http://www.physorg.com/news/2012-02-climate-today-year-years.html

# With climate change, today's '100-year floods' may happen every three to 20 years: research

***Last August, Hurricane Irene spun through the Caribbean and parts of the eastern United States, leaving widespread wreckage in its wake.***

The Category 3 storm whipped up water levels, generating storm surges that swept over seawalls and flooded seaside and inland communities. Many hurricane analysts suggested, based on the wide extent of flooding, that Irene was a "100-year event": a storm that only comes around once in a century.

However, researchers from MIT and Princeton University have found that with climate change, such storms could make landfall far more frequently, causing powerful, devastating storm surges every three to 20 years. The group simulated tens of thousands of storms under different climate conditions, finding that today's "500-year floods" could, with climate change, occur once every 25 to 240 years. The researchers published their results in the current issue of Nature Climate Change.

MIT postdoc Ning Lin, lead author of the study, says knowing the frequency of storm surges may help urban and coastal planners design seawalls and other protective structures.

"When you design your buildings or dams or structures on the coast, you have to know how high your seawall has to be," Lin says. "You have to decide whether to build a seawall to prevent being flooded every 20 years."

Lin collaborated with Kerry Emanuel, the Cecil and Ida Green Professor of Atmospheric Science at MIT, as well as with Michael Oppenheimer and Erik Vanmarcke at Princeton. The group looked at the impact of climate change on storm surges, using New York City as a case study.

To simulate present and future storm activity in the region, the researchers combined four climate models with a specific hurricane model. The combined models generated 45,000 synthetic storms within a 200-kilometer radius of Battery Park, at the southern tip of Manhattan.

They studied each climate model under two scenarios: a "current climate" condition representing 1981 to 2000 and a "future climate" condition reflecting the years 2081 to 2100, a prediction based on the Intergovernmental Panel on Climate Change's projections of future moderate carbon dioxide output. While there was some variability among the models, the team generally found that the frequency of intense storms would increase due to climate change.

Once they simulated storms in the region, the researchers then simulated the resulting storm surges using three different models, including one used by the National Hurricane Center (NHC). In the days or hours before a hurricane hits land, the NHC uses a storm-surge model to predict the risk and extent of flooding from the impending storm. Such models, however, have not been used to evaluate multiple simulated storms under a scenario of climate change.

Again, the group compared results from multiple models: one from the NHC which simulates storm surges quickly, though coarsely; another model that generates more accurate storm surges, though less efficiently; and a model in between, developed by Lin and her colleagues, that estimates relatively accurate surge floods, relatively quickly.

Today, a "100-year storm" means a surge flood of about two meters, on average, in New York. Roughly every 500 years, the region experiences towering, three-meter-high surge floods. Both scenarios, Lin notes, would easily top Manhattan's seawalls, which stand 1.5 meters high.

But with added greenhouse gas emissions, the models found that a two-meter surge flood would instead occur once every three to 20 years; a three-meter flood would occur every 25 to 240 years.

"The highest [surge flood] was 3.2 meters, and this happened in 1821," Lin says. "That's the highest water level observed in New York City's history, which is like a present 500-year event."

Carol Friedland, an assistant professor of construction management and industrial engineering at Louisiana State University, sees the group's results as a useful tool to inform coastal design - particularly, she notes, as most buildings are designed with a 60- to 120-year "usable lifespan."

"The physical damage and economic loss that result from storm surge can be devastating to individuals, businesses, infrastructure and communities," Friedland says. "For current coastal community planning and design projects, it is essential that the effects of climate change be included in storm-surge predictions."

*Provided by Massachusetts Institute of Technology This story is republished courtesy of MIT News (http://web.mit.edu/newsoffice/), a popular site that covers news about MIT research, innovation and teaching.*

http://medicalxpress.com/news/2012-02-stepped-up-leprosy.html

## WHO calls for stepped-up fight against leprosy

***The World Health Organization called Monday for greater efforts to fight leprosy, warning the disfiguring disease was defying efforts to wipe it out across many countries in the Asia-Pacific region.***

"We opened the champagne too early," said Shin Young-soo, chairman of the WHO's Western-Pacific region that covers 37 countries at the start of a three-day conference looking at how to combat leprosy and treat its victims. There are 5,000 new cases being reported each year in the Western Pacific, according to Shin.

He said the problem was most severe in Micronesia, the Marshall Islands and Kiribati, which had failed to meet the WHO's technical definition of "elimination" of fewer than one case per 10,000 people.

Even in the Philippines, where the disease was officially "eliminated" in 1998, 2,000 new cases are still recorded every year, according to Shin. Outside of the Western Pacific, the problem is worse.

India leads the world with more than 130,000 new leprosy cases every year since 2006, while Brazil is second with about 40,000 new cases annually, according to WHO documents.

Shin called for a renewed commitment to fight leprosy, stressing that it had to be long-term because the disease could incubate for as long as 20 years. "We have the drugs, we have the knowledge. It does not take a lot of money. We must make a final push," he said.

Leprosy is an infectious bacterial disease that has been recorded for thousands of years. If left untreated it can damage the nerves, leading to paralysis in the extremities of the body and horrible disfigurements.

However it is curable with early detection and modern drugs.

The WHO has been providing free drug therapy to patients anywhere in the world since 1995.

Shin said that, with the medical hurdles overcome, the major challenge in countries with enduring leprosy was to ensure long-term commitment from governments. *(c) 2012 AFP*

http://www.eurekalert.org/pub_releases/2012-02/ru-puc021312.php

## Plants use circadian rhythms to prepare for battle with insects

***Rice University study: Plants make predawn preparations to fend off hungry caterpillars***

HOUSTON - In a study of the molecular underpinnings of plants' pest resistance, Rice University biologists have shown that plants both anticipate daytime raids by hungry insects and make sophisticated preparations to fend them off.

"When you walk past plants, they don't look like they're doing anything," said Janet Braam, an investigator on the new study, which appears this week in the Proceedings of the National Academy of Sciences. "It's intriguing to see all of this activity down at the genetic level. It's like watching a besieged fortress go on full alert."

Braam, professor and chair of Rice's Department of Biochemistry and Cell Biology, said scientists have long known that plants have an internal clock that allows them to measure time regardless of light conditions. For example, some plants that track the sun with their leaves during the day are known to "reset" their leaves at night and move them back toward the east in anticipation of sunrise.

In recent years, scientists have begun to apply powerful genetic tools to the study of plant circadian rhythms. Researchers have found that as many as one-third of the genes in Arabidopsis thaliana -- a widely studied species in plant biology -- are activated by the circadian cycle. Rice biochemist Michael Covington found that some of these circadian-regulated genes were also connected to wounding responses.

"We wondered whether some of these circadian-regulated genes might allow plants to anticipate attacks from insects, in much the same way that they anticipate the sunrise," said Covington, now at the University of California, Davis.

Danielle Goodspeed, a graduate student in biochemistry and cell biology, designed a clever experiment to answer the question. She used 12-hour light cycles to entrain the circadian clocks of both Arabidopsis plants and cabbage loopers, a type of caterpillar that eats Arabidopsis. Half of the plants were placed with caterpillars on a regular day-night cycle, and the other half were placed with "out-of-phase" caterpillars whose internal clocks were set to daytime mode during the hours that the plants were in nighttime mode.

"We found that the plants whose clocks were in phase with the insects were relatively resistant, whereas the plants whose clocks were out of phase were decimated by the insects feeding on them," Goodspeed said.

Wassim Chehab, a Rice faculty fellow in biochemistry and cell biology, helped Goodspeed design a follow-up experiment to understand how plants used their internal clocks to resist insect attacks. Chehab and Goodspeed examined the accumulation of the hormone jasmonate, which plants use to regulate the production of metabolites that interfere with insect digestion.

They found that Arabidopsis uses its circadian clock to increase jasmonate production during the day, when insects like cabbage loopers feed the most. They also found that the plants used their internal clocks to regulate the production of other chemical defenses, including those that protect against bacterial infections.

"Jasmonate defenses are employed by virtually all plants, including tomatoes, rice and corn," Chehab said. "Understanding how plants regulate these hormones could be important for understanding why some pests are more damaging than others, and it could help suggest new strategies for insect resistance."

*The research was supported by the National Science Foundation and Rice University. Additional co-authors include Rice undergraduate Amelia Min-Venditti.* ***VIDEO*** *is available at: http://www.youtube.com/watch?v=b9IdZb3z7Jw*
*A copy of the PNAS paper is available at: http://www.pnas.org/content/early/2012/02/07/1116368109.abstract*

***http://www.eurekalert.org/pub_releases/2012-02/uoaf-uoa021312.php***

## U of A medical researchers discover 'very promising' treatment for Huntington disease

### *New potential therapy that restores motor function being planned for clinical trial*

Edmonton - Medical researchers at the University of Alberta have discovered a promising new therapy for Huntington disease that restores lost motor skills and may delay or stop the progression of the disease based on lab model tests, says the lead researcher. Because the new therapy uses a molecule already being used in clinical trials for other diseases, it could be used in a clinical trial for Huntington disease within the next one to two years.

"We didn't expect to see such dramatic changes after administering this therapy," said Simonetta Sipione, the Principal Investigator "We expected to see improvement, but not complete restoration of motor skills. When we saw this, we were jumping with excitement in the lab. This is very promising and should give hope to those with Huntington disease. I think it's a treatment that deserves to go to clinical trials because it could have huge potential."

Those with this inherited brain disorder, where a mutant protein triggers brain cell death causing loss of motor and cognitive skills and eventually death, have slightly lower levels of a brain molecule known as GM1. When U of A medical researchers restored GM1 to normal levels in lab models with the disease, motor skills in the lab models returned to normal within days, said Sipione, a researcher in the Department of Pharmacology and the Centre for Neuroscience, both within the Faculty of Medicine & Dentistry.

Her team's research was published in the peer-reviewed journal Proceedings of the National Academy of Sciences today.

The molecule used in the lab tests at the U of A was produced both naturally and synthetically through chemical production. This same molecule has been used in clinical trials for the treatment of Parkinson's and other neurodegenerative diseases, so using this molecule to treat patients with Huntington disease in a small first stage clinical trial could happen relatively quickly. Details are still being worked out about where the trial would take place, but researchers are hoping it will be at the U of A and are in discussions with a University of Alberta Hospital neurologist.

During the research stage, lab models at the U of A were given the GM1 molecule therapy for four weeks. During the first two weeks after the treatment finished, the lab models still had normal motor function. But after that, motor function started to decline and return to pre-treatment levels by the end of the fourth week. So a potential treatment with this molecule would involve repeated treatments over the long-term, says Sipione.

Sipione and her team are continuing their research to see if restored levels of the GM1 molecule can also reverse cognitive damage in lab models with Huntington disease. They hope to publish the results from these tests within one year. It seems the GM1 therapy improves the way neurons work and makes the mutant huntingtin protein less toxic.

"Because of the way it works, we think it will work on cognitive symptoms of the disease too," says Sipione, a Canada Research Chair Tier 2 in Neurobiology of Huntington disease and an Alberta Innovates-Health Solutions Scholar.

The Huntington Society of Canada funded the research and the CEO said she is excited about the promising results.

"The Huntington Society of Canada is proud to support the excellent research of Dr. Sipione," said Bev Heim-Myers, CEO, Huntington Society of Canada. "Dr. Sipione, for the first time, has demonstrated that in a Huntington disease laboratory model, the treatment reverts the lab model back to normal, not just slightly better.

