqueline Amony who now suffers regular seizures, which have affected her mental ability. Neither of these children are going to school and most worrying for Ojok is that they have not shown any sign of improvement since beginning medication to control seizures a year ago.

There is also stigma. "Neighbours don't allow their children to interact with Samuel and Jaqueline. Our family is now isolated." I am told some motorbike taxis have been refusing to carry affected children.

Cases were seen in Tanzania back in the 1960s and in recent years in South Sudan. Despite its devastating impact, health officials have been left baffled as they try to find the cause and the cure.

Analysis
The cause of nodding disease, which causes wastage of the brain tissue, abnormal brain waves and seizures, remains unknown.
One theory is that it is connected to infection with a parasitic worm carried by the black fly - already known to cause river blindness. ***In an outbreak in 2004, more than nine out of 10 children who showed symptoms were found to be harbouring the parasite.***
It is possible that infection with the parasite triggers the immune system to over-react, and to inflict damage on the brain and nervous system. ***However, scientists have been unable to explain why Nodding disease does not occur in some areas where the worm is common.***
Other theories have linked the condition to exposure to toxic chemicals, contaminated meat, a lack of a form of vitamin B and psychiatric problems.

Some of the symptoms are similar to epilepsy and health workers prescribe the anti-convulsant medicine carbamazepine, which is used to manage epileptic seizures.

In northern Uganda they have just started trying out a new drug, sodium valproate, which is also used to treat epilepsy. The medical staff point out that when brought to hospital and given the medication some children improve and suffer less frequent seizures. But, sadly, by this stage the damage to the child's development has already been done and cannot be reversed.

In November 2009, the Ugandan government requested help from the US-based Centers For Disease Control and Prevention. CDC experts have visited the affected area and taken away samples to test but they are struggling to find the underlying cause.

"The CDC teams documented for the first time that Nodding Syndrome is a novel complex epilepsy syndrome, and that the head nodding was a direct manifestation of seizures that cause a brief lapse in muscle tone due to alterations in brain function," the public health organisation says.

"Children living under the poorest conditions where there is not enough food, pure water, or decent housing seem most susceptible to this condition."

The last time I was in Kitgum hospital in 2004 it was a nightly refuge for thousands of children who could not sleep at home for fear of abduction by the Lord's Resistance Army rebels. Northern Uganda is now peaceful but the families and health workers tell me that they feel like they are fighting a new war.

"We are getting stressed dealing with this traumatic situation. You see new cases every day - children having fits, others falling down. As a human being you feel the pain," says Adong Josephine, a psychiatric nurse in Kitgum hospital. "Even me I have started to develop stress on seeing these patients. You don't know what really causes the problem," Ms Josephine says.

"You put yourself into the shoes of these parents. Supposing these were my children; what if the problem comes to my family; where will I be? Where will I go?"

"So many have died and others are still at the critical stages of dying because when you look at them - some are too wasted unable to eat unable to walk. If they are not brought to the hospital they are most likely to die in some days to come," she adds.

On the ward are two badly burnt children. The cooking fire at home is a death trap for the affected children because when a seizure strikes they are unable to move away from the flames.

Outside at the screening centre 13-year-old Augustin Oola goes into spasms as a nurse tries to get him to stand up. He only appears moderately comfortable when he is allowed to flop on the floor where he sits silently.

With no known cause or cure speculation is rife. "It could be an effect of the war. There were many deaths around this area - many people were killed. So maybe it is their spirit which is making these children sick," suggests Ms Olana, whose home is in an area the LRA rebels used as a base. "We see many people coming now to try to investigate. So we have a little bit of hope. We are praying that these children will be cured."

http://blogs.scientificamerican.com/guest-blog/2012/04/03/are-dna-patents-doomed/

Are DNA Patents Doomed?

DNA is special. Unlike other body parts, it holds information. Even discarding a blood spot or saliva sample doesn't necessarily prevent the telltale DNA sequences from living on in a database.

By Ricki Lewis | April 3, 2012

We guard our DNA data in a way that we don't other test results, such as cholesterol levels. "Genes are uniquely 'ours.' They say something about us at some fundamental level, more than a mammogram or a Pap smear or an x-ray," said James Evans, MD PhD, professor of genetics at the University of North Carolina, Chapel Hill, at a symposium on DNA patenting at the International Congress of Human Genetics in Montreal in October 2011.

Our emotional attachment to our genomes may be part of why the patentability of the breast and ovarian cancer susceptibility genes BRCA1 and BRCA2 has been pinging from court to court for years. The latest chapter: on March 26, the U.S. Supreme Court asked the U.S. Court of Appeals for the Federal Circuit, which had upheld the Myriad Genetics and University of Utah's patents on the two genes, to reconsider.

This move, many think, stemmed from the March 20 invalidation of Prometheus Laboratories patents on measuring levels of a metabolite to assess whether a dosage of 6-mercaptopurine to treat inflammatory bowel disease is too low to work or high enough to cause adverse effects, procedures that the court found "add nothing specific to the laws of nature other than what is well-understood, routine, conventional activity." Patents for cancer genes useful as a diagnostic may seem to have little to do with measuring a metabolite to adjust a dosage, but, at the risk of evoking a double negative, they share a lack of "non-obviousness," one of the key requirements of a patentable invention.

Cervixes and Spleens Led the Way

The two most famous cases of body parts exploited for profit – Henrietta Lacks's wild cancer cells and John Moore's celebrated spleen – can't match the power in the 3-billion-bit identifier that is a human genome.

HeLa cells originated in the cervix of a poor, uneducated African-American woman. In 1951 Henrietta Lacks's unusually prolific cells were sampled, cultured, and sent to labs all over the world, without her or her family's knowledge.

John Moore gave up his swollen spleen in 1976 to treat his leukemia, unaware that his physician, hospital, and a biotech company would patent the cells and sell an unusual protein that they produced. Moore sued, but the California Supreme Court ruled against him, finding that removed cells are not the equivalent nor the product of a person.

A Brief History of DNA Patents

The U.S. Patent Act, passed in 1790, defined a patentable invention as novel, useful, and non-obvious to an expert in the field. It's easy to see how a patent might apply to a self-flushing toilet or an electronic gadget, but the picture gets murky on the matter of DNA.

One can't patent ideas, laws of nature, or products of nature. But it's been okay to isolate a chemical from nature since Parke-Davis claimed adrenaline in 1911, deeming it different outside a body.

U.S. patent law ventured into biology in 1980, with General Electric's "oil eater" bacterium that combined DNA rings from four microbes. Nature hadn't invented that. Then in 1990, the patent office added rules for claiming DNA sequences. Within a year, Amgen patented the first gene, erythropoietin (EPO), used to treat anemia. The European Union declared genes patentable in 1998.

Isolating a gene supposedly renders it patentable, for it is no longer a "product of nature," separate from its chromosome, its non-essential parts removed. The remaining DNA becomes a novel 'composition of matter.'

Two Controversial Cases

An early gene patenting battle concerned Canavan disease, which strips brain cells of their insulating myelin coating beginning in infancy and usually lethal in childhood. The patent holders – four researchers and the University of Miami – developed a diagnostic test using the gene. Then families who had donated their children's brains to the gene researchers found themselves having to pay to test their other children. Although that case was settled when the plaintiffs ran out of funds, with the cost of the test dropped and the gene made available to researchers, it set an informal precedent for the current BRCA controversy.

Three years after the Canavan gene patent issued, in 2000, the first of Myriad Genetics' BRCA patents issued. They've triggered outrage ever since.

The American Civil Liberties Union and the Association for Molecular Pathology, representing 150,000 geneticists, cancer survivors, pathologists, and others, sued the U.S. Patent and Trademark Office (USPTO), Myriad, and the University of Utah in May 2009. On March 29, 2010, senior judge Robert W. Sweet for the

Federal District Court for the Southern District of New York ruled the patents invalid. Said he at the International Congress of Human Genetics, "A human gene is not an invention. DNA's existence in an isolated form alters neither this fundamental quality of DNA as it exists in the body nor the information it holds." In August 2011 the U.S. Court of Appeals for the Federal Circuit overturned Judge Sweet's decision, and now the March 26, 2012 Supreme Court's setting aside of that ruling for further discussion is keeping the patent ball in play. The final result, whatever it is, will be important to many, because a fifth of the 20,325 or so human genes are patented.

The Cat's Out of the Bag

It looks, at least this week, like DNA patents will become a thing of the past as consensus slowly builds that a product of nature isn't an invention after all. But another reason why such patents are headed for extinction, I think, is that DNA testing has transcended the single-gene legacy of Gregor Mendel that peaked in a crescendo of discoveries in the 1990s, giving way to the age of genomics. Genes are no longer seen as islands. Even the three most common mutations in the much-discussed BRCA genes impart susceptibility, not certainty, with actual risk reflecting influences of other genes and the environment.

Here are four ways that average people are encountering genes, without a thought to who or what entity "owns" them.

Will gene patents interfere with whole exome and whole genome sequencing? Gavin Stevens' diagnosis of Leber congenital amaurosis, which causes his blindness, would not have been possible without the sequencing of his, his parents', and his grandparents' exomes. Photo: Jennifer Stevens

Three-year-old Gavin Stevens, blind from birth, had been tested for more than a dozen genes known to cause Leber congenital amaurosis when his parents, Troy and Jennifer, met with John Chiang, PhD, director of the Molecular Diagnostic Laboratory at the Casey Eye Institute in Portland, Oregon, who sent the family's DNA samples to the Beijing Genomics Institute to have their exomes sequenced. And within this protein-encoding portion of the genome lay the answer: Gavin's mutant gene – the first stage in developing a gene therapy.

Clients of direct-to-consumer genetic testing companies are providing the numbers to accelerate discovery of the many genes that contribute to common diseases. 23andMe has already found two new Parkinson's disease, susceptibility genes, thanks to the DNA of 3,426 customers with the disease and nearly 30,000 controls.

Forensic DNA databanks track some two dozen sites of short repeated sequences in the genome that collectively vary in more ways than there are people on the planet. DNA profiling has led to hundreds of exonerations and convictions. Several states have recently expanded their forensic DNA databases.

Every few weeks, Family Tree DNA alerts me to the existence of a possible long-long-lost cousin among the many consumers sending in DNA samples. The power of tracing the deeper branches of the human family tree, of inferring when and from where and how many times some of our ancestors left Africa and spread around the globe, depends upon amassing DNA information from as many populations as possible.

Could any of these potentially life-altering uses of DNA sequencing have happened if gene patent-holders charged prohibitively high licensing fees to the companies that package their discoveries into test panels and kits? Will our courts allow patented genes to impede progress at the genomic level, like tiny private stretches of sand interrupting an extensive public beach? Because so many applications of the information in our genomes are clearly already here, I think that the idea of owning a gene is already obsolete, and the courts need to catch up with the science.