"It is important to understand that some treatments may work in laboratory models, but not in people. The applicability of the treatment discovered by Dr. Sipione to Huntington disease patients will be determined in clinical trials. We are optimistic that this research demonstrates real potential for a Huntington disease therapy."

http://www.eurekalert.org/pub_releases/2012-02/asoc-fpa021312.php

# First prospective analysis links breast and pancreatic cancer risk with lynch syndrome

***A new prospective study of patients with Lynch syndrome provides the first strong evidence that people with Lynch syndrome face significantly increased risks of breast and pancreatic cancers.***

A new prospective study of patients with Lynch syndrome – an inherited disorder of cancer susceptibility caused by mutations in specific DNA repair genes – provides the first strong evidence that people with Lynch syndrome face significantly increased risks of breast and pancreatic cancers. The study also provided new, clearer estimates of the risks of cancers already recognized to be associated with Lynch syndrome, including those of the colon, uterus, ovary, kidney, stomach and bladder.

The findings – published February 13 in the Journal of Clinical Oncology – may have implications for screening and early detection of cancers in patients with the condition. Additionally, the study showed that relatives of individuals with Lynch syndrome who do not carry a genetic mutation associated with the condition have no increased risk of developing cancer, compared to the general population.

While the risk of pancreatic cancer had been suggested in prior research on Lynch syndrome, the elevated risk of breast cancer was an unexpected finding in this study. "Our study is the first prospective analysis to find a strong association between breast cancer and Lynch syndrome," explained Mark A. Jenkins, PhD, senior author and Associate Professor at The University of Melbourne in Australia. "Further clarification of the risk of breast cancer for women at various ages is needed to determine the recommended age for mammography for each patient, and to determine whether additional tests such as MRI are warranted for women with Lynch syndrome."

DNA errors are a common occurrence within cells. Under normal circumstances, cells are able to correct these errors – for example, by using a set of genes called "mismatch repair genes," which correct a specific type of DNA errors called mismatches. When these errors are not repaired, due to the failure of the mismatch repair genes, additional gene mutations can occur, which can lead to cancer development.

Lynch syndrome is an inherited condition characterized by a mutation in one of the four key mismatch repair genes, and carriers are known to be at high risk for developing cancer, particularly colon cancer. In fact, researchers estimate that three to five of every 100 colon cancers are caused by Lynch syndrome. Individuals with Lynch syndrome are also at greater risk of developing multiple cancers during their lifetime, and tend to be diagnosed with cancer at a younger age than people in the general population.

According to the National Cancer Institute, one to three percent of the population may have Lynch syndrome.

In this study, researchers followed a group of 446 carriers of one of four mismatch repair mutations related to Lynch Syndrome, as well as 1,029 of their relatives who did not carry these mutations ("non-carriers"). Participants were evaluated every five years at recruitment centers affiliated with the Colon Cancer Family Registry in Australia, New Zealand, Canada and the United States. The analysis was conducted by Aung Win, MPH, MBBS, Research Fellow at The University of Melbourne.

After a median follow-up of five years, the researchers found that, compared to the general population, carriers had a 20-fold greater risk of colorectal cancer; a 30-fold greater risk of endometrial (uterine) cancer; a 19-fold higher risk of ovarian cancer; an 11-fold greater risk of renal (kidney) cancer; a 10-fold greater risk of pancreatic, stomach, and bladder cancers; and a four-fold greater risk of breast cancer. They confirmed that carriers who developed cancer also tended to be diagnosed at an earlier age than the general population. The researchers did not find evidence that family members who were non-carriers had any increased risk of cancer, suggesting that they do not need more intense cancer screening than the general population.

"Eventually, we expect that the management of cancer risk, including the choice and timing of screening, will be able to be tailored to the specific underlying gene mutation in a person with Lynch syndrome," said Jenkins.

"Currently, individuals with the syndrome are typically advised to undergo colonoscopy at an earlier age and more frequently than the general population. However, there is no data demonstrating that screening for these other cancers is beneficial, in part due to the absence of effective screening tests."

The researchers are continuing to follow this cohort. Since much larger numbers of carriers are needed to determine cancer risks specific to each of the four genes for Lynch syndrome, they are establishing the International Mismatch Repair Consortium to pool data from 51 clinical research centers in Africa, Asia, Australia, Europe, and North and South America. Collectively, these centers treat more than 7,500 families with Lynch syndrome and over 13,000 mismatch mutation carriers.

**ASCO Perspective:**
*Robert Sticca, MD, ASCO Cancer Communications Committee member, executive editor of CancerProgress.Net and surgical oncologist*
"This study adds to growing evidence linking specific inherited genetic mutations to certain cancers. Additional follow-up will lead to even more accurate estimates of cancer risk for individuals with Lynch syndrome. Such data may help us refine screening guidelines and could ultimately lead to early detection of the cancers these patients are at risk for, preventing the long-term consequences of cancer development. In addition, these data may indicate that some patients with Lynch syndrome are eligible for prophylactic treatments to prevent the development of these cancers."
*Helpful Links from Cancer.Net Lynch Syndrome (http://www.cancer.net/patient/Cancer+Types/Lynch+Syndrome)*

***http://bit.ly/zuf61Y***

## Russian hot springs point to rocky origins for life

***New findings challenge the widespread view that it all kicked off in the oceans. Life may have begun on land instead – just as Darwin thought***

**20:00 13 February 2012 by Colin Barras**

It's a question that strikes at the very heart of one of the deepest mysteries in the universe: how did life begin on Earth? New evidence challenges the widespread view that it all kicked off in the oceans, around deep-sea hydrothermal vents. Instead, hot springs on land, similar to the "warm little pond" favoured by Charles Darwin, may be a better fit for the cradle of life. The controversial new theory suggests the search for extraterrestrial life must go beyond a hunt for alien oceans

Life appeared sometime before 3.8 billion years ago, towards the end of a turbulent phase in our planet's early history dubbed Hadean Earth. Exactly where and how this happened is still a mystery. The first fossils are about 3.4 billion years old, and all we know about life's very first stages comes from chemical signatures in rocks.

This hasn't stopped endless speculation. Conventional wisdom has it that hydrothermal vents on the ocean floor offered an ideal chemical environment for the earliest life. Deep, dark oceans would also have protected the delicate cells from the harmful ultraviolet light that bathed early Earth before the ozone layer formed.

Case closed? Not quite. Armen Mulkidjanian at the University of Osnabrück in Germany says there is a fundamental problem with the ocean floor hypothesis: salt. The cytoplasm found inside all cells contains much more potassium than sodium. Mulkidjanian thinks that chemistry reflects the chemistry of the water life first appeared in, yet salty seawater is sodium-rich and potassium-poor.

"The ancient sea contained the wrong balance of sodium and potassium for the origin of cells," says Mulkidjanian. Now, after extensive field studies, he claims to have found the one place on Earth where that balance is right: in the thermal springs of Kamchatka in far-east Siberia. Mulkidjanian found that puddles condensing from the hydrothermal vapour at Siberia's Mutnovsky thermal springs are potassium-rich, just like cell cytoplasm (Proceedings of the National Academy of Sciences, DOI: 10.1073.pnas.1117774109). Life first appeared in similar pools, says Mulkidjanian.

And while early life would have been damaged if over-exposed to UVs, Mulkidjanian's theory solves another puzzle. Most evolutionary biologists agree that life at this stage would have been little more than floating strands of DNA and RNA. The nucleotides that make up DNA and RNA are all surprisingly stable when exposed to UV light, suggesting they evolved in an environment where UV exposure weeded out all but the most photostable molecules. "You don't get UV light around deep-sea vents," says Mulkidjanian.

"I do not think the oceans were a favourable environment for the origin of life – freshwater ponds seem more favourable," says Nobel laureate Jack Szostak at Harvard University, a key player in the field. "Freshwater ponds have lower salt concentrations, which would allow for fatty acid based membranes to form."

While Darwin's warm little ponds appear to be coming back in vogue, this is a highly polarised field of research and many origin-of-life researchers are not convinced. Nick Lane at University College London disputes the claims that the first cells couldn't cope with life in sodium-rich water. Early cells could have actively pumped out sodium ions, he says. "This is exactly what many methanogens and acetogens do," he points out, referring to microbes that are thought to be among the earliest cellular life forms. This, says Lane, is good evidence that the earliest living cells did indeed actively pump out sodium ions.

Carrine Blank, a geologist at the University of Montana in Missoula says life was unlikely to survive on land 3.8 billion years ago, at a time when meteorites were pummelling Earth. Mulkidjanian counters that some geologists now question whether the late heavy bombardment, as it is known, really happened at that time (Elements, DOI: 10.2113/gselements.5.1.23).

Others contacted by New Scientist labelled Mulkidjanian's ideas absurd and declined to comment. Undoubtedly, most researchers still favour the sea as the cradle of life. Still, Mulkidjanian is not alone in looking for a land-based alternative.

Paul Knauth, a geologist at Arizona State University in Tempe, also thinks life may not have begun in the sea – which he says has ramifications for the search for extraterrestrial life. He has analysed the oxygen isotopes in the silica-rich rocks deposited early in Earth's history, from which you can work out temperatures at the time the rocks formed. He says that the entire planet was much hotter than anyone suspected – surface temperatures of 50 to 80°C may have been common. The seas were also twice as salty as today, because so-called "evaporitic" deposits - which locked away vast quantities of salt - had not begun to form. "The early ocean was a deathtrap of hot salty water," he says. "I like the idea of a non-marine origin."

Then there is the fossil evidence. Although the fossil record doesn't capture events at the origin of life, it does record some slightly later chapters in life's history, which origin-of-life researchers "ignore at their peril", according to Martin Brasier at the University of Oxford. Last year Brasier unearthed the oldest fossils so far: 3.43-billion-year-old bacteria. He found them in Australia, in non-marine rocks that formed on a beach. "I am coming round to the opinion that we may be wrong about the ocean as the mother of life," says Brasier.

This doesn't mean that Mulkidjanian has all the details correct, though. Brasier agrees with Lane that early cells probably could pump out enough sodium from their cytoplasm to survive in sodium-rich environments – so life might have emerged in salty pools or shorelines rather than in Siberian-style thermal springs.

Using observations from living cells to work out what the first cells could – and could not – do underpins most models for life's beginnings. But there will always be a degree of interpretation in how we reconstruct history based on observations of living things, and that leaves room for alternative explanations.

This situation might soon change, though. Brasier's discovery last year paves the way for fossil hunting in even older non-marine rocks – something previously considered a waste of time. Studies of early rocks will take some big steps forward in the coming decade, predicts Brasier. The evidence locked inside them might help settle the debate – and say whether Darwin's hunch was correct after all. "The rock record," says Brasier, "is the only safe witness we have."

**Land ho! The search for ET**

"Follow the water," NASA astrobiologists like to say in conversations about the search for extraterrestrial life. "The problem," says Paul Knauth, a geologist at Arizona State University in Tempe, "is that chlorine follows the water better than any astrobiologist."

Knauth says chlorine-rich salts made the seas on early Earth far too saline for life to emerge. Only once large quantities of salt had evaporated and were locked safely away in land-based deposits could complex life take off in the oceans, suggesting rocks played a key role in life's early stages.

What's more, many of the elements life relies on probably came from the weathering of rocks, like granite, that form only on continents, says Martin Brasier at Oxford University. "If so, the prospects for life on Mars and Titan [where such rocks aren't found] seem a bit bleak."

The same rules probably apply elsewhere in the galaxy. "So, a pale blue dot would be an exciting discovery," says Knauth. "But one with brown spots would be more encouraging."