Summed up James Evans, "the human genome is a shared legacy." I couldn't agree more.

http://www.sciencedaily.com/releases/2012/04/120403135516.htm

Coral Links Ice Sheet Collapse to Ancient 'Mega Flood'

Coral off Tahiti has linked the collapse of massive ice sheets 14,600 years ago to a dramatic and rapid rise in global sea-levels of around 14 metres.

ScienceDaily - Previous research could not accurately date the sea-level rise but now an Aix-Marseille University-led team, including Oxford University scientists Alex Thomas and Gideon Henderson, has confirmed that the event occurred 14,650-14,310 years ago at the same time as a period of rapid climate change known as the Bølling warming.

The finding will help scientists currently modelling future climate change scenarios to factor in the dynamic behaviour of major ice sheets. A report of the research is published in this week's Nature.

'It is vital that we look into Earth's geological past to understand rare but high impact events, such as the collapse of giant ice sheets that occurred 14,600 years ago,' said Dr Alex Thomas of Oxford University's Department of Earth Sciences, an author of the paper. 'Our work gives a window onto an extreme event in which deglaciation coincided with a dramatic and rapid rise in global sea levels -- an ancient 'mega flood'. Sea

level rose more than ten times more quickly than it is rising now! This is an excellent test bed for climate models: if they can reproduce this extraordinary event, it will improve confidence that they can also predict future change accurately.'

During the Bølling warming high latitudes of the Northern hemisphere warmed as much as 15 degrees Celsius in a few tens of decades. The team has used dating evidence from Tahitian corals to constrain the sea level rise to within a period of 350 years, although the actual rise may well have occurred much more quickly and would have been distributed unevenly around the world's shorelines.

Dr Thomas said: 'The Tahitian coral is important because samples, thousands of years old, can be dated to within plus or minus 30 years. Because Tahiti is an ocean island, far away from major ice sheets, sea-level evidence from its coral reefs gives us close to the 'magic' average of sea levels across the globe, it is also subsiding into the ocean at a steady pace that we can easily adjust for.'

The research is part of a large international consortium, the Integrated Ocean Drilling Program (IODP), and the coral samples were obtained by drilling down to the sea floor from a ship positioned off the coast of Tahiti.

What exactly caused the Bølling warming is a matter of intense debate: a leading theory is that the ocean's circulation changed so that more heat was transported into Northern latitudes.

The new sea-level evidence suggests that a considerable portion of the water causing the sea-level rise at this time must have come from melting of the ice sheets in Antarctica, which sent a 'pulse' of freshwater around the globe. However, whether the freshwater pulse helped to warm the climate or was a result of an already warming world remains unclear.

Pierre Deschamps, Nicolas Durand, Edouard Bard, Bruno Hamelin, Gilbert Camoin, Alexander L. Thomas, Gideon M. Henderson, Jun'ichi Okuno, Yusuke Yokoyama. Ice-sheet collapse and sea-level rise at the Bølling warming 14,600 years ago. Nature, 2012; 483 (7391): 559 DOI: 10.1038/nature10902

http://www.bbc.co.uk/nature/17525070

Woolly mammoth carcass may have been cut into by humans

The discovery of a well-preserved juvenile woolly mammoth suggests that ancient humans "stole" mammoths from hunting lions, scientists say.

By Ben Aviss Reporter, BBC Nature

Bernard Buigues of the Mammuthus organisation acquired the frozen mammoth from tusk hunters in Siberia.

Scientists completed an initial assessment of the animal, known as Yuka, in March this year.

Wounds indicate that both lions and humans may have been involved in the ancient animal's death.

"Already there is dramatic evidence of a life-and-death struggle between Yuka and some top predator, probably a lion," says leading mammoth expert, Daniel Fisher, professor of earth and environmental sciences at the University of Michigan. "Even more interesting, there are hints that humans may have taken over the kill at an early stage."

If further investigation by Mr Buigues, Professor Fisher and fellow scientists at the Sakha Academy of Sciences in Yakutsk confirms this analysis, it will be the first carcass to show signs of interaction with ancient humans found in this part of the world. The Yuka mammoth was filmed as part of the BBC/Discovery Co-Production programme Woolly Mammoth: Secrets from the Ice.

By analysing the teeth and tusks, the team estimate Yuka was about two and a half years old when it died.

Teeth, tusks and bone are the most common ways extinct animals such as mammoths are studied, as these parts of the body take a relatively long time to decompose. Soft tissues such as muscle, skin and internal organs decompose far quicker, and are very rarely found on old carcasses. This means that vital information is usually lost. But much of Yuka's soft tissue as well as its woolly coat has remained intact, well-preserved in its icy tomb for possibly more than 10,000 years.

Kevin Campbell, associate professor of environmental and evolutionary physiology at the University of Manitoba said: "These are remarkably rare finds and have huge significance." One of the most striking things about Yuka is its strawberry-blonde hair, he said. The possibility of mammoths having lighter coat colours was proposed in 2006 after scientists studied the genes extracted solely from a mammoth bone. Yuka provides direct evidence that mammoths did have lighter-coloured coats.

Associate Professor Campbell said the find "will be a boon to researchers as it will help them link observed phenotypes (morphological features that we can see) with genotype (DNA sequences)". These links will allow scientists to determine how widespread physical traits such as eye and hair colour were "within and among mammoth populations" simply from studying genes from bone or hair samples in the future.

Professor Fisher agreed the find was extraordinary: "It's like a diary or journal someone has just handed you - you just haven't had a chance to read it."

Lion attacks

Healed scratches found on the skin indicate a lion attack that Yuka survived earlier in its relatively short life.

However, similar deep cuts that had not healed suggest a subsequent lion attack that either caused or happened very near the time of Yuka's death. Also, when moving one of Yuka's legs, Professor Fisher recognised evidence of a freshly broken leg when it died and suggested this may have occurred as Yuka tried to flee from attackers. The lions in question (Panthera leo spelea) are an extinct subspecies of the African lion, known commonly as Eurasian cave lions but were present at the same time as the mammoths.

"Did we know lions hunted mammoths? Well, we guessed they did. But could we ever have expected to see such graphic evidence? No - but here it is," explained Professor Fisher.

In modern-day Africa, young elephants are attacked by lions, providing a means of comparing their injuries with Yuka's. Lions will usually enter the carcass through the belly, clamp their teeth over the mouth in order to suffocate their prey, and chew at an elephant's muscular trunk. However, Yuka's trunk is not damaged and there is only slight damage to the hide around the face.

Instead of entering Yuka's body via the belly, there is what Professor Fisher describes as "a bizarre set of damage on the hide". This includes a "long, straight cut that stretches from the head to the centre of the back" as well as "very unusual patterned openings" into the skin and "scalloped margins" on the upper right-hand flank. The skull, spine, ribs and pelvis were all removed from Yuka's body, but the skull and pelvis were found nearby. However, most of the spine and three-quarters of the ribs are missing.

Human interference

Each scalloped mark on the skin is made up by 15-30 small, serrations that "could be the saw-like motion of a human tool" and there are "some quite striking cut marks" on the leg bones, according to Professor Fisher.

Prof Fisher said they had questioned whether the cuts could have been made more recently. "We asked the people who found this mammoth multiple times if they had done this. They replied 'No! We did not get our knives out' which suggests we're looking at some sort of interaction of humans, mammoths and lions.

"Were humans using the lions to catch mammoths and then moving the lions off their kill... was that what happened? I don't know but I wouldn't have thought about it without seeing it [the evidence]."

Supporting this argument, the Dorobo tribe still practise the art of stealing kills from lions in Kenya.

"Each new specimen has something to teach us, but Yuka provides some of the most dramatic evidence yet available for events surrounding the death of a woolly mammoth on the arctic steppes of Siberia."

Professor Alice Roberts was part of the film crew that followed Yuka being recovered from the tusk hunters.

She said: "There are some odd things. What we need to do is find out if this was human interference near the time of death or was it something that happened much later? "If it happened near the time of death then it means Yuka is a very important specimen as there are not many [mammoths] that show human interactions." She said seeing Yuka in the flesh was almost "poignant". "You feel it has only just died as it is so beautifully preserved."

http://www.eurekalert.org/pub_releases/2012-04/ind-ang040412.php

A new gene thought to be the cause in early-onset forms of Alzheimer's disease

A new gene that causes early-onset of Alzheimer's disease has been discovered by the research team of Dominique Campion at the Insert unit 1079 "Genetics of cancer and neuropsychiatric diseases" in Rouen.

The research scientists showed that in the families of 5 of 14 patients suffering from the disease, mutations were detected on the gene SORL1. This gene regulates the production of a peptide involved in Alzheimer's disease. The results of this study have been published in the review Molecular Psychiatry issued April 3rd.

Precise genetic mutations have been seen to play a part in early-onset forms of Alzheimer's disease. However, there is a sub-population of patients in whom there is no mutation of these genes. So how can these patients, in whom there are no pre-established mutations, be suffering from early-onset Alzheimer's?

To reply to this question, the research team working under the leadership of Dominique Campion and Didier Hannequin (Inserm unit 1079 and Centre national de référence malades Alzheimer jeunes, University hospital Rouen), studied the genes from 130 families suffering from early-onset forms of Alzheimer's disease. These families were identified by 23 French hospital teams within the framework of the "Alzheimer Plan". Of these families, 116 presented mutations on the already known genes. But in the 14 remaining families, there was no mutation at all observed on these genes.

A study of the genome of the 14 families using new whole DNA sequencing techniques showed evidence of mutations on a new SORL1 gene. The SORL1 gene is a coding gene for a protein involved in the production of the beta-amyloid peptide. This protein is known to affect the functioning of the brain cells (see insert).

Two of the identified mutations are responsible for an under-expression of SORL1, resulting in an increase in the production of the beta-amyloid peptide. "The mutations observed on SORL1 seem to contribute to the development of early-onset Alzheimer's disease. However, we still need to identify more clearly the way in which these mutations are transmitted on the SORL1 gene within families" states Dominique Campion.

Alzheimer's disease is one of the main causes of dependency among the elderly. It results from neuron degradation in different areas of the brain. Its symptoms include increased alterations to memory, cognitive functions and behaviour disorders that lead to a progressive loss of independence.

Alzheimer's disease is characterized by the development of two types of lesion in the brain: amyloid plaques and neurofibrillary degenerescence. Amyloid plaques are caused by extracellular accumulation of a peptide, beta-amyloid peptide (Aβ) in specific areas of the brain. Neurofibrillary degenerescences are intraneuronal lesions caused by abnormal filamentary aggregation of a protein known as a Tau protein.

High frequency of potentially pathogenic SORL1 mutations in autosomal dominant early-onset Alzheimer disease Molecular Psychiatry, April 3, doi: 10.1038/mp.2012.15

http://www.eurekalert.org/pub_releases/2012-04/uota-auo040412.php

A University of Tennessee professor's hypothesis may be game changer for evolutionary theory

A new hypothesis counters popular evolutionary thinking that living organisms evolve by adding genes rather than discarding them

A new hypothesis posed by a University of Tennessee, Knoxville, associate professor and colleagues could be a game changer in the evolution arena. The hypothesis suggests some species are surviving by discarding genes and depending on other species to play their hand.

The groundbreaking "Black Queen Hypothesis" got its name from the game of Hearts.

In Hearts, the goal is to avoid "winning" the Queen of Spades (the Black Queen), which is worth a lot of points. Subsequently, players allow others to take the high-point card while they enjoy low-score tallies.

This same premise applies in evolution, the scientists say.

According to the hypothesis, evolution pushes microorganisms to lose essential functions when there is another species around to perform them. This idea counters popular evolutionary thinking that living organisms evolve by adding genes rather than discarding them.

"A common assumption about evolution is that it is directed toward increasing complexity," said Erik Zinser, associate professor of microbiology. "But we know from analysis of microbial genomes that some lineages trend toward decreasing complexity, exhibiting a net loss of genes relative to their ancestor."

Zinser's opinion piece is published in mBio, the online open-access journal of the American Society for Microbiology. Jeffrey Morris and Richard Lenski of Michigan State University are co-authors. Morris was Zinser's doctoral student at UT.

The authors formed their theory after studying photosynthetic bacteria called Prochlorococcus.

"This marine microorganism continued to mystify us because it is the most common photosynthetic organism on Earth, but it is extremely difficult to grow in pure culture," Zinser said. "A major reason for this difficulty is that Prochlorococcus is very sensitive to reactive oxygen species such as hydrogen peroxide and relies on other bacteria to protect them by breaking down these toxic substances for them."

Prochlorococcus had once performed this function itself, but natural selection decided it was too costly, like carrying the Queen of Spades, and discarded this ability. Instead Prochlorococcus benefits from the hard work of others within its community allowing it to concentrate its energies elsewhere—such as multiplying.

The hypothesis offers a new way of looking at complicated, interdependent communities of microorganisms.

"We know that certain microbial activities, such as hydrogen peroxide scavenging, are 'leaky,' meaning their impacts extend beyond the cell and into the environment," Zinser said. "What the hypothesis suggests is that this leakiness can drive a community toward greater interdependence, even if some members are unwitting participants in this process."

This interdependence could lend itself to vulnerabilities. The scientists say the work highlights the importance of biological diversity, because if rare members are lost, "the consequences for the community could be disastrous." This would be analogous to attempting to play Hearts without the Queen of Spades.

Currently, the hypothesis is limited to microorganisms, but Zinser thinks the hypothesis could be extended to larger free-living organisms. All that is needed is a card which no player wants yet is crucial for the game to be played.

http://www.eurekalert.org/pub_releases/2012-04/aaon-smd032712.php

Studies: Memory declines faster in years closest to death; mental activity best protection

New research finds that a person's memory declines at a faster rate in the two- and-a-half years before death than at any other time after memory problems first begin.

ST. PAUL, Minn. - A second study shows that keeping mentally fit through board games or reading may be the best way to preserve memory during late life. Both studies are published in the April 4, 2012, online issue of Neurology®, the medical journal of the American Academy of Neurology.

For the study, 174 Catholic priests, nuns and monks without memory problems had their memory tested yearly for six to 15 years before death. After death, scientists examined their brains for hallmarks of Alzheimer's disease called plaques and tangles. "In our first study, we used the end of life as a reference point for research on memory decline rather than birth or the start of the study," said study author Robert S. Wilson, PhD, of Rush University Medical Center in Chicago.

The study found that at an average of about two-and-a-half years before death, different memory and thinking abilities tended to decline together at rates that were 8 to 17 times faster than before this terminal period. Higher levels of plaques and tangles were linked to an earlier onset of this terminal period but not to rate of memory decline during it.

In an accompanying editorial, author Hiroko H. Dodge, PhD, with Oregon Health and Science University in Portland and a member of the American Academy of Neurology, noted, "The findings suggest that the changes in mental abilities during the two to three years before death are not driven directly by processes related to Alzheimer's disease, but instead that the memory and other cognitive decline may involve some biological changes in the brain specific to the end of life. The study by Wilson and his co-authors deepens our understanding of terminal cognitive decline."

The second study, also conducted by Wilson, focused on mental activities and involved 1,076 people with an average age of 80 who were free of dementia. Participants underwent yearly memory exams for about five years. They reported how often they read the newspaper, wrote letters, visited a library and played board games such as chess or checkers. Frequency of these mental activities was rated on a scale of one to five, one meaning once a year or less and five representing every day or almost every day.

The results showed that people's participation in mentally stimulating activities and their mental functioning declined at similar rates over the years. The researchers also found that they could predict participants' level of cognitive functioning by looking at their level of mental activity the year before but that level of cognitive functioning did not predict later mental activity. "The results suggest a cause and effect relationship: that being mentally active leads to better cognitive health in old age," said Wilson.

The studies were supported by the National Institute on Aging and the Illinois Department of Health.

http://www.eurekalert.org/pub_releases/2012-04/iop-hpf040212.php

Handheld plasma flashlight rids skin of notorious pathogens

A group of Chinese and Australian scientists have developed a handheld, battery-powered plasma-producing device that can rid skin of bacteria in an instant.

The device could be used in ambulance emergency calls, natural disaster sites, military combat operations and many other instances where treatment is required in remote locations. The plasma flashlight, presented today, 5 April, in IOP Publishing's Journal of Physics D: Applied Physics is driven by a 12 V battery and doesn't require any external generator or wall power; it also doesn't require any external gas feed or handling system. In the experiment, the plasma flashlight effectively inactivated a thick biofilm of one of the most antibiotic- and heat-resistant bacteria, Enterococcus faecalis – a bacterium which often infects the root canals during dental treatments.

The biofilms were created by incubating the bacteria for seven days. The biofilms were around 25 micrometres thick and consisted of 17 different layers of bacteria. Each one was treated for five minutes with the plasma flashlight and then analysed to see how much of the bacteria survived. Results showed that the plasma not only inactivated the top layer of cells, but penetrated deep into the very bottom of the layers to kill the bacteria.

Co-author of the study, Professor Kostya (Ken) Ostrikov, from the Plasma Nanoscience Centre Australia, CSIRO Materials Science and Engineering, said: "The bacteria form thick biofilms, which makes them enormously resistant against inactivation which is extremely difficult to implement. High temperatures are commonly used but they would obviously burn our skin.

"In this study we chose an extreme example to demonstrate that the plasma flashlight can be very effective even at room temperature. For individual bacteria, the inactivation time could be just tens of seconds."

Plasma – the fourth state of matter in addition to solids, liquids and gases – has previously shown its worth in the medical industry by effectively killing bacteria and viruses on the surface of the skin and in water.

Although the exact mechanism behind the anti-bacterial effect of plasma is largely unknown, it is thought that reactions between the plasma and the air surrounding it create a cocktail of reactive species that are similar to the ones found in our own immune system.

The researchers ran an analysis to see what species were present in the plasma and found that highly-reactive nitrogen- and oxygen-related species dominated the results. Ultraviolet radiation has also been theorised as a reason behind plasma's success; however, this was shown to be low in the jet created by the plasma flashlight, adding to the safety aspect of the device. The temperature of the plume of plasma in the experiments was between 20-230C, which is very close to room temperature and therefore prevents any damage to the skin. The device itself is fitted with resistors to stop it heating up and making it safe to touch.

"The device can be easily made and costs less than 100 US dollars to produce. Of course, some miniaturisation and engineering design may be needed to make it more appealing and ready for commercialisation," Ostrikov continued. The device was created by an international team of researchers from Huazhong University of Science and Technology, CSIRO Materials Science and Engineering, The University of Sydney and the City University of Hong Kong.

From Thursday 5 April, this paper can be downloaded from http://iopscience.iop.org/0022-3727/45/16/165205

http://www.eurekalert.org/pub_releases/2012-04/uoea-efp032812.php

Eating flavonoids protects men against Parkinson's disease

Men who eat flavonoid-rich foods such as berries, tea, apples and red wine significantly reduce their risk of developing Parkinson's disease, according to new research by Harvard University and the University of East Anglia (UEA).

Published today in the journal Neurology ®, the findings add to the growing body of evidence that regular consumption of some flavonoids can have a marked effect on human health. Recent studies have shown that these compounds can offer protection against a wide range of diseases including heart disease, hypertension, some cancers and dementia. This latest study is the first study in humans to show that flavonoids can protect neurons against diseases of the brain such as Parkinson's.

Around 130,000 men and women took part in the research. More than 800 had developed Parkinson's disease within 20 years of follow-up. After a detailed analysis of their diets and adjusting for age and lifestyle, male participants who ate the most flavonoids were shown to be 40 per cent less likely to develop the disease than those who ate the least. No similar link was found for total flavonoid intake in women.

The research was led by Dr Xiang Gao of Harvard School of Public Health in collaboration with Prof Aedin Cassidy of the Department of Nutrition, Norwich Medical School at UEA. "These exciting findings provide further confirmation that regular consumption of flavonoids can have potential health benefits," said Prof Cassidy. "This is the first study in humans to look at the associations between the range of flavonoids in the diet and the risk of developing Parkinson's disease and our findings suggest that a sub-class of flavonoids called anthocyanins may have neuroprotective effects."

Prof Gao said: "Interestingly, anthocyanins and berry fruits, which are rich in anthocyanins, seem to be associated with a lower risk of Parkinson's disease in pooled analyses. Participants who consumed one or more portions of berry fruits each week were around 25 per cent less likely to develop Parkinson's disease, relative to those who did not eat berry fruits. Given the other potential health effects of berry fruits, such as lowering risk of hypertension as reported in our previous studies, it is good to regularly add these fruits to your diet."

Flavonoids are a group of naturally occurring, bioactive compunds found in many plant-based foods and drinks. In this study the main protective effect was from higher intake of anthocyanins, which are present in berries and other fruits and vegetables including aubergines, blackcurrants and blackberries. Those who consumed the most anthocyanins had a 24 per cent reduction in risk of developing Parkinson's disease and strawberries and blueberries were the top two sources in the US diet.

The findings must now be confirmed by other large epidemiological studies and clinical trials.