***http://medicalxpress.com/news/2012-02-treatment-breast-cancer-cells-stem.html***

## Radiation treatment transforms breast cancer cells into cancer stem cells

***Breast cancer stem cells are thought to be the sole source of tumor recurrence and are known to be resistant to radiation therapy and don't respond well to chemotherapy.***

Now, researchers with the UCLA Department of Radiation Oncology at UCLA's Jonsson Comprehensive Cancer Center report for the first time that radiation treatment –despite killing half of all tumor cells during every treatment - transforms other cancer cells into treatment-resistant breast cancer stem cells.

The generation of these breast cancer stem cells counteracts the otherwise highly efficient radiation treatment. If scientists can uncover the mechanisms and prevent this transformation from occurring, radiation treatment for breast cancer could become even more effective, said study senior author Dr. Frank Pajonk, an associate professor of radiation oncology and Jonsson Cancer Center researcher.

"We found that these induced breast cancer stem cells (iBCSC) were generated by radiation-induced activation of the same cellular pathways used to reprogram normal cells into induced pluripotent stem cells (iPS) in regenerative medicine," said Pajonk, who also is a scientist with the Eli and Edythe Broad Center of Regenerative Medicine at UCLA. "It was remarkable that these breast cancers used the same reprogramming pathways to fight back against the radiation treatment."

The study appears Feb. 13, 2012 in the early online edition of the peer-reviewed journal Stem Cells.

"Controlling the radiation resistance of breast cancer stem cells and the generation of new iBCSC during radiation treatment may ultimately improve curability and may allow for de-escalation of the total radiation doses currently given to breast cancer patients, thereby reducing acute and long-term adverse effects," the study states.

There are very few breast cancer stem cells in a larger pool of breast cancer cells. In this study, Pajonk and his team eliminated the smaller pool of breast cancer stem cells and then irradiated the remaining breast cancer cells and placed them into mice.

Using a unique imaging system Pajonk and his team developed to visualize cancer stem cells, the researchers were able to observe their initial generation into iBCSC in response to the radiation treatment. The newly generated iBCSC were remarkably similar to breast cancer stem cells found in tumors that had not been irradiated, Pajonk said.

The team also found that the iBCSC had a more than 30-fold increased ability to form tumors compared to the non-irradiated breast cancer cells from which they originated.

Pajonk said that the study unites the competing models of clonal evolution and the hierarchical organization of breast cancers, as it suggests that undisturbed, growing tumors maintain a small number of cancer stem cells. However, if challenged by various stressors that threaten their numbers, including ionizing radiation, the breast cancer cells generate iBCSC that may, together with the surviving cancer stem cells, repopulate the tumor.

"What is really exciting about this study is that it gives us a much more complex understanding of the interaction of radiation with cancer cells that goes far beyond DNA damage and cell killing," Pajonk said. "The study may carry enormous potential to make radiation even better."

Pajonk stressed that breast cancer patients should not be alarmed by the study findings and should continue to undergo radiation if recommended by their oncologists.

"Radiation is an extremely powerful tool in the fight against breast cancer," he said. "If we can uncover the mechanism driving this transformation, we may be able to stop it and make the therapy even more powerful."

*Provided by University of California - Los Angeles*

***http://www.eurekalert.org/pub_releases/2012-02/ors-ifm021412.php***

## Immunization for MRSA on the horizon

### *New hope for total joint replacement patients*

Methicillin resistant staph aureus (MRSA) infections are resistant to antibiotics and can cause a myriad of problems -- bone erosion, or osteomyelitis, which shorten the effective life of an implant and greatly hinder replacement of that implant. MRSA can result in prolonged disability, amputation and even death.

Although only 2 percent of the American population that undergo total joint replacement surgery will suffer an infection, half of those infections are from MRSA. The results of a MRSA infection after a total joint replacement can be devastating. Currently, there is no effective treatment for MRSA-infected implants. With the increasing incidence of total joint replacement surgeries, the prevalence of MRSA-infected implants is expected to rise.

A team of investigators from the University of Rochester Medical Center has developed a vaccine that can prevent bacterial infection of orthopaedic implants. Their findings were presented at the Orthopaedic Research Society (ORS) 2012 Annual Meeting in San Francisco, California.

The team, led by Edward Schwarz, PhD, Professor of Orthopaedics and Associate Director of the Center for Musculoskeletal Research, has generated an antibody that prevents MRSA bacteria from dividing properly.

"What makes the staph such a challenging pathogen is that is has an ironclad cell wall. But that is also its Achilles' heel," Dr. Schwarz said. He explained that if the cell wants to divide, it has to "unzip the cell wall" to break into two "daughter cells." Their team produced an antibody that targets a component of the zipper, Gmd - preventing normal bacterial cell division by causing them to form clusters of cells.

The researchers tested the antibody prior to implantation of a MRSA-infected pin to simulate an infected joint replacement. They monitored bacterial growth and found that their antibody protected 50 percent of their sample from infection. Further analysis found that the antibody prevented formation of sequestrum, or a piece of dead bone, which is a hallmark of osteomyelitis. Additionally, immunization led to decreased bacterial presence on the pins themselves.

Based on these findings, this immunization appears to be a promising treatment to prevent the MRSA infection/reinfection of orthopaedic implants.

Dr. Schwarz and his team were recently awarded a five-year multimillion dollar grant from AOTrauma, a not for profit Swiss foundation, for the Clinical Priority Program grant on infection. This grant deals with the diagnosis, treatment, prevention, and education about musculoskeletal infection.

http://www.eurekalert.org/pub_releases/2012-02/eu-nco021412.php

## New class of potential drugs inhibits inflammation in brain

***Scientists at Emory University School of Medicine have identified a new group of compounds that may protect brain cells from inflammation linked to seizures and neurodegenerative diseases.***

**Writer: Quinn Eastman**

The compounds block signals from EP2, one of the four receptors for prostaglandin E2, which is a hormone involved in processes such as fever, childbirth, digestion and blood pressure regulation. Chemicals that could selectively block EP2 were not previously available. In animals, the EP2 blockers could markedly reduce the injury to the brain induced after a prolonged seizure, the researchers showed. The results were published online this week in the Proceedings of the National Academy of Sciences Early Edition.

"EP2 is involved in many disease processes where inflammation is showing up in the nervous system, such as epilepsy, stroke and neurodegenerative diseases," says senior author Ray Dingledine, PhD, chairman of Emory's Department of Pharmacology. "Anywhere that inflammation is playing a role via EP2, this class of compounds could be useful. Outside the brain, EP2 blockers could find uses in other diseases with a prominent inflammatory component such as cancer and inflammatory bowel disease."

Prostaglandins are the targets for non-steroid anti-inflammatory drugs (NSAIDs) such as aspirin and ibuprofen. NSAIDSs inhibit enzymes known as cyclooxygenases, the starting point for generating prostaglandins in the body. Previous research indicates that drugs that inhibit cyclooxygenases can have harmful side effects. For example, sustained use of aspirin can weaken the stomach lining, coming from prostaglandins' role in the stomach. Even drugs designed to inhibit only cyclooxygenases involved in pain and inflammation, such as Vioxx, have displayed cardiovascular side effects.

Dingledine's team's strategy was to bypass cyclooxygenase enzymes and go downstream, focusing on one set of molecules that relay signals from prostaglandins. Working with Yuhong Du in the Emory Chemical Biology Discovery Center, postdoctoral fellows Jianxiong Jiang, Thota Ganesh and colleagues sorted through a library of 262,000 compounds to find those that could block signals from the EP2 prostaglandin receptor but not related receptors. One of the compounds could prevent damage to neurons in mice after "status epilepticus," a prolonged drug-induced seizure used to model the neurodegeneration linked to epilepsy.

The team found that a family of related compounds had similar protective effects.

Dingledine says that the compounds could become valuable tools for exploring new ways to treat neurological diseases. However, given the many physiological processes prostaglandins regulate, more tests are needed, he says. Prostaglandin E2 is itself a drug used to induce labor in pregnant women, and female mice engineered to lack the EP2 receptor are infertile, so the compounds would need to be tested for effects on reproductive organs, for example.

*The research was supported by the National Institutes of Health.*

***Reference**: J. Jiang et al. Small molecule antagonist reveals seizure-induced mediation of neuronal injury by prostaglandin E2 receptor subtype EP2. PNAS 2012, doi:10.1073/pnas.1120195109*

http://www.eurekalert.org/pub_releases/2012-02/bu-wnm021312.php

## When nerve meets muscle, biglycan seals the deal

***Research identifies protein with potential relevance to motor neuron diseases***

PROVIDENCE, R.I. [Brown University] - A protein that has shown early promise in preventing the loss of muscle function in mouse models of Duchenne muscular dystrophy, has been found in a new study to be a key player in the process of joining nerves to muscles.

The protein biglycan needs to be present to stabilize synapses at the neuromuscular junction after they have formed, according to research led by Brown University that appears in the Feb. 14, 2012, issue of the Journal of Neuroscience.

"What neuromuscular junctions do second-by-second is essential for our brain to control movement and they are also important for the long-term health of both muscle and motor neurons," said Justin Fallon, professor of neuroscience at Brown University and the paper's senior author. "A treatment that sustains or supports the synapse could promote the health of motor neurons and muscle."

In previous work, Fallon, a member of the Brown Institute for Brain Science, has shown that in mice with the same genetic mutation as Duchenne patients, biglycan promotes the activity of another natural protein, utrophin, that can significantly reduce the muscle degradation that patients suffer. Utrophin essentially takes over for dystrophin, which is the protein Duchenne patients cannot produce. In 2010 Brown licensed Fallon's biglycan intellectual property to the Providence startup company Tivorsan Pharmaceuticals, which is working toward human trials of biglycan.

Last month the Muscular Dystrophy Association, which helped support Fallon's new study, gave Tivorsan a $1 million grant.

Now Fallon's research group has found another important role for biglycan. In the new multi-institutional study, lead author Alison Amenta and a team of other scientists found that biglycan binds and helps activate and target a receptor enzyme called MuSK, which works like a foreman or master regulator over other proteins that build and stabilize the neuromuscular junction.

Mice engineered to lack biglycan developed normal junctions at first, but by five weeks after birth their synapses became much more likely to break into fragmented shadows of their former selves. In experiments the scientists saw that up to 80 percent of synapses in biglycan-lacking mice were unstable.

Biglycan-lacking mice also showed other structural defects including misaligned neurotransmitter receptors and extra folds near synapses. "We think it is most likely that these folds are remnants of previous synaptic sites," that have since withered, the authors wrote in the paper.

Amenta, Fallon, and their team also found that in mice lacking biglycan, levels of MuSK at neuromuscular junction synapses were reduced by a factor of more than 10. In another experiment, they found that recombinant biglycan could rescue the stability of synaptic structures in model cell culture system.

**Relevance to motor neuron diseases**

The findings help set the stage for testing biglycan as a potential therapy in animal models of motor neuron disease, Fallon said. "As an extracellular protein that can be delivered systemically that acts to stabilize the neuromuscular junction, we propose that biglycan could be a protein therapeutic for motor neuron diseases such as spinal muscular atrophy and amyotrophic lateral sclerosis, or ALS," Fallon said.

*In addition to Fallon and Amenta, other Brown authors include Hillary Creely, Mary Lynn Mercado, Hiroki Hagiwara, Beth McKechnie and Beatrice Lechner. Other authors are Susana Rossi, Emilio Marrero and Richard Rotundo of the University of Miami; Qiang Wang and Lin Mei of the Medical College of Georgia; Rick Owens and David McQuillan of Lifecell Corp.; the late Werner Hoch of the University of Houston; and Marian Young of the National Institute of Dental and Craniofacial Research.*

*Several grants from the National Institutes of Health and support from the Muscular Dystrophy Association funded the research.*

***http://arstechnica.com/science/news/2012/02/fossil-hips-dont-lie-rock-record-got-a-bad-rap.ars***

## The rock record got a bad rap. Fossil diversity accurately reflects history

***Does the fossil record we see reflect the state of ancient ecosystems, or is it just the readout from a defective instrument? A recent paper in Science gives reassuring support to the fidelity of the rock record.***

**By Scott K. Johnson | Published 3 days ago**

Say an EKG machine is monitoring your heart, when it suddenly flatlines. You'd be keenly interested to know whether your heart had stopped or the machine had simply gone on the fritz. Paleontologists have faced a similar (if slightly less urgent) puzzle when it comes to the geologic record of life: does the fossil record we see reflect the state of ancient ecosystems, or is it just the readout from a defective instrument? A recent paper in Science gives reassuring support to the fidelity of the rock record.

It’s fascinating to study how species diversity has changed through time, since we can see the effects of major events in Earth’s past and watch evolution play out. It's literally reading the history of life on Earth. That’s a story we naturally want to know and tell. But fossils are difficult to come by - after all, less than one percent of extinct species are represented in the fossil record. As an imperfect recorder, we have to worry how much the evidence in the rocks is telling us about the organisms, and how much we're just seeing changes in the rocks themselves.

Prof. Shanan Peters, co-author of the recent paper, explained to Ars by e-mail that paleontologists have wrestled with this for a long time. "This goes right back to Darwin... The Origin of Species even has a chapter titled 'On the imperfection of the geological record,' with a subheading titled 'On the poorness of our paleontological collections.'"

A paper by David Raup in 1972 set out the problem for his fellow researchers. While it was tempting to interpret trends and patterns in the fossil record of diversity, Raup showed that it was important to look carefully for preservation biases. If the formation of sedimentary rocks decreases for a time, perhaps the fossil diversity would also decrease even if there was no change in the actual ecosystems.

"Raup’s novel comparison of global compilations of rock quantity to diversity estimates based on the stratigraphic ranges of fossil taxa yielded compelling correlations that did 'justify further investigation,'" Peters wrote. Since the paper was published, researchers have worked on ways to account for variations in preservation when estimating fossil diversity.

In this new study, Peters and co-author Prof. Bjarte Hannisdal re-examined the sampling bias in the rock record using sophisticated statistics that measure information transfer. "Basically [information transfer] allows us to do two things," Peters explained. "First, it allows us to evaluate the statistical dependence between two variables (e.g., fossil diversity and temperature) beyond their mutual relationship to a third variable (e.g., sea level). In this sense, it is like a partial correlation, with the advantage that the variables don't have to be linearly related. Second, it allows us to determine the directionality of coupling between two variables (e.g., does the information in temperature flow into diversity?)"

This provides a window into the nature of the correlation between rock formation and species diversity. Are trends in species diversity really caused by changes in rock formation, or are they both responding to some other factor? The researchers compared records of marine diversity, marine sedimentary rock formation, sea level, and isotopes of carbon, oxygen, sulfur, and strontium.

Carbon-13 tracks changes in the carbon cycle, such as sequestration in wetlands or volcanic emissions. Oxygen-18, which provides a proxy for temperature in ice cores, shows changes in climate in carbonate rocks. Sulfur-34 records how heavily oxidized the environment was, among other things. The ratio of strontium-87 to strontium-86 is used to examine changes in continental weathering and volcanism at mid-ocean ridges.

They found that our record of species diversity might not be as bad as we thought. Changes in sedimentation and species diversity do correlate, but that's because they both respond to other geological forces. "Raup was right, in that there is a strong correlation between the variable rock record and fossil diversity," wrote Peters, "but he was wrong about the meaning of that correlation. It exists because the biosphere actually responds to the environmental changes that are responsible for producing variability in the marine sedimentary rock record."

Our record of the history of life isn't strongly biased by preservation; it's actually a pretty faithful telling. The results showed that the carbon cycle was a driver of climate (no surprise there) as well as the sulfur record, which was also related to sea level. Sea level exerted control on fossil diversity (probably due to the species-area effect), while the formation of marine sedimentary rocks is, of course, also related to sea level. But critically, sediment formation did not drive fossil diversity.

Peters suggested that some analytic techniques meant to adjust for changes in geology may have actually created their own biases. "This is so cool because paleontologists have been freaking out about the variability of the rock record and fossil record for a long time, and they have spent a great deal of energy trying to remove this variability analytically by sampling standardization techniques," Peters noted. "[Information transfer] analysis clearly shows that [those techniques do] a very good job of removing the signal of sea level, but that sea level actually contains an important signal! The great irony may end up being that the face-value fossil record is our best estimate of the true history of life."

Analyzing the relationships between all these parameters could tell us a lot about the mechanisms by which environmental changes have affected life on Earth. A major event that prominently affects many records is the formation and subsequent breakup of the supercontinent Pangaea, which involved the closing and re-opening of ocean basins. Many smaller-scale and less obvious events that affected fossil diversity remain poorly understood.

In the interim, the study suggests that paleontologists don't have to worry that their understanding of the history of fossil diversity comes from a dodgy translation. "In short," Peters wrote, "there is a really important signal about the state of the Earth that is captured quantitatively by Darwin's geological incompleteness."

*Science, 2012. DOI: 10.1126/science.1210695 (About DOIs).*

***http://www.sciencedaily.com/releases/2012/02/120214100554.htm***

## Turmeric-Based Drug Effective On Alzheimer Flies

### *Curcumin prolongs life and enhances activity of fruit flies with a nervous disorder similar to Alzheimers*

ScienceDaily - Curcumin, a substance extracted from turmeric, prolongs life and enhances activity of fruit flies with a nervous disorder similar to Alzheimers, according to new research. The study conducted at Linköping University, indicates that it is the initial stages of fibril formation and fragments of the amyloid fibrils that are most toxic to neurons.

Ina Caesar, as the lead author, has published the results of the study in the journal PLoS ONE.

For several years curcumin has been studied as a possible drug candidate to combat Alzheimer's disease, which is characterized by the accumulation of sticky amyloid-beta and Tau protein fibres. Linköping researchers wanted to investigate how the substance affected transgenic fruit flies (Drosophila melanogaster),

which developed evident Alzheimer's symptoms. The fruit fly is increasingly used as a model for neurodegenerative diseases.

Five groups of diseased flies with different genetic manipulations were administered curcumin. They lived up to 75 % longer and maintained their mobility longer than the sick flies that did not receive the substance.

However, the scientists saw no decrease of amyloid in the brain or eyes. Curcumin did not dissolve the amyloid plaque; on the contrary it accelerated the formation of fibres by reducing the amount of their precursor forms, known as oligomers.

"The results confirm our belief that it is the oligomers that are most harmful to the nerve cells," says Professor Per Hammarstrom, who led the study.

"We now see that small molecules in an animal model can influence the amyloid form. To our knowledge the encapsulation of oligomers is a new and exciting treatment strategy," he said.

Several theories have been established about how oligomers can instigate the disease process. According to one hypothesis, they become trapped at synapses, inhibiting nerve impulse signals. Others claim that they cause cell death by puncturing the cell membrane.

Curcumin is extracted from the root of herbaceous plant turmeric and has been used as medicine for thousands of years. More recently, it has been tested against pain, thrombosis and cancer.

***http://medicalxpress.com/news/2012-02-weight-loss-contagious.html***

## Weight loss can be contagious, study suggests

### *Is weight loss "contagious"?*

According to a new study published online in the journal Obesity, teammates in a team-based weight loss competition significantly influenced each other's weight loss, suggesting that shedding pounds can have a ripple effect. Researchers from The Miriam Hospital's Weight Control and Diabetes Research Center and The Warren Alpert Medical School of Brown University found that team members not only achieved similar weight loss outcomes, but participants who said their teammates played a large role in their weight loss actually lost the most weight.

"We know that obesity can be socially contagious, but now we know that social networks play a significant role in weight loss as well, particularly team-based weight loss competitions," said lead author Tricia Leahey, Ph.D., of The Miriam Hospital and Alpert Medical School. "In our study, weight loss clearly clustered within teams, which suggests that teammates influenced each other, perhaps by providing accountability, setting expectations of weight loss, and providing encouragement and support."

Obesity remains a common, serious and costly disease in the United States. About one-third of American adults are obese, according to the Centers for Disease Control and Prevention, and no state has met the nation's Healthy People 2010 goal to lower obesity prevalence to 15 percent. Obesity and its associated health problems, including heart disease and diabetes, continue to have a significant economic impact on the U.S. health care system, costing the nation hundreds of billions of dollars each year.

To promote cost-effective weight loss initiatives, online team-based weight loss interventions are increasing in popularity as a way to encourage weight loss in large groups of people. The current study is the first to examine the effects of teammates and social influence on individual weight loss during one of these weight loss competitions.

The findings are based on the results of the 2009 Shape Up Rhode Island (SURI) campaign, a 12-week statewide online weight loss competition designed by study co-author Rajiv Kumar, M.D. Participants joined with a team and could compete against other teams in three divisions: weight loss, physical activity and pedometer steps. The weight loss competition included 3,330 overweight or obese individuals (BMI of 31.2 or greater), representing 987 teams averaging between 5 and 11 members each. The majority of these individuals enrolled in all three divisions.

Weight loss outcomes were clearly determined by which team an individual was on. Participants who lost clinically significant amounts of weight (at least 5 percent of their initial body weight) tended to be on the same teams, and being on a team with more teammates in the weight loss division was also associated with a greater weight loss. Individuals who reported higher levels of teammate social influence increased their odds of achieving a clinically significant weight loss by 20 percent. This effect was stronger than any other team characteristic, Leahey said.

"This is the first study to show that in these team-based campaigns, who's on your team really matters," she added. "Being surrounded by others with similar health goals all working to achieve the same thing may have really helped people with their weight loss efforts."

However, Leahey noted that individual characteristics were also associated with weight outcomes. Obese individuals had a greater percentage of weight loss than overweight participants. Team captains also lost more weight than team members, possibly due to their increased motivation and engagement in the campaign. Leahey says that future weight loss team competitions may consider requiring team members to share the leadership role.

"We're all influenced by the people around us, so if we can harness this positive peer pressure and these positive social influences, we can create a social environment to help encourage additional weight loss," she said. *Provided by Lifespan*

***http://www.physorg.com/news/2012-02-prions-ozone.html***

## Killing prions with ozone

***When it comes to infectious agents, it doesn't get much worse than prions.***

These misfolded proteins are highly resistant to a wide variety of extreme disinfectant procedures. They have been identified as the culprits behind mad cow disease and chronic wasting disease in animals and humans, and are also implicated in Creutzfeldt-Jakob disease and other prion-related disorders.

But an interdisciplinary University of Alberta research team has come a step closer to finding a way of inactivating these highly infectious proteins. The team, lead by environmental health professors Mike Belosevic and Norm Neumann from the School of Public Health and engineering professor Mohamed Gamal El-Din from the Department of Civil and Environmental Engineering, have demonstrated for the first time that prions are highly susceptible to molecular ozone.

The discovery could have implications for decontaminating medical and dental surgical instruments or treating water and wastewater in settings where prions might appear, such as in slaughterhouse waste.

"Although we know that they have a very high-level resistance, it's possible that we've discovered their Achilles' heel," said Neumann. "This means there might be simple solutions to dealing with contaminated medical instruments and waste products from slaughterhouses."

Human transmission of these devastating infectious agents through patient exposure to surgical equipment and blood transfusions has been documented. If these proteins can be neutralized, the result will be improved patient care.