Parkinson's disease is a progresssive neurological condition affecting one in 500 people, which equates to 127,000 people in the UK. There are few effective drug therapies available.

Dr Kieran Breen, director of research at Parkinson's UK said: "This study raises lots of interesting questions about how diet may influence our risk of Parkinson's and we welcome any new research that could potentially lead to prevention. "While these new results look interesting there are still a lot of questions to answer and much more research to do before we really know how important diet might be for people with Parkinson's."

'Habitual intake of dietary flavonoids and risk of Parkinson's disease' by X Gao (Harvard), A Cassidy (UEA), M Schwarzschild (Massachusetts General Hospital), E Rimm (Harvard) and A Ascherio (Harvard) is published on April 4 by Neurology ® – the medical journal of the American Academy of Neurology.

http://www.eurekalert.org/pub_releases/2012-04/iofs-gif040412.php

Glycemic index foods at breakfast can control blood sugar throughout the day

Eating foods at breakfast that have a low glycemic index may help prevent a spike in blood sugar throughout the morning and after the next meal of the day, researchers said at the Institute of Food Technologists' Wellness 12 meeting.

These breakfast foods also can increase feelings of satiety and fullness and may make people less likely to overeat throughout the day, acdcording to presentations Wednesday by Kantha Shelke, Ph.D., principal, Corvus Blue LLC, and Richard Mattes, M.P.H., R.D., distinguished professor of foods and nutrition at Purdue University.

The glycemic index ranks foods on the extent to which they raise blood sugar levels after eating. Foods with a high index are rapidly digested and result in high fluctuations in blood sugar levels. Foods with a low glycemic index produce gradual rises in blood sugar and insulin levels and are considered healthier, especially for people with diabetes.

Mattes' research specifically focused on the advantages of having almonds, a low glycemic index food, with the morning meal. In his study, published last year in the Journal of Nutrition and Metabolism, participants who ate a breakfast containing whole almonds experienced longer feelings of fullness and had lower blood glucose concentrations after breakfast and lunch, compared to those who did not have a low-glycemic breakfast.

When a low glycemic food is added to the diet, people spontaneously choose to eat less at other times throughout the day. Mattes added that while the calories need to be taken into consideration as part of a person's overall diet, almonds can be incorporated in moderate amounts without an effect on body weight.

Both Mattes and Shelke stressed the importance of eating a healthy, low-glycemic breakfast in maintaining a healthy weight and blood sugar levels. A 2009 study found that about 30 percent of people skip breakfast one to three times per week. Among those who eat breakfast, cold cereal is the most popular (83 percent), followed by eggs (71 percent). In addition to low glycemic index, Dr. Shelke said the ideal breakfast for consumers has these attributes:

Savory	***Satiates quickly so less is consumed***
Portable	***Affordable for the whole family to eat every day***
Pleasing texture	***Non-fried***
Fills you up for extended periods of time	***Delicious without making you feeling guilty***

"This is a very tall order for food product manufacturers," Shelke said. "It takes a lot of skill and understanding."

While it may present challenges for food manufacturers, it is well worth it to develop these products because of the prevalence of diabetes and pre-diabetes in the United States and beyond. It is estimated that by 2030, more than 16 percent of the global population will have a blood sugar problem.

"Most of the risk factors are things that can be managed and modified," Shelke said. "We can reverse pre-diabetes and prevent it from becoming diabetes. Food has become the reason for what's ailing us, but it can actually be a solution in a number of different ways."

http://www.eurekalert.org/pub_releases/2012-04/niom-sqg040412.php

Spontaneous gene glitches linked to autism risk with older dads

Non-inherited mutations spotlight role of environment – NIH-supported study, consortium

Researchers have turned up a new clue to the workings of a possible environmental factor in autism spectrum disorders (ASDs): fathers were four times more likely than mothers to transmit tiny, spontaneous mutations to their children with the disorders. Moreover, the number of such transmitted genetic glitches increased with paternal age. The discovery may help to explain earlier evidence linking autism risk to older fathers.

The results are among several from a trio of new studies, supported in part by the National Institutes of Health, finding that such sequence changes in parts of genes that code for proteins play a significant role in ASDs. One of the studies determined that having such glitches boosts a child's risk of developing autism five to 20 fold.

Taken together, the three studies represent the largest effort of its kind, drawing upon samples from 549 families to maximize statistical power. They reveal sporadic mutations widely distributed across the genome,

sometimes conferring risk and sometimes not. While the changes identified don't account for most cases of illness, they are providing clues to the biology of what are likely multiple syndromes along the autism spectrum.

"These results confirm that it's not necessarily the size of a genetic anomaly that confers risk, but its location – specifically in biochemical pathways involved in brain development and neural connections. Ultimately, it's this kind of knowledge that will yield potential targets for new treatments," explained Thomas, R. Insel, M.D., director of the NIH's National Institute of Mental Health (NIMH), which funded one of the studies and fostered development of the Autism Sequencing Consortium, of which all three groups are members.

Multi-site research teams led by Mark Daly, Ph.D., of the Harvard/MIT Broad Institute, Cambridge, Mass., Matthew State, M.D., Ph.D., of Yale University, New Haven, Conn., and Evan Eichler, Ph.D., of the University of Washington, Seattle, report on their findings online April 4, 2012 in the journal Nature.

The study by Daly and colleagues was supported by NIMH – including funding under the American Recovery and Reinvestment Act. The State and Eichler studies were primarily supported by the Simons Foundation Autism Research Initiative. The studies also acknowledge the NIH's National Human Genome Research Institute, National Heart Lung and Blood Institute, and National Institute on Child Health and Human Development and other NIH components.

All three teams sequenced the protein coding parts of genes in parents and an affected child - mostly in families with only one member touched by autism. One study also included comparisons with healthy siblings. Although these protein-coding areas represent only about 1.5 percent of the genome, they harbor 85 percent of disease-causing mutations. This strategy optimized the odds for detecting the few spontaneous errors in genetic transmission that confer autism risk from the "background noise" generated by the many more benign mutations.

Like larger deletions and duplications of genetic material previously implicated in autism and schizophrenia, the tiny point mutations identified in the current studies are typically not inherited in the conventional sense – they are not part of parents' DNA, but become part of the child's DNA. Most people have many such glitches and suffer no ill effects from them. But evidence is building that such mutations can increase risk for autism if they occur in pathways that disrupt brain development. State's team found that 14 percent of people with autism studied had suspect mutations – five times the normal rate. Eichler and colleagues traced 39 percent of such mutations likely to confer risk to a biological pathway known to be important for communications in the brain.

Although Daly and colleagues found evidence for only a modest role of the chance mutations in autism, those pinpointed were biologically related to each other and to genes previously implicated in autism.

The Eichler team turned up clues to how environmental factors might influence genetics. The high turnover in a male's sperm cells across the lifespan increases the chance for errors to occur in the genetic translation process. These can be passed-on to the offspring's DNA, even though they are not present in the father's DNA. This risk may worsen with aging. The researchers discovered a four-fold marked paternal bias in the origins of 51 spontaneous mutations in coding areas of genes that was positively correlated with increasing age of the father. So such spontaneous mutations could account for findings of an earlier study that found fathers of boys with autism were six times - and of girls 17 times - more likely to be in their 40's than their 20's.

"We now have a path forward to capture a great part of the genetic variability in autism - even to the point of being able to predict how many mutations in coding regions of a gene would be needed to account for illness," said Thomas Lehner, Ph.D., chief of the NIMH Genomics Research Branch, which funded the Daly study and helped to create the Autism Sequencing Consortium. "These studies begin to tell a more comprehensive story about the molecular underpinnings of autism that integrates previously disparate pieces of evidence."

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http://www.eurekalert.org/pub_releases/2012-04/uoma-tp5040412.php

Thawing permafrost 50 million years ago led to global warming events

Researchers propose new mechanism of past global warming

AMHERST, Mass. – In a new study reported in Nature, climate scientist Rob DeConto of the University of Massachusetts Amherst and colleagues elsewhere propose a simple new mechanism to explain the source of carbon that fed a series of extreme warming events about 55 million years ago, the Paleocene-Eocene Thermal Maximum (PETM), and a sequence of similar, smaller warming events afterward.

"The standard hypothesis has been that the source of carbon was in the ocean, in the form of frozen methane gas in ocean-floor sediments," DeConto says. "We are instead ascribing the carbon source to the continents, in polar latitudes where permafrost can store massive amounts of carbon that can be released as CO2 when the permafrost thaws."

The new view is supported by calculations estimating interactions of variables such as greenhouse gas levels, changes in the Earth's tilt and orbit, ancient distributions of vegetation, and carbon stored in rocks and in frozen soil.

While the amounts of carbon involved in the ancient soil-thaw scenarios was likely much greater than today, implications of the study appear dire for the long-term future as polar permafrost carbon deposits have begun to thaw due to burning fossil-fuels, DeConto adds. "Similar dynamics are at play today. Global warming is degrading permafrost in the north polar regions, thawing frozen organic matter, which will decay to release CO2 and methane into the atmosphere. This will only exacerbate future warming in a positive feedback loop."

He and colleagues at Yale, the University of Colorado, Penn State, the University of Urbino, Italy, and the University of Sheffield, U.K., designed an accurate model—elusive up to now—to satisfactorily account for the source, magnitude and timing of carbon release at the PETM and subsequent very warm periods, which now appear to have been triggered by changes in the Earth's orbit.

Earth's atmospheric temperature is a result of energy input from the sun minus what escapes back to space. Carbon dioxide in the atmosphere absorbs and traps heat that would otherwise return to space. The PETM was accompanied by a massive carbon input to the atmosphere, with ocean acidification, and was characterized by a global temperature rise of about 5 degrees C in a few thousand years, the researchers point out. Until now, it has been difficult to account for the massive amounts of carbon required to cause such dramatic global warming events.

To build the new model, DeConto's team used a new, high-precision geologic record from rocks in central Italy to show that the PETM and other hyperthermals occurred during periods when Earth's orbit around the sun was both highly eccentric (non-circular) and oblique (tilted). Orbit affects the amount, location and seasonality of solar radiation received on Earth, which in turn affects the seasons, particularly in polar latitudes, where permafrost and stored carbon can accumulate.

They then simulated climate-ecosystem-soil interactions, accounting for gradually rising greenhouse gases and polar temperatures plus the combined effects of changes in Earth orbit. Their results show that the magnitude and timing of the PETM and subsequent hyperthermals can be explained by the orbitally triggered decomposition of soil organic carbon in the circum-Arctic and Antarctica.