"Because ozone is already commonly used in the hospital environment, the technology for this disinfection process already exists," says Neumann. "It is possible to take a medical instrument, put it in an ozone bath and very quickly destroy 99.99% of the prions that are there."

However, there is still much work to do. "The only proof of final inactivation is to actually infect animals, and it may take years for the animal to start demonstrating the behavioural changes associated with these diseases caused by prions," says Neumann. "We need more research in this area to increase our understanding of the relationship between ozone and all types of prions, including bovine spongiform encephalopathy or BSE, and that's what we're working on now."

The interdisciplinary nature of the research proved to be crucial to the success. "Nobody has really taken the biological diagnostics and methods and then applied them in the engineering context, and that's what we did here," Neumann said. The importance of the interdisciplinary approach to this research is echoed by Gamal El-Din. "We have the expertise in microbiology and engineering to make a difference. The ultimate goal is to protect the health of people as well as the environment."

*The research was funded in part by the Alberta Prion Research Institute, PrioNet Canada and the Natural Sciences and Engineering Research Council of Canada and published in the February issue of the journal Applied and Environmental Microbiology.* ***More information:*** ***aem.asm.org … e557b232ed57*** *Provided by University of Alberta*

***http://www.sciencedaily.com/releases/2012/02/120215083025.ht***

## Pocket Microscope With Accessory for Ordinary Smart Phone

***A pocket-size microscope accessory developed by Finnish scientists will be accurate to one hundredth of a millimetre.***

ScienceDaily - VTT Technical Research Centre of Finland has developed an optical accessory that turns an ordinary camera phone into a high-resolution microscope. The device is accurate to one hundredth of a millimetre. Among those who will benefit from the device are the printing industry, consumers, the security business, and even health care professionals. A new Finnish enterprise called KeepLoop Oy and VTT are already exploring the commercial potential of the invention.

The first industrial applications and consumer models will be released in early 2012. The operation of the device is based on images produced by the combined effect of an LED light and an optical lens. Various surfaces and structures can be examined in microscopic detail and the phone's camera used to take sharp, high-resolution images that can be forwarded as MMS messages.

An ordinary mobile phone turns into an instant microscope by attaching a thin, magnetic microscope module in front of the camera's normal lens. The device fits easily in the user's pocket, unlike conventional tubular microscopes.

The plastic macro lens of the mobile phone microscope magnifies objects effectively. The camera's field of view is 2 x 3 millimetres. A number of LEDs have been sunk into the outer edge of the lens, allowing objects to be illuminated from different angles.

***Mobile microscope.*** **(Credit: Image courtesy of VTT Technical Research Centre of Finland)**

Images illuminated from several different angles could be used to produce 3D topographic maps, for example, with mobile phone software. The 3D maps would be accurate to one hundredth of a millimetre. The competitive edge of the product is based on next-generation lens technology, the compact and user-friendly structure, and customisable extra features.

The mobile phone microscope is suited for examining surfaces and surroundings, for security, health care, and even games. The mobile phone microscope could also be used to study surface formations, especially in the printing industry as part of quality control and in field conditions. In the security business the device could be used, for example, to read microcode in various logistics systems, while it is also suited for studying security markings, and for authenticating products as genuine as part of brand protection. The microscope is capable of detecting hidden symbols in products that are not visible to the naked eye.

The device can also be applied to study of the environment. Consumers could use the instant microscope when out and about to examine the leaves of trees and plants, for example, or study insects. Another potential application is in examining textile structures such as strands of hair, or the fibrous structure of paper. The device also has uses in social media, and in community-based hybrid media where traditional forms of media are used in combination with each other. There are also several potential applications in the gaming world.

*Story Source: The above story is reprinted from materials provided by VTT Technical Research Centre of Finland.*

***http://www.eurekalert.org/pub_releases/2012-02/aaon-hfy020712.php***

## How fast you walk and your grip in middle age may predict dementia, stroke risk

### *Simple tests such as walking speed and hand grip strength may help doctors determine how likely it is a middle-aged person will develop dementia or stroke.*

NEW ORLEANS - That's according to new research that was released today and will be presented at the American Academy of Neurology's 64th Annual Meeting in New Orleans April 21 to April 28, 2012. "These are basic office tests which can provide insight into risk of dementia and stroke and can be easily performed by a neurologist or general practitioner," said Erica C. Camargo, MD, MSc, PhD, with Boston Medical Center.

More than 2,400 men and women with an average age of 62 underwent tests for walking speed, hand grip strength and cognitive function. Brain scans were also performed. During the follow-up period of up to 11 years, 34 people developed dementia and 70 people had a stroke.

The study found people with a slower walking speed in middle age were one-and-a-half times more likely to develop dementia compared to people with faster walking speed. Stronger hand grip strength was associated with a 42 percent lower risk of stroke or transient ischemic attack (TIA) in people over age 65 compared to those with weaker hand grip strength. This was not the case, however, for people in the study under age 65.

"While frailty and lower physical performance in elderly people have been associated with an increased risk of dementia, we weren't sure until now how it impacted people of middle age," said Camargo.

Researchers also found that slower walking speed was associated with lower total cerebral brain volume and poorer performance on memory, language and decision-making tests. Stronger hand grip strength was associated with larger total cerebral brain volume as well as better performance on cognitive tests asking people to identify similarities among objects. "Further research is needed to understand why this is happening and whether preclinical disease could cause slow walking and decreased strength," said Camargo.

*The study was supported by the National Heart, Lung and Blood Institute's Framingham Heart Study and by the National Institute of Neurological Disorders and Stroke and the National Institute on Aging.*

http://www.eurekalert.org/pub_releases/2012-02/wifb-ppp021312.php

# Prions play powerful role in the survival and evolution of wild yeast strains

***Prions, the much-maligned proteins most commonly known for causing "mad cow" disease, are commonly used in yeast to produce beneficial traits in the wild.***

**Written by Matt Fearer**

CAMBRIDGE, Mass. - Moreover, such traits can be passed on to subsequent generations and eventually become "hard-wired" into the genome, contributing to evolutionary change.

Prions were first found to produce heritable new traits more than a decade ago in laboratory studies of simple baker's yeast. The key discovery then was that some proteins could spontaneously switch from a normal shape into a self-perpetuating prion conformation. The switch to the prion state alters protein function, which can result in the appearance of new traits, some helpful, some detrimental. Sophisticated cellular machinery ensures that replicating prion templates are chopped into pieces that can be passed to daughter cells during cell division. Importantly, the rate at which proteins switch into and out of the prion state increases in response to environmental stress, suggesting that they are part of an inherent survival mechanism that helps yeasts adapt to changes in their surroundings.

Yet, as compelling as the case for this protein-based mechanism of inheritance is, its biological significance has been hotly debated for one key reason: prions capable of modifying phenotypes have never been found in nature. Until now.

In a massive undertaking, Whitehead Institute scientists have tested nearly 700 wild yeast strains isolated from diverse environments for the presence of known and unknown prion elements, finding them in one third of all strains. All the prions appear capable of creating diverse new traits, nearly half of which are beneficial. These unexpected findings, reported in this week's edition of the journal Nature, stand as strong evidence against the common argument that prions are merely yeast "diseases" or rare artifacts of laboratory culture.

"A huge amount of effort has gone into studying this paradigm-shifting mode of inheritance, but with no real understanding of whether it's genuinely important biologically," says Daniel Jarosz, co-first author of the Nature paper and a postdoctoral researcher in the lab of Whitehead Member Susan Lindquist. "Now it seems clear they do influence the way natural yeasts cope with changing environments and evolve in response to stress."

The hunt for prions in wild yeast strains began in the Lindquist lab when Jarosz and Randal Halfmann, then an MIT graduate student and now a fellow at the University of Texas Southwestern Medical Center, gathered hundreds of wild strains from stock centers all over the world. They then conducted a chemical screen for one well-studied prion, [PSI+], and found it in 10 wild strains. Genetic manipulations confirmed its status as a true prion. They then observed the effects of [PSI+] on biological traits by eliminating the prion conformation in these strains and exposing them to natural stresses, such as high acidity and the presence of antifungal agents. In one strain isolated from Beaujolais wine, for example, the prion resulted in the emergence of traits that could be beneficial or detrimental, depending upon environmental conditions. Another well-known prion, [MOT3+] was found in six wild strains.

To determine whether the wild yeasts might harbor other unknown prion elements, Jarosz and Halfmann exposed sets of cultures of all the wild strains to the same chemical protocol that switched [PSI+] and [MOT3+] out of their prion states. In all, 255 strains demonstrated different phenotypes under varying stressors after this treatment. "We certainly didn't expect to see this much prion-based phenotypic diversity," Jarosz says. "It's remarkable." Another surprise was that approximately 40% of the traits produced by the wild prions proved to beneficial to growth in the dozen different environmental conditions tested.

"How frequently beneficial they are suggests that the prions have already been subject to previous, positive selective events," says Lindquist. "We see them as part of a bet-hedging strategy that allows the yeast to alter their biological properties quickly when their environments turn unfavorable."

Convinced of the impact prions have had on yeast evolution, Lindquist speculates that these shape-shifting proteins may be "remnants of early life," from a time when inheritance was predominantly protein-based rather than nucleic-acid based. She also theorizes that prions may play such roles beyond yeast, and her lab intends to take similar approaches in the hunt for prion activity in other organisms.

*This work was supported by grants from the G. Harold and Leila Y. Mathers Foundation, HHMI, the Damon Runyon Cancer Research Foundation and the National Institutes of Health (NIH Pathway to independence award).*

***Full Citation:*** *"Prions are a common mechanism for phenotypic inheritance in wild yeasts" Nature, February 16, 2012 Randal Halfmann (1,2), Daniel F. Jarosz (1), Sandra K. Jones (1), Amelia Chang (1,2), Alex K. Lancaster (1), and Susan Lindquist (1,2,3)*

http://www.eurekalert.org/pub_releases/2012-02/wuso-dms021512.php

# Diabetes may start in the intestines, research suggests

***Scientists at Washington University School of Medicine in St. Louis have made a surprising discovery about the origin of diabetes.***

Their research suggests that problems controlling blood sugar - the hallmark of diabetes - may begin in the intestines. The new study, in mice, may upend long-held theories about the causes of the disease. Because insulin is produced in the pancreas and sugar is stored in the liver, many scientists have looked to those organs for the underlying causes of diabetes. The findings are reported Feb. 16 in the journal Cell Host & Microbe.

In the new research, scientists studied mice that are unable to make fatty acid synthase (FAS) in the intestine. FAS, an enzyme crucial for the production of lipids, is regulated by insulin, and people with diabetes have defects in FAS. Mice without the enzyme in the intestines develop chronic inflammation in the gut, a powerful predictor of diabetes. "Diabetes may indeed start in your gut," says principal investigator Clay F. Semenkovich, MD. "When people become resistant to insulin, as happens when they gain weight, FAS doesn't work properly, which causes inflammation that, in turn, can lead to diabetes."

First author Xiaochao Wei, PhD, and Semenkovich, the Herbert S. Gasser Professor of Medicine, professor of cell biology and physiology and director of the Division of Endocrinology, Metabolism and Lipid Research, collaborated with specialists in gastroenterology and genome sciences to determine what happens in mice that can't make FAS in their intestines. "The first striking thing we saw was that the mice began losing weight," says Wei, a research instructor in medicine. "They had diarrhea and other gastrointestinal symptoms, and when we looked closely at the tissue in the gut, we found a lot of inflammation."

Initially, the researchers thought that the mice became sick because of changes to the mix of microbes that naturally live in the gut, where they help digest food and synthesize vitamins.

In collaboration with Jeffrey I. Gordon, MD, director of the Center for Genome Sciences and Systems Biology at the School of Medicine, they looked more closely at gut microbes in the mice.

"The mice had substantial changes in their gut microbiome," Semenkovich says. "But it wasn't the composition of microbes in the gut that caused the problems."

Instead, Wei says, the mice got sick because of a defect in fatty acid synthase. The mice without fatty acid synthase had lost the protective lining of mucus in the intestines that separates the microbes from direct exposure to cells. This allowed bacteria to penetrate otherwise healthy cells in the gut, making the mice sick.

In a further collaboration with Nicholas O. Davidson, MD, director of the Division of Gastroenterology, the researchers found gastrointestinal effects resembling some features of inflammatory bowel disease. Other investigators studying humans with ulcerative colitis had previously made the unexplained observation that colon biopsies from these patients have low amounts of fatty acid synthase.

"Fatty acid synthase is required to keep that mucosal layer intact," Wei says. "Without it, bad bacteria invade cells in the colon and the small intestine, creating inflammation, and that, in turn, contributes to insulin resistance and diabetes."

Inflammation and insulin resistance reinforce each other. Inflammatory substances can cause insulin resistance and inhibit the production of insulin, both of which interfere with the regulation of blood sugar. In turn, insulin resistance is known to promote inflammation. Further study showed that the ability to build the thin, but important, layer of mucosal cells was hindered by faulty FAS.

That the gut is so important to the development of diabetes makes sense because many people with the condition not only have faulty FAS, but they also frequently develop gastrointestinal difficulties, Semenkovich says. "Abdominal pain and diarrhea are some of the most common problems we see in people with diabetes," he says. "We could only connect these 'dots' because other experts at the university could help us link what we observed in these mice to what occurs in patients with diabetes and inflammatory bowel disease," Semenkovich says.

Semenkovich and Wei say much more study is needed, but they say that FAS and a key component of the intestinal mucosa called Muc2 may be potential targets for diabetes therapy. They now plan to study people with diabetes to see whether FAS is altered in a similar way, producing damage to the mucosal layer in the intestines.

*Wei X, Yang Z, Rey FE, Ridaura VK, Davidson, NO, Gordon JI, Semenkovich CS, Fatty acid synthase modulates intestinal barrier function through palmitoylation of mucin2. Cell Host & Microbe, Feb. 16, 2012.*

*Funding for this research comes from grants awarded by the National Institute on Diabetes and Digestive and Kidney Diseases and the National Heart, Lung, and Blood Institute of the National Institutes of Health (NIH), by the American Heart Association and by the American Diabetes Association.*

http://news.discovery.com/history/sheba-queen-gold-121502.html#mkcpgn=rssnws1

## Queen of Sheba's Lost Gold Mine Discovered?

***A British archaeologist claims she may have uncovered the treasure mine from which the fabled Queen of Sheba drew her wealth, the UK daily The Observer reported.***

**By Rossella Lorenzi | Wed Feb 15, 2012 03:39 PM ET**

Hidden on a hill on the Gheralta plateau in northern Ethiopia, the unexplored mine lies within the Queen's former territory, a nearly 3,000-year-old Sheba kingdom that scholars believe spanned modern-day Ethiopia and Yemen.

"One of the things I've always loved about archaeology is the way it can tie up with legends and myths. The fact that we might have the Queen of Sheba's mines is extraordinary," Louise Schofield, an archaeologist and former British Museum curator, told The Observer.

According to the Old Testament, the Queen of Sheba travelled from her mysterious kingdom to meet King Salomon in Jerusalem "with a very great train, with camels that bear spices, and very much gold, and precious stones."

The Queen was "overwhelmed" by Solomon's wisdom and the splendour of his kingdom. As she departed, "she gave the king 120 talents of gold" - the equivalent of four-and-a-half tons.

***The Visit of the Queen of Sheba to King Solomon. Edward Poynter (1836–1919), Art* Gallery of New South Wales. Credit: Wikimedia Commons.**

Legend has it that she gave birth to Solomon's child shortly after their passionate encounter and that descendants of their child, Menelik, became the kings of Abyssinia.

Schofield found the ancient mine hidden behind a 20-foot wide that was carved with a sun and crescent moon, the "calling card of the land of Sheba," according to Schofield.

"I crawled beneath the stone -- wary of a 9-foot cobra I was warned lives here -- and came face to face with an inscription in Sabaean, the language that the Queen of Sheba would have spoken," she said.

Buried about four feet beneath the surface of a hill circled by vultures, the shaft featured an ancient skull embedded above the entrance. This boasted Sabaean chiselling, according to Schofield.

She said that the structure had gone unnoticed despite the fact that locals still pan for gold in a nearby river.

Not far away, the archaeologist discovered remains of columns and other finely carved stones possibly belonging to a buried temple believed to be dedicated to a moon deity of Sheba.

The site of a battlefield, complete with ancient bones, was also unearthed nearby.

The precise size of the mine, whose entrance is blocked by boulders, has not yet been determined, but tests by a gold prospector suggest it is extensive, with underground passages large enough to walk inside.

A full excavation of the site will begin as soon as Schofield secures the necessary fundings.

http://www.eurekalert.org/pub_releases/2012-02/f-sf-mod021612.php

## Microbial oasis discovered beneath the Atacama Desert

***Two metres below the surface of the Atacama Desert there is an 'oasis' of microorganisms.***

Researchers from the Center of Astrobiology (Spain) and the Catholic University of the North in Chile have found it in hypersaline substrates thanks to SOLID, a detector for signs of life which could be used in environments similar to subsoil on Mars.

Life is bustling under the driest desert on Earth. A Spanish-Chilean team of scientists have found bacteria and archaea (primitive microorganisms) living two metres below the hypersaline substrates in the Atacama Desert in Chile, according to the journal Astrobiology.

"We have named it a 'microbial oasis' because we found microorganisms developing in a habitat that was rich in halite (rock salt) and other highly hygroscopic compounds (anhydrite and perchlorate) that absorb water" explained Victor Parro, researcher from the Center of Astrobiology (INTA-CSIC, Spain) and coordinator of the study.

Furthermore, the substrates where the microbes live favour deliquescence, which means they can attract the limited moisture in the air, condensing it on the surface of the salt crystals. Thin films of water that are a few microns thick are thereby formed.

In this environment, the underground microorganisms grow with everything they need to live: food and water. The species are not very different from others in similar hypersaline environments, but the peculiar thing is that they were discovered at a depth of between 2 and 3 metres, without any oxygen or sunlight.

To carry out this investigation, scientists used an instrument called SOLID (Signs of Life Detector), which was developed by the research team with the aim of using it for future missions on Mars.

The core of SOLID is a biochip –called LDChip– which includes up to 450 antibodies to identify biological material, such as sugar, DNA and protein. Samples can be taken, incubated and processed automatically and the results can be observed in an image with shiny points that show the presence of certain compounds and microorganisms.

Using this technique, the researchers in collaboration with Catholic University of the North in Chile have confirmed the presence of underground archaea and bacteria in the desert. They also took samples from a depth of up to 5 metres and took them to the laboratory, where not only were they able to photograph the microorganisms with the electron microscope, but also 'brought them into life' when supplied with water.

**Lessons for Mars**

"If there are similar microbes on Mars or remains in similar conditions to the ones we have found in Atacama, we could detect them with instruments like SOLID" Parro highlighted.

The researcher explained that saline deposits have been found on the red planet, therefore it is possible to think that there maybe hypersaline environments in its subsoil. "The high concentration of salt has a double effect: it absorbs water between the crystals and lowers the freezing point, so that they can have thin films of water (in brine) at temperatures several degrees below zero, up to minus 20 C." The high salt level and lack of water help preserve biological molecules, so that it was possible to find biological products in materials of this type, even though there were no live microorganisms since millions of years ago.

***References:*** *Parro et al. "A microbial oasis in the hypersaline Atacama subsurface discovered by a life detector chip: implications for the search for life on Mars". Astrobiology 11(10): 969-96, December 2011. Doi: 10.1089/ast.2011.0654.*

***http://www.eurekalert.org/pub_releases/2012-02/bu-gmt021412.php***

## Genes may travel from plant to plant to fuel evolution

***The evolution of plants and animals generally has been thought to occur through the passing of genes from parent to offspring and genetic modifications that happen along the way.***

PROVIDENCE, R.I. [Brown University] - But evolutionary biologists from Brown University and the University of Sheffield have documented another avenue, through the passing of genes from plant to plant between species with only a distant ancestral kinship.

How this happened is unclear. But the researchers show that not only did a grouping of grasses pass genes multiple times over millions of years, but that some of the genes that were transferred became integral cogs to the plants' photosynthetic machinery, a critical distinguishing feature in C4 plants, which dominate in hot, tropical climes and now make up 20 percent of the Earth's vegetational covering.

"As far as we know, this is the first case where nuclear genes that have been transmitted between plants have been incorporated into the primary metabolism and contributed to the evolution of a new trait, in this case C4 photosynthesis," said Pascal-Antoine Christin, a postdoctoral researcher in the Department of Ecology and Evolutionary Biology at Brown.

In a paper published in Current Biology, the researchers from Brown, Sheffield, and other universities in the United States, United Kingdom, and France investigated the ancestry of two genes encoding enzymes important in C4 photosynthesis - phosphoenolpyruvate carboxylase (ppc) and phosphoenolpyruvate carboxykinase (pck) - and these enzymes' historical presence and function in a common and well-studied grass, Alloteropsis. The biologists initially studied the genes in closely related species, three C4 plants (Alloteropsis angusta, Alloteropsis cimicina, and Alloteropsis semialata) and one C3 plant (Alloteropsis eckloniana). The goal was to learn the evolutionary history of the ppc and pck genes, which were present in their C3 common ancestor and were thought to have been adapted to aid in photosynthesis in the offspring C4 plants.

"People were wondering how these genes evolved. The global assumption was that an ancestor had the genes, but they weren't involved in photosynthesis, and so were later modified to become C4 photosynthetic agents," said Christin, the paper's corresponding author.

To test the hypothesis, the scientists took a wider view, surveying C4 plants in which the ppc enzyme was integral to photosynthesis and plants where the enzyme was present but had no photosynthetic role. They figured the ppc enzymes used in C4 photosynthesis would be closely related to the non-photosynthetic genes from closely related C3 plants, given their common ancestry.

Instead, the ppc genes involved in C4 photosynthesis were closely related to ppc genes of other C4 species with no close relation in the phylogeny, or family tree. Closer analysis also revealed these plants sharing photosynthetic ppc enzymes had diverged as many as 20 million years ago; the new finding is that despite these ancestral divergences, they exchanged genes.

In all, the researchers documented four instances in which the ppc enzyme or the pck enzyme found in the Alloteropsis C4 plants popped up in other C4 clades – Andropogoneae, Cenchrinae and Melinidinae. These clades include such diverse species as corn, foxtail millet, and guinea grass.

"We've long understood how evolutionary adaptations are passed from parents to offspring. Now we've discovered in plants that they can be passed between distant cousins without direct contact between the species," added Colin Osborne, an evolutionary biologist from the University of Sheffield and a corresponding author on the paper.

"What is so exciting here is that these genes are moving from plant to plant in a way we have not seen before," said Erika Edwards, assistant professor of biology at Brown and the second author on the paper. "There is no host-parasite relationship between these plants, which is usually what we see in this kind of gene movement."

Scientists call this evolutionary event "lateral gene transfer." The question, then, how are the plants passing their genes? The best guess at this point is that genetic material carried airborne in pollen grains land on a different species and a small subset of genes somehow get taken up by the host plant during fertilization. Such "illegitimate pollination events," as Edwards described it, have been seen in the laboratory. "There are reproductive mishaps that occur. In some cases, these could turn out to be highly advantageous," she said.