This massive carbon reservoir at the poles "had the potential to repeatedly release thousands of petagrams of carbon to the atmosphere-ocean system once a long-term warming threshold was reached just prior to the PETM," DeConto and colleagues say. Until now, Antarctica, which today is covered by kilometers of ice, has not been appreciated as an important player in such global carbon dynamics.

In the past, "Antarctica and high elevations of the circum-Arctic were suitable locations for massive carbon storage," they add. "During long-term warming, these environments eventually reached a climatic threshold," with permafrost thaw and the sudden release of stored soil carbon triggered during the Earth's highly eccentric orbits coupled with high tilt.

The model described in the paper also provides a mechanism that helps to explain relatively rapid recovery from hyperthermals associated with orbital extremes occurring about every 1.2 million years, which had until now been difficult.

Overall, they conclude, "an orbital-permafrost soil carbon mechanism provides a unifying model accounting for the salient features of the hyperthermals that other previously proposed mechanisms fail to explain." Further, if the analysis is correct and past extreme warm events can be attributed to permafrost loss, it implies that thawing of permafrost in similar environments observed today "will provide a substantial positive feedback to future warming."

http://www.sciencedaily.com/releases/2012/04/120404125355.htm

Potential Method to Control Obesity: Red Wine, Fruit Compound Could Help Block Fat Cell Formation

A compound found in red wine, grapes and other fruits, and similar in structure to resveratrol, is able to block cellular processes that allow fat cells to develop, opening a door to a potential method to control obesity.

by Brian Wallheimer.

ScienceDaily - A compound found in red wine, grapes and other fruits, and similar in structure to resveratrol, is able to block cellular processes that allow fat cells to develop, opening a door to a potential method to control obesity, according to a Purdue University study. Kee-Hong Kim, an assistant professor of food science, and Jung Yeon Kwon, a graduate student in Kim's laboratory, reported in this week's issue of the Journal of Biological Chemistry that the compound piceatannol blocks an immature fat cell's ability to develop and grow.

While similar in structure to resveratrol -- the compound found in red wine, grapes and peanuts that is thought to combat cancer, heart disease and neurodegenerative diseases -- piceatannol might be an important weapon against obesity. Resveratrol is converted to piceatannol in humans after consumption.

"Piceatannol actually alters the timing of gene expressions, gene functions and insulin action during adipogenesis, the process in which early stage fat cells become mature fat cells," Kim said. "In the presence of piceatannol, you can see delay or complete inhibition of adipogenesis."

Over a period of 10 days or more, immature fat cells, called preadipocytes, go through several stages to become mature fat cells, or adipocytes. "These precursor cells, even though they have not accumulated lipids, have the potential to become fat cells," Kim said. "We consider that adipogenesis is an important molecular target to delay or prevent fat cell accumulation and, hopefully, body fat mass gain."

Kim found that piceatannol binds to insulin receptors of immature fat cells in the first stage of adipogenesis, blocking insulin's ability to control cell cycles and activate genes that carry out further stages of fat cell formation. Piceatannol essentially blocks the pathways necessary for immature fat cells to mature and grow.

Piceatannol is one of several compounds being studied in Kim's laboratory for its health benefits, and it is also present in different amounts in red grape seeds and skin, blueberries, passion fruit, and other fruits.

Kim would like to confirm his current finding, which is based on a cell culture system, using an animal model of obesity. His future work would also include determining methods for protecting piceatannol from degrading so that concentrations large enough would be available in the bloodstream to stop adipogenesis or body fat gain. We need to work on improving the stability and solubility of piceatannol to create a biological effect," Kim said. *The Purdue Research Foundation funded the work.*

J. Y. Kwon, S. G. Seo, Y.-S. Heo, S. Yue, J.-X. Cheng, K. W. Lee, K.-H. Kim. Piceatannol, Natural Polyphenolic Stilbene, Inhibits Adipogenesis via Modulation of Mitotic Clonal Expansion and Insulin Receptor-dependent Insulin Signaling in Early Phase of Differentiation. Journal of Biological Chemistry, 2012; 287 (14): 11566 DOI: 10.1074/jbc.M111.259721

http://bit.ly/HogaXg

Feathers may have helped T. rex's relatives ride out a cold climate

Feathers are the defining feature of birds, but that wasn't always the case.

By John Timmer | Published 2 days ago

For millions of years, various species of dinosaurs sported feathers, some of which have left behind fossilized impressions. But for the most part, the feathers we've found have been attached to smaller dinosaurs, many of them along the lineage that gave rise to birds.

That situation was changed dramatically by a species that is described in today's issue of Nature. Three nearly complete skeletons have revealed a feathered dinosaur that its finders term "gigantic." At nearly 1,500kg and over forty times the weight of any previous feathered dinosaur, Yutyrannus huali was a beast—almost certainly an apex predator, and related to the ancestors of Tyrannosaurus Rex.

Feathers may have helped T. rex's relatives ride out a cold climate **Photograph by Dr. Brian Choo**

The relationship to its fellow Tyrannosaurids is implied by the genus name Yutyrannus; huali means "beautiful" in Mandarin. Fossils of three distinct individuals were discovered, largely intact. All of them share the feathers that are 10-15cm long filaments, and are present on the neck, back, and legs of the fossils. Other

areas aren't as well preserved, but the authors suspect that this broad distribution indicates the feathers were pretty much everywhere. Yutyrannus also displays a second distinct feature: a crest that runs along the center of its snout, from its nostrils back to just in front of its eyes.

Beyond that, Yutyrannus is a mix of features common to the ancestors of T. rex and specializations. So, for example, its forelimbs have three fingers, a trait that's called "basal," since it was present at the base of this lineage. Other features are called "derived." Some of these are somewhere intermediate between Yutyrannus and its relatives from later in the Cretaceous. Others are distinct to this species.

Feathers are a basal trait that show up elsewhere in the Tyrannosaurids, so one of the key questions is why the giants of the late Cretaceous had lost them (and not why they appeared in Yutyrannus). Feathers probably got their start as a means of insulation, and the authors argue that T. rex et. al probably didn't need anything of the sort. For starters, the late Cretaceous was a hothouse, with temperatures well above anything like today's.

But, just as significantly, they note that many of the world's largest animals—think whales and elephants—have also lost most of their fur. That may be a consequence of the fact that, as an animal's size increases, there's relatively more volume of tissue generating metabolic heat than there is surface area to radiate it away. When an animal gets large enough, the insulation becomes superfluous, and could actually put a limit on the animal's activity.

Yutyrannus' skull, showing the ridge in the center of its head, feather impressions, and some serious looking teeth.
Zang Hailong

There are exceptions to that. Large animals that live in cold climates, like the bison and mammoth, have retained their fur to deal with the chill. And that's exactly what the authors think is going on with Yutyrannus. At the time that the species was around, the area where it was found was about 10°C colder than it was during the late Cretaceous. In that climate, the feathers may have been necessary for even a giant Tyranosaurid to beat the chill. *Nature, 2012. DOI: 10.1038/nature10906.*

http://news.discovery.com/tech/seismic-wallpaper-120404.html

Seismic Wallpaper Stabilizes Walls in an Earthquake

During an earthquake, regular walls get shaken much like humans do. The side-to-side strain causes the masonry to crumble. This year, German materials scientists are producing seismic wallpaper that can hold a wall together.

Analysis by Alyssa Danigelis

Karlsruhe Institute of Technology's Institute of Solid Construction and Construction Material Technology has been simulating quake conditions to find better ways to shore up walls since the 1990s. Early attempts involved bonding thin carbon fiber slats to masonry, but that only made the crumbling worse.

Several years ago, they came up with another approach, one in which the surface of a whole wall could be reinforced using special wallpaper. In 2010, the institute's director, Lothar Stempniewski, and his colleagues began perfecting the material. They used stiff, high-strength glass fibers woven together to form a strong, elastic covering. The fibers run in four directions to distribute energy evenly when the walls are shaking, according to an article by Brigitte Osterath in Deutsche Welle.

Photo: The earthquake wallpaper is fixed to walls with a special adhesive. Credit: Bayer Material Science.

This special spun-glass covering alone isn't enough, though. Standard wallpaper glue can't hold up to an earthquake, so the KIT group collaborated with the polymer makers in the materials science division of the chemical company Bayer. They made a flexible, soft adhesive from water and a large amount of polyurethane beads. Once the adhesive penetrates grooves in the masonry, the water evaporates to anchor the substance in the wall. Similarly, when the wallpaper goes on, it gets completely surrounded by the beads. Together the whole setup won't tear during an earthquake.

To find out just how well it works, the seismic fabric was tested on a replica house in an earthquake simulator. "Because of the earthquake wallpaper, we were unable to make the building collapse," KIT researcher Mortiz Urban told Deutsche Welle. In a recent press video, Bayer indicated that the wallpaper will start going into commercial production this year through partner companies. It's expected to cost more than regular wallpaper, but the drastic difference for people living in earthquake-prone areas should be well worth the price.

http://blogs.scientificamerican.com/artful-amoeba/2012/04/04/can-diseases-cross-oceans-by-wind/

Can Diseases Cross Oceans By Wind?

Kawasaki Disease causes little kids to develop rash; fever; swollen hands, feet, and lymph nodes; red tongue and cracked lips and, develop coronary artery aneurysms that can kill right away or years later by heart attacks in otherwise totally healthy young adults

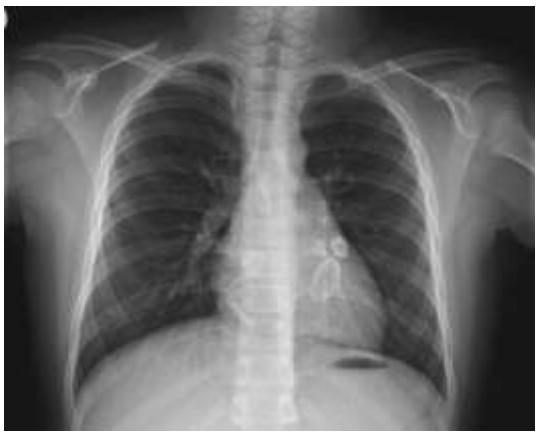
By Jennifer Frazer | April 4, 2012

That's the question I examine in my first feature story for Nature, published today online and in the print magazine April 5. A bizarre disease of toddlers and infants called Kawasaki Disease — which only emerged in the 1960s in Japan — causes little kids to develop rash; fever; swollen hands, feet, and lymph nodes; red tongue and cracked lips; and, bizarrely, to develop coronary artery aneurysms that can kill them right away or years later by heart attacks in otherwise totally healthy young adults.

Here's a story that didn't make it into the final cut of the article that describes one such case:

In one case, a 42-year-old white triathelete keeled over of a massive heart attack 10 miles into a 100 mile bike ride. He had no classic risk factors: he was not a smoker, and he was an extremely fit elite athlete with inklings he might have heart disease.