Christin, Osborne and Edwards think gene-swapping among plants continues today. "Is it good? Bad? I don't know," Christin said. "It's good for the plants. It means that plants can adapt to new environments by taking genes from others."

"It's like a short cut," Edwards added, "that could present itself as a mechanism for rapid evolution."

*Contributing authors include Guillaume Besnard, from the Universite Paul Sabatier in Toulouse; Susanna Boxall, Richard Gregory and James Hartwell, from the University of Liverpool; and Elizabeth Kellogg from the University of Missouri-St. Louis. The Swiss National Science Foundation, the Natural Environment Research Council's Molecular Genomics Facilities (U.K.) and a European Commission Marie Curie fellowship supported the research.*

***http://medicalxpress.com/news/2012-02-counterfeit-drugs-big-business-worldwide.html***

## Counterfeit drugs becoming big business worldwide

***The discovery that a fake version of the widely used cancer medicine Avastin is circulating in the United States is raising new fears that the multibillion-dollar drug-counterfeiting trade is increasingly making inroads in the U.S.***

(AP) - The criminal practice has largely been relegated to poor countries with lax regulations. But with more medicines and drug ingredients for sale in the U.S. being manufactured overseas, American authorities are afraid more counterfeits will find their way into this country, putting patients' lives at risk.

The Avastin discovery follows other recent instances in the U.S. of counterfeiting, involving such drugs as Viagra, the cholesterol medicine Lipitor and the weight-loss pill Alli.

"We do know there are counterfeits continuing to try and make their way onto the U.S. supply chain," said Connie Jung, an associate director in the Food and Drug Administration's office of drug security.

The FDA announced Tuesday it is investigating fake vials of Avastin that were sold to at least 19 doctors and clinics, including 16 sites in California, two in Texas and one in Chicago. Tests showed the vials did not contain the active ingredient in Avastin, which is given intravenously in hospitals, clinics and doctors' offices to treat several types of cancer. The contents of the vials are still being analyzed, and the FDA said it has not received any reports of patients who were harmed.

FDA officials said the counterfeit Avastin was imported from Britain and distributed by Volunteer Distribution, a wholesaler based in Gainesboro, Tenn. British regulators notified the FDA about the products in December, but the agency didn't confirm they were fake until last week.

The FDA gave assurances Wednesday that the U.S. remains one of the most secure pharmaceutical markets in the world. But the news sent cancer doctors scrambling to check their records.

Mary Mathias, a nurse who orders drugs for one doctor on the FDA list - Dr. Phillip L. Chatham in Granada Hills, Calif., - said they had stopped using the company in question at least a year ago.

Because Avastin treatments are spaced one to two weeks apart, it is not likely that someone would get more than one infusion from the same vial. And because these are people facing a life-threatening disease, it is hard to say whether missing one treatment with the real drug would compromise their care.

Gauging harm from a counterfeit cancer treatment is nearly impossible, said Dr. Robert C. Young, former president of the Fox Chase Cancer Center in Philadelphia and now a consultant to cancer centers.

A colon cancer patient, for example, might receive 18 to 20 Avastin infusions over six months. Missing one dose seems unlikely to have a dramatic effect on survival odds, but it's not provable either way because cancer's course and a patient's response to treatment are not predictable, he said.

Counterfeits have traditionally been more of a concern in developing regions like Asia and Latin America, where as many as 30 percent of drugs sold are fake, according to the World Health Organization. The group estimates just 1 percent of drugs dispensed in the U.S. and other developed nations are fake.

But incidents of counterfeiting reported by drugmakers have increased steadily over the decade to more than 1,700 worldwide last year, though only 6 percent of those were in the U.S. There are few reliable estimates on the value of the global counterfeit drug trade, though most place it in the tens of billions.

Counterfeiting has become more prevalent as pharmaceutical supply chains increasingly stretch across continents. Over 80 percent of the active ingredients used in U.S. pharmaceuticals are now manufactured overseas, according to a recent congressional report, and experts say this has made it easier to move counterfeit products into this country.

"With today's transportation networks, it's no longer a stretch to move these materials from a source in Pakistan or India to the U.S." said Tom Kubic, president of Pharmaceutical Security Institute, a trade association set up by two dozen pharmaceutical companies. In 2005, federal prosecutors indicted 11 employees of a Missouri business on charges of conspiring to sell $42 million in counterfeit Lipitor. It was manufactured in Costa Rica and illegally imported to the U.S., where it was sold to wholesalers.

Industry experts also say a combination of big profits and low penalties has made drug counterfeiting an increasingly attractive business for criminals in the U.S. and abroad. A single vial of Avastin sells for $2,400, and the drug had nearly $2.7 billion in U.S. sales last year, while the sentence for drug counterfeiting in the United States is about three years in prison. That compares with 15 years for counterfeiting money.

John Clark, head of global security for Pfizer Inc., said counterfeiters can make several million dollars quickly and, if they're caught, get off with as little as six months in jail. He also said counterfeiters can set up an operation at a fairly low cost - perhaps $50,000, including about $20,000 for a pill press.

"It's a no-brainer for criminal organizations that it's worth a gamble," Clark said.

Clark said Pfizer's anti-counterfeiting team around the world has seen a number of fake vaccines and biologic drugs sold in developing countries, not just pill-based drugs.

"They're getting much more sophisticated," often getting ahold of legitimate vials that had held such medicines, from patients, trash cans or recycling operations, and then filling them with oil or water.

***http://www.physorg.com/news/2012-02-malaria-method-boost-drug-production.html***

## New malaria method could boost drug production

***German scientists have developed a new way to make a key malaria drug that they say could easily quadruple production and drop the price significantly, increasing the availability of treatment for a disease that kills hundreds of thousands every year.***

Chemists at the Max Planck Institute take the waste product from the creation of the drug artemisinin - artemisinic acid - and convert it into the drug itself. The entire apparatus is compact, about the size of a carry-on suitcase, and inexpensive. That means it can be easily added to production sites anywhere around the world.

"Four hundred of these would be enough to make a world supply of artemisinin," said unit director Peter Seeberger, pointing to the machine on a table in his lab in Berlin's Dahlem neighborhood. "The beauty of these things is they're very small and very mobile."

A paper on the new technique was published this month in chemistry journal Angewandte Chemie.

Artemisinin is extracted from sweet wormwood, a plant that primarily grows in China and Vietnam and varies in its availability according to the season. In the extraction process, for every part artemisinin produced, there is 10 times the amount of artemisinic acid discarded as waste.

Past attempts to convert the acid using ultraviolet light to trigger the conversion have been unsuccessful because the process took several steps in a large tank of acid, making production inefficient and far too expensive. So the Max Planck chemists thought small - creating a machine that pumps all of the required ingredients through a thin tube wrapped around a UV lamp in a continuous process that takes 4 1/2 minutes from start-to-finish to produce the artemisinin.

The technique can convert about 40 percent of the waste acid into artemisinin - producing four times more of the drug from what had in the past been discarded, Seeberger said.

Colin Sutherland, a malaria expert at the London School of Hygiene and Tropical Medicine who was not involved in the Max Planck research, said the development could be significant in boosting production of the key malaria drug. He noted that currently very little artemisinin can be made from a large amount of the sweet wormwood, which is also difficult to grow. "If it's a simple process, given a certain amount of plant material, you can generate more drugs, that will make things cheaper and faster," he said. Since the end product is the same molecule, there should be no decrease in effectiveness of the synthetic product, Sutherland said.

Seeberger said a commercial prototype of the Max Planck machine could be ready in about six months and that it could go into production in about a year. He said current price estimates are around euro100,000 (US$132,000). When it's in production, the idea is to make it available for a minimal fee to cover costs, he said.

"The goal is to make sure that the drug is produced and made available to as many people as possible," said Seeberger, a former Massachusetts Institute of Technology professor who now teaches at Berlin's Free University.

Sabine Haubenreisser, a spokeswoman at the European Medicines Agency, said that if the new drug is close enough to the original, its producers could apply for it to be considered as a generic product or use older data proving artemesinin's effectiveness - which could speed the approval process.

Malaria cases and deaths have been dropping since 2004, due largely to campaigns to distribute bednets, spray homes with insecticide and make better drugs available. The World Health Organization estimates that at least 655,000 people die of malaria every year, mostly children under 5 in Africa.

At the moment, artemisinin-based therapies are considered the best treatment, but cost about $10 per dose - far too much for impoverished communities.

Former U.S. President Bill Clinton's Clinton Foundation currently has a program to purchase the treatments, then sell them at a deeply discounted 50 cents to communities where they're most needed. Cutting the price further while increasing production could "make a big difference," said Sutherland. "Many times more children will have access to the right drug early in their disease and that's likely to have an impact on mortality."

***http://medicalxpress.com/news/2012-02-androgen-boosts-hepatitis-virus-replication.html***

## Androgen boosts hepatitis B virus replication

***Androgen enhances replication of hepatitis B virus (HBV), rendering males more vulnerable than females to this virus, according to research published in the February Journal of Virology.***

"Our studies allowed us to understand the gender disparity of HBV carriers, and why this virus tends to cause more severe liver disease in men than in women," says principal investigator James Ou of the University of Southern California. The researchers found no difference between levels of virus in prepubescent male and female mice. However, post-puberty, levels in males exceeded those in females, in some cases by more than double, says Ou. Subsequently castrating male mice reduced the viral load, but injecting castrated mice with an androgen agonist resulted in a rising viral load again.

In a third set of experiments, the researchers removed the androgen receptor by genetic knockout, once again abolishing the androgen's effect on hepatitis B replication. Then they drilled down still further, discovering elements within the HBV genome which are recognized by the host's activated androgen receptor, which then boosts viral gene expression and replication. Epidemiologic studies have shown that men are three to seven times more likely than women to become HBV carriers, and male HBV carriers are more likely to develop cancer of the liver (hepatocellular carcinoma) than female carriers.

"Hepatitis B virus is one of the most important human pathogens," says Ou. "Approximately 350 million people worldwide are chronically infected, and roughly one million die annually." HBV can be transmitted sexually, as well as via non-sterile needles, and perinatally. In areas where the virus is endemic, it is frequently transmitted among young children. It is present in blood (including menstrual blood), vaginal secretions, saliva, semen, breast milk, and to a lesser extent in other bodily fluids. A vaccine is available for HBV.

***More information:*** *Y. Tian, C.f. Kuo, W.-l. Chen, and J.-h.J. Ou, 2012. Enhancement of hepatitis B virus replication by androgen and its receptor in mice. J. Virol. 86:1904-1910. Provided by American Society For Microbiology*

***http://www.physorg.com/news/2012-02-posits-theory-moral-behavior.html***

## Study posits a theory of moral behavior

***To understand the illicit behavior of some, we need to study the moral dimension of the self and what makes some individuals more dishonest than others.***

PhysOrg.com - Why do some people behave morally while others do not? Sociologists at the University of California, Riverside and California State University, Northridge have developed a theory of the moral self that may help explain the ethical lapses in the banking, investment and mortgage-lending industries that nearly ruined the U.S. economy.

For decades, sociologists have posited that individual behavior results from cultural expectations about how to act in specific situations. In a study, "A Theory of the Self for the Sociology of Morality," published in the February issue of the journal American Sociological Review, Jan E. Stets of UC Riverside and Michael J. Carter of CSU Northridge found that how individuals see themselves in moral terms is also an important motivator of behavior.