When the cardiologist, who happened to be Burns's husband, injected die into the man's arteries, he saw the telltale Kawasaki aneurysms immediately. When the patient's mother was consulted, it was discovered that when the man was five, he had been diagnosed with "atypical scarlet fever" in Florida. The man lived, but suffered ongoing cardiac disability as a result.



Aneurysms in the coronary arteries of a Kawasaki victim. **Public domain**

Despite decades of searching, no one has ever been able to figure out what causes it, even though some sort of infectious agent has long been suspected. And now, it seems that whatever is causing it — whether dead or alive — might be able to cross the Pacific Ocean and sicken children in Hawaii, North America, and beyond.

If this proves to be true, it will be the first time a human pathogen has ever been shown to do that. So head on over to Nature and check it out — and check out the podcast interview with me embedded in the story halfway through as well. You can hear what I sound like!

Dr. Jane Burns, who I interviewed for nearly two hours for the story and who graciously shared with me her lifetime of experience with the disease, was featured in the lead of the story about the infant from Wyoming. That baby was only 3 months old when she was airlifted to Colorado and died within 12 hours of arriving there.

Occasionally when you report a story you encounter amazing details that just don't fit within the context of the story you want to write, but are nonetheless powerful. Burns told me this further story about that baby's parents. It really pulled on my heartstrings, so I wanted to share it with you:

A month later the physicians invited the family back to go over the autopsy and interpret the results for them. Burns attended the meeting along with the pathologist. The family handed her a brown paper bag with $1,500 in small bills. They had gone door to door in their town in Wyoming to collect the money.

"[They] said, 'We hope you'll take this money and do research on Kawasaki Disease,'" Burns recalled. "That was obviously a very powerful moment for me."

Dr. Burns went on to devote nearly her entire career to Kawasaki Disease.

On a personal note, can I just say what a thrill it was seeing the color proofs of a page from Nature with my name on the story? I kept looking at the bottom of the page "Nature, Vol. x, Issue x", and then looking back up at my name. Surreal. Very surreal.

Thank you to my editor at Nature for all the hard work on the story and for encouraging me at every step of the way. Couldn't have done it without you, Mitch!

http://www.eurekalert.org/pub_releases/2012-04/cp-tdb033012.php

Tackling dyslexia before kids learn to read

For children with dyslexia, the trouble begins even before they start reading and for reasons that don't necessarily reflect other language skills.

That's according to a report published online on April 5 in Current Biology, a Cell Press publication, that for the first time reveals a causal connection between early problems with visual attention and a later diagnosis of dyslexia. "Visual attention deficits are surprisingly way more predictive of future reading disorders than are language abilities at the prereading stage," said Andrea Facoetti of the University of Padua in Italy.

The researchers argue that the discovery not only closes a long-lasting debate on the causes of dyslexia but also opens the way to a new approach for early identification and interventions for the 10 percent of children for whom reading is extremely difficult.

The researchers studied Italian-speaking children for a period of three years, from the time they were prereading kindergarteners until they entered second grade. Facoetti's team, including Sandro Franceschini, Simone Gori, Milena Ruffino, and Katia Pedrolli, assessed prereaders for visual spatial attention—the ability to filter relevant versus irrelevant information - through tests that asked them to pick out specific symbols amid distractions. The children also took tests on syllable identification, verbal short-term memory, and rapid color naming, followed over the next two years by measures of reading. Those test results showed that kids who initially had trouble with visual attention were also the ones to later struggle in reading.

"This is a radical change to the theoretical framework explaining dyslexia," Facoetti said. "It forces us to rewrite what is known about the disorder and to change rehabilitation treatments in order to reduce its impact."

He says that simple visual-attention tasks should improve the early identification of children at risk for dyslexia. "Because recent studies show that specific prereading programs can improve reading abilities, children at risk for dyslexia could be treated with preventive remediation programs of visual spatial attention before they learn to read." *Franceschini et al.: "A Causal Link between Visual Spatial Attention and Reading Acquisition."*

http://www.eurekalert.org/pub_releases/2012-04/mgh-baa040512.php

Big advance against cystic fibrosis

Stem cell researchers create lung surface tissue in a dish

Harvard stem cell researchers at Massachusetts General Hospital (MGH) have taken a critical step in making possible the discovery in the relatively near future of a drug to control cystic fibrosis (CF), a fatal lung disease that claims about 500 lives each year, with 1,000 new cases diagnosed annually.

Beginning with the skin cells of patients with CF, Jayaraj Rajagopal, MD, and colleagues first created induced pluripotent stem (iPS) cells, and then used those cells to create human disease-specific functioning lung epithelium, the tissue that lines the airways and is the site of the most lethal aspect of CF, where the genes cause irreversible lung disease and inexorable respiratory failure.

That tissue, which researchers now can grow in unlimited quantities in the laboratory, contains the delta-508 mutation, the gene responsible for about 70 percent of all CF cases and 90 percent of the ones in the United States. The tissue also contains the G551D mutation, a gene that is involved in about 2 percent of CF cases and the one cause of the disease for which there is now a drug.

The work is featured on the cover of this month's Cell Stem Cell journal, which appeared online today. Postdoctoral fellow Hongmei Mou, PhD, is first author on the paper, and Rajagopal is the senior author.

Mou credits learning the underlying developmental biology in mice as the key to making tremendous progress in only two years. "I was able to apply these lessons to the iPS cell systems," she said. "I was pleasantly surprised the research went so fast, and it makes me excited to think important things are within reach. It opens up the door to identifying new small molecules [drugs] to treat lung disease."

Doug Melton, PhD, co-director of the ***Harvard Stem Cell Institute***, said, "This work makes it possible to produce millions of cells for drug screening, and for the first time human patients' cells can be used as the target." Melton, who is also co-chair of Harvard's inter-School Department of Stem Cell and Regenerative Biology and is the Xander University Professor, added, "I would expect to see rapid progress in this area now that human cells, the very cells that are defective in the disease, can be used for screening."

Rajagopal said, "The key to our success was the ecosystem of the Harvard Stem Cell Institute and MGH. HSCI investigators pioneered the strategies we used, helped us at the bench, and gave us advice on how to combine our knowledge of lung development with their exciting new platforms. Indeed, we also enjoyed a wonderful collaboration with Darrell Kotton's lab at Boston University that was able to convert mouse cells into lung tissue. These interactions really helped fuel us ahead."

The epithelial tissue created by Rajagopal and his colleagues at the MGH Center for Regenerative Medicine (http://www.massgeneral.org/regenmed/) also provides researchers with the same cells that are involved in a number of common lung conditions, including asthma, lung cancer, and chronic bronchitis, and may hasten the development of new insights and treatments into those conditions as well.

"We're not talking about a cure for CF; we're talking about a drug that hits the major problem in the disease. This is the enabling technology that will allow that to happen in a matter of years," said Rajagopal, a Harvard Medical School assistant professor of Medicine. Also a physician trained as a pulmonologist, the specialty that treats CF patients, Rajagopal said, "When we talk about research and advances, donors and patients ask: 'When? How soon?' And we usually hesitate to answer. But we now have every single piece we need for the final push. So I have every hope that we'll have a therapy in a matter of years."

Cystic fibrosis, which used to claim its victims in infancy or early childhood, has evolved into a killer of those in their 30s because treatments of the infections that characterize the disease have improved. But despite those advances, there has been little progress in treating the underlying condition that affects the vast majority of patients: a defect in a single gene that interferes with the fluid balance in the surface layers of the airways and leads to a thickening of mucus, difficulty breathing and repeated infections and hospitalizations.

The discovery and recent FDA approval of the drug Ivacaftor, which corrects the G551D defect seen in about 2 percent of CF patients, has served as a proof of concept to demonstrate that the disease can be attacked with a conventional molecular treatment. In fact, Ivacaftor was found by screening thousands of drugs on a far less than ideal cell line. In the end, many drugs that functioned well on this cell line proved ineffective when used on genuine human airway tissue.

Genuine human airway tissue is the gold standard prior to drugs being tested clinically, but it has been extremely difficult to obtain the tissue from patients, and when it could be obtained, the tissue rarely survived long in the lab – all of which created a major bottleneck in screening for a therapy. But by creating iPS cells that contain the entire genome of a CF patient and directing those cells to develop into lung progenitor cells, which then develop into epithelium, the group appears to have solved this key problem.

Rajagopal, who did his own postdoctoral fellowship in Melton's laboratory during the first half of the past decade after completing his training in pulmonary medicine, said that having both the G551D and 508 genes in the epithelial tissue provides a way to prove that the tissue will be effective in testing drugs against CF.

"We've created the perfect cell line to show that the drug out there that works against G551D mutation works in this system, and then we're in business to screen for a drug against delta 508," he said. "We'll know soon that the cell line works. We know it makes bonafide airway epithelium, and we'll have the proof of principle that the tissue responds properly to the only known drug. We think this is the near-ideal tissue platform to find a drug for the majority of CF." Rajagopal's lab has created numerous other cell lines to further show that a CF drug that works in one patient should work in others and to see whether this will be an area that allows a more personalized approach to medicine.

"I'm most looking forward to working with the community of pulmonologists that concentrate in CF to generate therapies. This is occurring more than two decades after the remarkable work that identified the CF gene. Looking forward, I'm very excited that CF may lead the way in lung disease once more, by demonstrating that our iPS platform can be used to probe the diseases that are much less well understood. CF has more than two decades of great biology behind it. The reason we chose to attack this disease first was because of that pioneering work that lets us use our system with a very firm foundation," Rajagopal said.

http://www.eurekalert.org/pub_releases/2012-04/bmj-aas040412.php

Antibiotics a safe and viable alternative to surgery for uncomplicated appendicitis, say experts

Research: Safety and efficacy of antibiotic therapy with appendicectomy in the treatment of uncomplicated acute appendicitis: Meta-analysis

Giving antibiotics to patients with acute uncomplicated appendicitis is a safe and viable alternative to surgery, say experts in a study published on bmj.com today.

Surgery to remove an inflamed appendix (appendicectomy) has been the mainstay of treatment for acute appendicitis since 1889 and the general assumption is that, without surgery, the risk of complications, such as perforation or infection, is high. However, recent studies have reported fewer problems with antibiotic therapy than surgery in patients with uncomplicated appendicitis, but results have been inconclusive.

So a team of researchers at the Nottingham Digestive Diseases Centre NIHR Biomedical Research Unit set out to compare the safety and efficacy of antibiotic therapy as an initial treatment for uncomplicated acute appendicitis.

They analysed the results of four randomised controlled trials involving 900 adult patients diagnosed with uncomplicated acute appendicitis. A total of 470 patients received antibiotics and 430 underwent surgery.

Differences in study design and quality were taken into account to minimise bias.

Antibiotic therapy was associated with a 63% success rate at one year and a 31% relative reduction in complications compared with surgery. Even after excluding patients from one study who crossed over from the antibiotic group to the surgery group, a significant (39%) reduction in complications with antibiotic therapy compared with surgery remained.

Of 68 patients treated with antibiotics who were readmitted with recurrence of symptoms, four had normal appendix and 13 had complicated appendicitis. Three patients were treated successfully with another course of antibiotics. There were no significant differences in either length of hospital stay or risk of developing complicated appendicitis between the two groups of patients.

The authors argue that the role of antibiotics in acute uncomplicated appendicitis "has been overlooked based mainly on tradition rather than evidence" and they suggest that a careful 'wait, watch and treat' policy may be adopted in patients considered to have uncomplicated appendicitis or in whom the diagnosis is uncertain. However, they stress that for those with clear signs of perforation or peritonitis (inflammation of the abdominal wall) … early appendicectomy still remains the 'gold standard.' They conclude that antibiotic therapy "is a safe initial therapy for patients with uncomplicated acute appendicitis" and that it "merits consideration as a primary treatment option for early uncomplicated appendicitis."

In an accompanying editorial, Dr Olaf Bakker from the Department of Surgery at the University Medical Center Utrecht in the Netherlands argues that treating appendicitis conservatively has "major certain disadvantages" as the reoccurrence rate of appendicitis is up to 20% in the first year. He argues that until more convincing and longer term results are published, "appendectomy for uncomplicated appendicitis will probably continue."

http://www.realclimate.org/index.php/archives/2012/04/evaluating-a-1981-temperature-projection/

Evaluating a 1981 temperature projection

Sometimes it helps to take a step back from the everyday pressures of research (falling ill helps).

Guest commentary from Geert Jan van Oldenborgh and Rein Haarsma, KNMI

Sometimes it helps to take a step back from the everyday pressures of research (falling ill helps). It was in this way we stumbled across Hansen et al (1981) (pdf). In 1981 the first author of this post was in his first year at university and the other just entered the KNMI after finishing his masters. Global warming was not yet an issue at the KNMI where the focus was much more on climate variability, which explains why the article of Hansen et al. was unnoticed at that time by the second author. It turns out to be a very interesting read.

They got 10 pages in Science, which is a lot, but in it they cover radiation balance, 1D and 3D modelling, climate sensitivity, the main feedbacks (water vapour, lapse rate, clouds, ice- and vegetation albedo); solar and volcanic forcing; the uncertainties of aerosol forcings; and ocean heat uptake. Obviously climate science was a mature field even then: the concepts and conclusions have not changed all that much. Hansen et al clearly indicate what was well known (all of which still stands today) and what was uncertain.

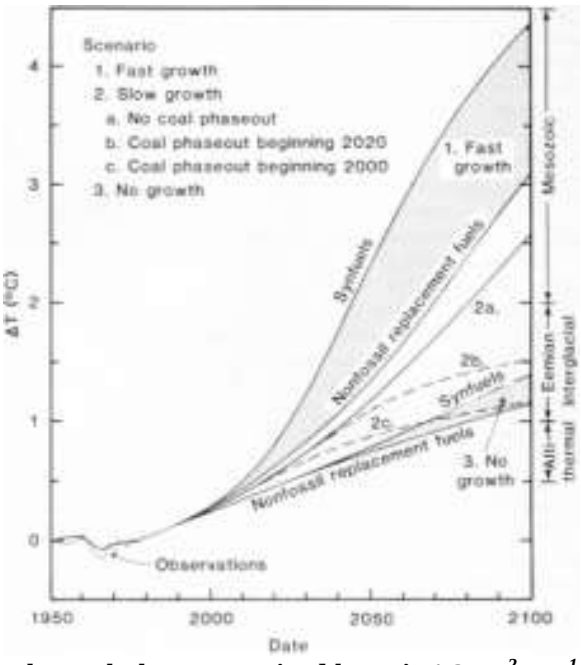


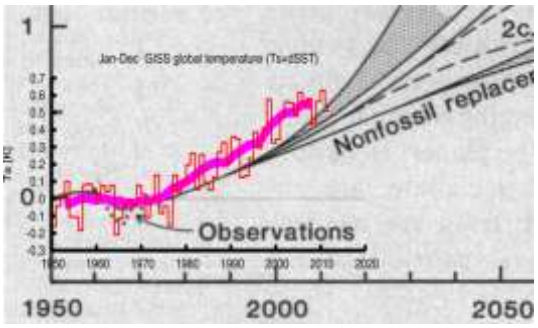
Fig. 6. Projections of global temperature. The diffusion coefficient beneath the ocean mixed layer is 1.2 cm^2 sec^{-1}, as required for best fit of the model and observations for the period 1880 to 1978. Estimated global mean warming in earlier warm periods is indicated on the right.

Next they attribute global mean temperature trend 1880-1980 to CO2, volcanic and solar forcing. Most interestingly, Fig.6 (below) gives a projection for the global mean temperature up to 2100. At a time when the northern hemisphere was cooling and the global mean temperature still below the values of the early 1940s, they confidently predicted a rise in temperature due to increasing CO2 emissions. They assume that no action will be taken before the global warming signal will be significant in the late 1990s, so the different energy-use scenarios only start diverging after that.

The first 31 years of this projection are thus relatively well-defined and can now be compared to the observations. We used the GISS Land-Ocean Index that uses SST over the oceans (the original one interpolated from island stations) and overlaid the graph from the KNMI Climate Explorer on the lower left-hand corner of their Fig.6.

Given the many uncertainties at the time, notably the role of aerosols, the agreement is very good indeed. They only underestimated the observed trend by about 30%, similar or better in magnitude than the CMIP5 models over the same period (although these tend to overestimate the trend, still mainly due to problems related to aerosols).

To conclude, a projection from 1981 for rising temperatures in a major science journal, at a time that the temperature rise was not yet obvious in the observations, has been found to agree well with the observations since then, underestimating the observed trend by about 30%, and easily beating naive predictions of no-change or a linear continuation of trends. It is also a nice example of a statement based on theory that could be falsified and up to now has withstood the test. The "global warming hypothesis" has been developed according to the principles of sound science.



References *J. Hansen, D. Johnson, A. Lacis, S. Lebedeff, P. Lee, D. Rind, and G. Russell, "Climate Impact of Increasing Atmospheric Carbon Dioxide", Science, vol. 213, 1981, pp. 957-966. DOI.*

http://news.discovery.com/human/jesus-happy-120406.html

Was Jesus Happy?

How you answer that question says a lot about the culture that influenced you most.

By Jennifer Viegas

Although Biblical descriptions of Jesus are essentially the same worldwide, responses to the question "Was Jesus happy?" widely differ. Researchers have found that the answers are predicted by the respondent's country and culture. The image of Jesus might be culturally constructed to fit an existing ideal, or it could be a reflection of the individual's self-image. Americans tend to think Jesus was happy, extroverted, agreeable, kind and caring. Koreans, on the other hand, associate Jesus more with suffering, sacrifice, and pity, according to a recent analysis in Personality and Social Psychology Connections and a paper published in the Journal of Research in Personality.

Responses to the simple question about Jesus and happiness, whose Biblical depiction is essentially the same worldwide, turn out to involve complex factors, such as shared life histories among groups of people, culture and possibly even genetics. All of these can affect how an individual defines what the optimal personality or self should look like.

"Americans meet far more strangers than others and need to be more extroverted than the Japanese, Koreans and others who tend to interact with a small number of people repeatedly, so extroversion is a highly valued asset in the U.S.," Shigehiro Oishi, lead author of the study, told Discovery News. "In the end, happiness, extroversion, and kindness are all highly valued qualities among Americans, and they might just see Jesus to have these highly desirable characteristics."

For the study, Oishi, an associate professor in the Department of Psychology at the University of Virginia, and colleagues Kyoung Ok Seol, Minkyung Koo, and Felicity Miao asked Korean and American university students to engage in a free association task with Jesus as a target. The researchers said they chose Americans and Koreans because many people identifying as Christian exist in both populations. Americans associated Jesus primarily with positive connotations ("awesome" was a common response) and rarely with negative connotations, such as "pain," which was more frequently mentioned by the Korean study participants.

In a second part of the study, the researchers asked the test subjects to rate Jesus and themselves using personality and well-being scales. Americans again tended to emphasize happiness over sorrow.

Oishi said that "Buddhism and other religions had been firmly in place in Korea before the introduction of Christianity, and life is suffering in Buddhism."

"Buddhism came out of a really tough societal condition," he continued. "Most people were suffering. The main goal of Buddhism was to reduce pain and suffering. Because Christianity was introduced long after Buddhism in Korea, probably the part of Christianity that fits well with Buddhism was emphasized in Korea."

Other cultural differences may further explain the American and Korean responses. Oishi said such differences pose "an egg and chicken problem" involving genetics and shared life experiences, since one can

affect the other. It is also unclear if the image of Jesus might be culturally constructed to fit an existing ideal, or if it could reflect an individual's self-image.

Casey Eggleston, a researcher at the University of Virginia, told Discovery News that language differences also come into play, with the meaning of happiness differing across cultures over time.

"The historical definition included concepts of luck and good fortune, but that meaning has fallen out of use in the U.S., where many believe they can pursue and obtain happiness by their own effort, while it remains a major part of the concept in most other cultures," she explained. "Similarly, the emotional connotation of the word happy varies substantially. While the American concept typically includes upbeat positive emotions like excitement, the concept in East Asia tends to focus more on calm positive emotions like peace and contentment."

The researchers chose to focus on two particular countries, but they expect respondents in other nations with a large Christian base would also provide different, culture-predicted responses to the question, "Was Jesus happy?"

As for their own answers, Eggleston said, "There are two primary approaches to happiness: hedonia and eudaimonia. Hedonia is a state of pleasure and physical enjoyment. In the sense of feeling good, I don't think Jesus was happy most of the time, although he undoubtedly had pleasant moments during his ministry."

"Eudaimonia, on the other hand, is happiness achieved through virtuous living—pursuing a meaningful, viable life and doing so with integrity," she added. "If we use this Aristotelian understanding of happiness, I think Jesus must have been exceedingly happy."

As for Oishi's answer to whether or not Jesus was happy, he said, "I don't know for sure, but I don't think so. He had a tough life."

http://www.eurekalert.org/pub_releases/2012-04/tcd-trr040612.php

Trinity researchers report major eye disease breakthrough

Controlling an inflammatory component IL-18 in age-related macular degeneration could prevent the development of the disease

Scientists at Trinity College Dublin have discovered that a part of the immune system called the inflammasome is involved in regulating the development of one of the most common forms of blindness, called Age-Related Macular Degeneration (AMD). They have discovered that controlling an inflammatory component IL-18, in cases of Age-Related Macular Degeneration (AMD) could prevent the development of the disease.

The disease AMD involves loss of central vision, people with advanced disease being unable to read, watch TV, enjoy the cinema, drive, or use a computer − in short, everyday living becomes very difficult. The research, which is published this week in the international medical journal, Nature Medicine, is supported by Science Foundation Ireland, the American Health Assistance Foundation (AHAF), the Health Research Board (HRB) and Fighting Blindness Ireland.

The key diagnostic feature of AMD is the presence of "drusen", which are recognised during an eye exam as yellowish/white deposits in the central region of the retina called the macula. Dry AMD is characterised by the presence of excessive amounts of drusen and there are currently no forms of therapy other than recommended lifestyle changes such as giving up smoking, which is a recognised risk factor. However, a significant number of cases of the "dry" form of AMD can progress to the "wet" form, where blood vessels underneath the retina begin to grow, leading to central blindness. If you hold two coins immediately in front of your eyes, you will see a single large black circle blocking out your central vision. This is a very realistic simulation of what it is like to live with advanced disease.

The leading co-authors of the Nature Medicine paper, Trinity College scientists, Dr Sarah Doyle and Dr Matthew Campbell have together discovered that drusen accumulating in the macula can lead to the production of two inflammatory components termed IL-1beta and IL-18. These findings were based on studies involving drusen isolated from donor AMD eyes in tandem with pre-clinical studies on models of the disease.

"Traditionally, inflammation in the retina or indeed the eye in general is not beneficial and is a pathological hallmark of many eye diseases, including AMD. However we have identified, that one inflammatory component termed IL-18 acts as a so-called anti-angiogenic factor, preventing the progression of wet AMD" says Dr. Campbell. "The progression from "dry" to "wet" AMD appears to be mediated by the inflammatory component IL-18, our results directly suggest that controlling or indeed augmenting the levels of IL-18 in the retinas of patients with dry AMD could prevent the development of the wet form of disease, which leads us to an exciting new prospect for a novel therapy for AMD" says Dr Doyle.

The research was undertaken at Trinity College's Ocular Genetics Unit, Director, Professor Pete Humphries and at the laboratories of Professor Luke O'Neill at the Trinity Biomedical Sciences Institute, in collaboration with Professor Joe Holyfield at the Cole Eye Institute at Cleveland, Ohio.

Full title of the paper: 'NLRP3 has a protective role in age-related macular degeneration through the induction of IL-18 by drusen components'

http://www.eurekalert.org/pub_releases/2012-04/tmsh-nb040512.php

New 'genetic bar code' technique establishes ability to derive DNA information from RNA

Discovery may create dialogue about DNA and RNA data bank privacy issues

Researchers from Mount Sinai School of Medicine have developed a method to derive enough DNA information from non-DNA sources—such as RNA—to clearly identify individuals whose biological data are stored in massive research repositories. The approach may raise questions regarding the ability to protect individual identity when high-dimensional data are collected for research purposes. A paper introducing the technique appears in the April 8 online edition of Nature Genetics.

DNA contains the genetic instructions used in the development and functioning of every living cell. RNA acts as a messenger that relays genetic information in the cell so that the great majority of processes needed for tissue to function properly can be carried out.

To date, access to data bases with DNA information has been restricted and protected as it has long been considered the sole genetic fingerprint for every individual. However, vast amounts of RNA data have been made publicly available via a number of databases in the United States and Europe. These databases contain thousands of genomic studies from around the world.

In this study, lead authors Eric E. Schadt, PhD, and Ke Hao, PhD, developed a technique whereby a person's DNA could be inferred from RNA data using gene-expression levels monitored in any of a number of tissues. In contrast, most studies involving DNA and RNA begin with DNA sequences and then seek to associate expression patterns with changes in DNA between individuals in a population. This is the first time going from RNA levels to DNA sequence has been described.

"By observing RNA levels in a given tissue, we can infer a genotypic barcode that uniquely tags an individual in ways that enables matching the individual to an independently derived DNA sample," said Dr. Schadt, Director of the Institute for Genomics and Multiscale Biology, the Jean C. and James W. Crystal Professor of Genomics, and Chair of the Department of Genetics and Genomics Sciences, Mount Sinai School of Medicine. "The potential uses for this information are significant. Not only can genotypic barcodes be deduced from RNA, but RNA levels in some tissue can inform not only individual characteristics like age and sex, but on diseases such as Alzheimer's and cancer, as well as the risks of developing those diseases."

Schadt adds, "The significance of our findings goes beyond medicine. For example, barcodes derived from individuals who participated in a research study, where RNA levels were monitored and deposited into publicly available databases, could be tested against DNA samples left at a crime scene as a way of identifying persons of interest."

Deducing a person's DNA sequence from gene expression patterns could have repercussions in health care and privacy. While specific laws and government regulations have been written to protect DNA-based information from misuse, it is unclear whether such laws apply to RNA—even though this study shows that RNA is informative at a deeper level compared to DNA regarding the current state of health of an individual.

"Rather than developing ways to further protect an individual's privacy given the ability to collect mountains of information on him or her, we would be better served by a society that accepts the fact that new types of high-dimensional data reflect deeply on who we are," Dr. Schadt said. "We need to accept the reality that it is difficult—if not impossible—to shield personal information from others. It is akin to trying to protect privacy regarding appearances, for example, in a public place."

Dr. Schadt said he hopes the research will catalyze a discussion that might ultimately help resolve privacy debates, and encourage patients to provide data that will help their doctors better diagnose and treat their conditions. Increased access to, and greater quantities of, DNA and other biological information would also contribute to the greater good of medical science.

In the Nature Genetics study, Drs. Schadt and Hao, Associate Professor of Genetics at Mount Sinai School of Medicine, together with Sangsoon Woo, PhD, from the Department of Biostatistics at the University of Washington, analyzed RNA and DNA from 378 livers donated by European-Americans for transplant, as well as liver and adipose tissues from 580 people from the same population group undergoing gastric bypass surgery. The authors found that levels of RNA across many genes correlate with age, sex, body weight, and other risk factors for diseases like diabetes and heart disease, but then they also correlate in many cases with changes in DNA that are unique to a given individual.

The investigators used an algorithm that matches patterns of gene expression to variations at 1,000 single-DNA-base sites in the genome. It is an application of integrative biology that examines multiple dimensions of data (DNA and RNA) to better inform a given dimension (RNA).

"The relationship of DNA to RNA is like that of an orchestra and the symphony it plays," said Schadt describing the new technique. "The DNA (orchestra) remains the same, while the RNA pattern (quality of the music) changes in response to outside factors. The new technique is like hearing a symphony and deducing which instruments are in the orchestra, essentially unwinding the developmental process to trace tissue samples back to RNA and the gene that instructed it."

http://www.sciencedaily.com/releases/2012/04/120408212319.htm

Evolution at Sea: Long-Term Experiments Indicate Phytoplankton Can Adapt to Ocean Acidification

Scientists have now demonstrated the potential of unicellular algae to adapt to changing pH

ScienceDaily - Fossil fuel derived carbon dioxide has a serious impact on global climate but also a disturbing effect on the oceans, know as the other CO2 problem. When CO2 dissolves in seawater it forms carbonic acid and results in a drop in pH, the oceans acidify. A wealth of short-term experiments has shown that calcifying organisms, such as corals, clams and snails, but also micron size phytoplankton are affected by ocean acidification. The potential for organisms to cope with acidified oceanic conditions via evolutionary adaptations has so far been unresolved.

Scientists of the Helmholtz Centre for Ocean Research Kiel (GEOMAR) have now for the first demonstrated the potential of the unicellular algae Emiliania huxleyi to adapt to changing pH conditions and thereby at least partly to mitigate negative effects of ocean acidification. These results raised by the biologists Kai Lohbeck, Prof. Ulf Riebesell and Prof. Thorsten Reusch are published in the current issue of Nature Geoscience.

Experimental Emiliania huxleyi strains were isolated in Norwegian coastal waters and cultured in the laboratory under projected future ocean CO2 conditions. After about one year, which translates into 500 generations in this rapidly reproducing species, the biologists detected adaptation to high CO2 – adapted populations grew and calcified significantly better than non adapted control populations when tested under ocean acidification condition.

“From a biogeochemical perspective the most interesting finding was probably a partly restoration in calcification rates” GEOMAR scientist Prof. Ulf Riebesell notes. Emiliania huxleyi covers its cell surface with minute calcite scales that were found to decrease in weight under increased CO2 concentrations. “This is what we expected from the literature. But we were fascinated to find impaired calcification to partly recover after only 500 generations” says biologist Kai Lohbeck.

The evolutionary mechanisms proposed by the GEOMAR scientists are selection on different genotypes and the accumulation of novel beneficial mutations. Such an adaptation has not been shown earlier in any key phytoplankton species. “With this study we have shown for the fist time that evolutionary processes may have the potential to act on climate change relevant time scales and thereby mitigate negative effects of ongoing ocean acidification” says evolutionary biologist Thorsten Reusch and adds “These findings emphasize the need for a consideration of evolutionary processes in future assessment studies on the biological consequences of global change”.

Despite this finding, the GEOMAR scientists by no means think about an all-clear signal for ocean acidification. The potential for adaptive evolution may be large in rapidly reproducing species with large population sizes as is Emiliania huxleyi. “This is one reason why we have chosen this species for our studies” say the biologists. Long-lived species and especially those having only a few offspring per generation commonly have a much lower adaptive potential on climate change relevant time scales. “Earth history tells a convincing story about the limitations to evolutionary adaptation” Prof. Ulf Riebesell explains, “environmental changes comparable to what happens right now in the oceans have repeatedly resulted in mass extinctions, even though these changes were 10-100 times slower than what we observe today”.

Another open question is to what extent the evolutionary changes observed under laboratory conditions are transferable to the oceans where other environmental factors and ecological interactions play along. Therefore, the GEOMAR scientists already started to prepare follow-up experiments. In the framework of the BIOACID (Biological Impacts of Ocean ACIDification) project, funded by the German Federal Ministry of Education and Research (BMBF), the biologists plan to use the Kiel Off-Shore Mesocosms to investigate the adaptive potential of Emiliania huxleyi under natural conditions.

Kai T. Lohbeck, Ulf Riebesell, Thorsten B. H. Reusch. Adaptive evolution of a key phytoplankton species to ocean acidification. Nature Geoscience, 2012; DOI: 10.1038/NGEO1441