Bankers, stock brokers, and mortgage lenders who caused the recession were able to act as they did, without shame or guilt, perhaps because their moral identity standard was set at a low level, and the behavior that followed from their personal standard went unchallenged by their colleagues, Stets explained.

"To the extent that others in a situation verify or confirm the meanings set by a person's identity standard and as expressed in a person's behavior, the more the person will continue to engage in these behaviors," Stets said of the theory of moral identity she and Carter advance. "One's identity standard guides a person's behavior. Then the person sees the reactions of others to his or her behavior. If others have a low moral identity and others do not challenge the illicit behavior that follows from it, then the person will continue to do what he or she is doing. This is how immoral practices can emerge."

The sociologists surveyed a diverse group of more than 350 university students in a two-phase study that measured students' moral identity, assessment of specific situations as having a moral component, and moral emotions, such as guilt and shame. The students first were asked how they responded in specific situations where they had a choice to do the right or wrong thing; for example, copy another student's answers, drive home drunk, take an item, give to charity, allow another student to copy their answers, let a friend drive home drunk, return a lost item, or return money to a cashier.

Three months later, survey respondents were asked how to rate each scenario in moral terms, and how they thought individuals ought to feel following doing the right or wrong thing in each situation. The students placed themselves along a continuum between two contradictory characteristics - honest/dishonest, caring/uncaring, unkind/kind, unfair/fair, helpful/not helpful, stingy/generous, compassionate/hardhearted, untruthful/truthful, not hardworking/hardworking, friendly/unfriendly, selfish/selfless, and principled/unprincipled. The more that individuals endorsed themselves as honest, caring, kind, fair, helpful, generous, compassionate, truthful, hardworking, friendly, selfless, and principled, the higher their moral identity.

Wherever individuals are located on this continuum, they act with the goal of verifying the meanings of who they are that is set by their moral identity standard, Stets and Carter said. "We found that individuals with a high moral identity score were more likely to behave morally, while those with a low moral identity score were less likely to behave morally. Respondents who received feedback from others that did not verify their moral identity standard were more likely to report guilt and shame than those whose identities were verified," they said.

The goal is to live up to one's self-view however that appears across the moral continuum from being very uncaring and unjust to very caring and very just, the researchers said. "When the meanings of one's behavior based on feedback from others are inconsistent with the meanings in one's identity standard, the person will feel bad," they said.

More research is needed to identify the source of moral identity meanings, Stets and Carter said. "Exposure to particular social contexts and individuals may encourage a higher moral identity. For example, when parents are involved in their children's lives, their children are more likely to recognize moral values. Schools can also sensitize individuals to moral meanings by providing an atmosphere that fosters justice, virtue and volunteering. Religious traditions that promote reflection on moral issues and foster charitable work also help individuals recognize moral meanings."

Studying the moral self is opportune given the unregulated practices of bankers, stock brokers, and mortgage lenders whose behavior facilitated the recent recession in the United States, Stets and Carter said.

"The cost of their irresponsible practices has touched the lives of many innocent victims, as witnessed in the loss of individuals' retirement savings, homes, and jobs. The fact that a few greedy actors have the potential to damage the lives of many (as evidenced in the Bernie Madoff case) brings issues of right and wrong, good and bad, and just and unjust to public awareness," they said. "To understand the illicit behavior of some, we need to study the moral dimension of the self and what makes some individuals more dishonest than others."

*Provided by University of California, Riverside*

***http://www.sciencedaily.com/releases/2012/02/120216133442.htm***

## Preventing the Tasmanian Devil's Downfall: Genome of Contagious Cancer Sheds Light On Disease Origin and Spread

***Researchers have sequenced the genome of a contagious cancer that is threatening the Tasmanian devil, the world's largest carnivorous marsupial, with extinction.***

ScienceDaily - Cataloguing the mutations present in the cancer has led to clues about where the cancer came from and how it became contagious.

The research has revealed that the cancer, which is spread between animals by biting, first arose from the cells of a single female Tasmanian devil. This animal is nicknamed 'The Immortal Devil' because although she

died more than 15 years ago, her DNA is living on in the contagious cancer cell line that she spawned. The cancer causes the appearance of tumours on the face of affected devils which grow rapidly and cause death within months.

"The Tasmanian devil cancer is the only cancer that is threatening an entire species with extinction," says Dr Elizabeth Murchison, lead author from the Wellcome Trust Sanger Institute. "Sequencing the genome of this cancer has allowed us to catalogue the mutations that caused this cancer to arise and to persist in the Tasmanian devil population."

The team found evidence for genetic differences between tumours, indicating that the cancer has genetically diverged during its spread through the Tasmanian devil population. They searched for these genetic differences between the tumours of 69 different devils from distant locations in Tasmania, allowing them to build up a map of the cancer's spread through the devil population. This indicates that some cancer sub-types may be more virulent than others.

***Researchers have sequenced the complete genome of one Tasmanian devil.*** **(Credit: Save the Tasmanian Devil Program)**

"We found that devil cancer's genome has about 20,000 mutations. This is fewer mutations than are found in some human cancers and indicates that cancers do not need to be extremely unstable in order to become contagious." says Dr David Bentley, senior co-author from Illumina Cambridge Ltd. "Tracing the evolutionary history and spread of this cancer helps us to understand not only what caused this disease but also to predict how it might behave in the future."

The spread of cancer between individuals is normally prevented by the immune system, which can normally detect foreign tissues as 'non-self'. The team found some intriguing clues as to how the devil cancer may outwit the immune system, including mutations in a set of genes involved in immunity. However, future studies will be required to elucidate how cancer escapes the immune destruction.

"This research is important because it allows us to understand the pattern of disease spread and this may help contain the epidemic. However, we also now need to use the genome sequence to understand more about how this cancer became transmissible. Cancers that transmit through populations are obviously incredibly rare, but we should use the Tasmanian devil example to understand the process to be prepared in the extremely unlikely event that such an epidemic ever occurs in humans." says Professor Mike Stratton, senior author and Director of the Wellcome Trust Sanger Institute.

The next stage of the research will be to map the genomes of thousands of devil tumours in order to understand the genetic diversity present in the cancer and to investigate the genetic interactions between the cancer and the Tasmanian devil population.

***Journal Reference:*** *Elizabeth P. Murchison, Ole B. Schulz-Trieglaff, Zemin Ning, Ludmil B. Alexandrov, Markus J. Bauer, Beiyuan Fu, Matthew Hims, Zhihao Ding, Sergii Ivakhno, Caitlin Stewart, Bee Ling Ng, Wendy Wong, Bronwen Aken, Simon White, Amber Alsop, Jennifer Becq, Graham R. Bignell, R. Keira Cheetham, William Cheng, Thomas R. Connor, Anthony J. Cox, Zhi-Ping Feng, Yong Gu, Russell J. Grocock, Simon R. Harris, Irina Khrebtukova, Zoya Kingsbury, Mark Kowarsky, Alexandre Kreiss, Shujun Luo, John Marshall, David J. McBride, Lisa Murray, Anne-Maree Pearse, Keiran Raine, Isabelle Rasolonjatovo, Richard Shaw, Philip Tedder, Carolyn Tregidgo, Albert J. Vilella, David C. Wedge, Gregory M. Woods, Niall Gormley, Sean Humphray, Gary Schroth, Geoffrey Smith, Kevin Hall, Stephen M.J. Searle, Nigel P. Carter, Anthony T. Papenfuss, P. Andrew Futreal, Peter J. Campbell, Fengtang Yang, David R. Bentley, Dirk J. Evers, Michael R. Stratton. Genome Sequencing and Analysis of the Tasmanian Devil and Its Transmissible Cancer. Cell, 2012; 148 (4): 780 DOI: 10.1016/j.cell.2011.11.065*

***http://www.scientificamerican.com/article.cfm?id=how-first-plant-evolved***

## How the First Plant Came to Be

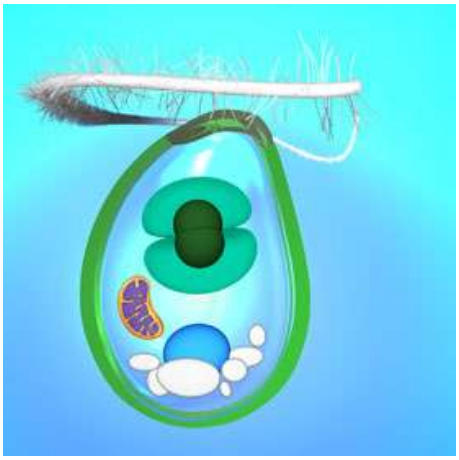
### *A genetic analysis reveals the ancient, complex--and symbiotic--roots of photosynthesis in plants*

**By David Biello | Thursday, February 16, 2012 | 24**

Earth is the planet of the plants - and it all can be traced back to one green cell. The world's lush profusion of photosynthesizers - from towering redwoods to ubiquitous diatoms - owe their existence to a tiny alga eons ago that swallowed a cyanobacteria and turned it into an internal solar power plant.

By studying the genetics of a glaucophyte - one of a group of just 13 unique microscopic freshwater blue-green algae, sometimes called "living fossils" - an international consortium of scientists led by molecular bioscientist Dana Price of Rutgers University, has elucidated the evolutionary history of plants. The glaucophyte Cyanophora paradoxa still retains a less domesticated version of this original cyanobacteria than most other plants.

According to the analysis of C. paradoxa's genome of roughly 70 million base pairs, this capture must have occurred only once because most modern plants share the genes that make the merger of photosynthesizer and larger host cell possible. That union required cooperation not just from the original host and the formerly free-ranging photosynthesizer but also, apparently, from a bacterial parasite.



Chlamydia-like cells, such as Legionella (which includes the species that causes Legionnaire's disease), provided the genes that enable the ferrying of food from domesticated cyanobacteria, now known as plastids, or chloroplasts, to the host cell.

"These three entities forged the nascent organelle, and the process was aided by multiple horizontal gene transfers as well from other bacteria," explains biologist Debashish Bhattacharya of Rutgers University, whose lab led the work published in Science on February 17. "Gene recruitment [was] likely ongoing" before the new way of life prospered and the hardened cell walls of most plants came into being.

***CYANOPHORA PARADOXA: Incorporating a cyanobacterium--the large two-toned green form inside--allowed this organism and its fellow plants to thrive.*** **Image: Courtesy of Debashish Bhattacharya**

In fact, such a confluence of events is so rare that evolutionary biologists have found only one other example: the photosynthetic amoeba Paulinella domesticated cyanobacteria roughly 60 million years ago. "The amoeba plastid is still a 'work in progress' in evolutionary terms," Bhattacharya notes. "We are now analyzing the genome sequence from Paulinella to gain some answers" as to how these events occur.

The work provides the strongest support yet for the hypothesis of late biologist Lynn Margulis, who first proposed in the 1960s to widespread criticism the theory that all modern plant cells derived from such a symbiotic union, notes biologist Frederick Spiegel of the University of Arkansas in Fayetteville, who was not involved in the work. That thinking suggests that all plants are actually chimeras - hybrid creatures cobbled together from the genetic bits of this ancestral union, including the enabling parasitic bacteria.

The remaining question is why this complex union took place roughly 1.6 billion years ago. One suggestion is that local conditions may have made it more beneficial for predators of cyanobacteria to stop eating and start absorbing, due to a scarcity of prey and an abundance of sunlight. "When the food runs out but sunlight is abundant, then photosynthesis works better" to support an organism, Bhattacharya notes. And from that forced union a supergroup of extremely successful organisms - the plants - sprang.

***http://medicalxpress.com/news/2012-02-inactive-genes-surprisingly-common-humans.html***

## Inactive genes surprisingly common in humans

### *Every person carries on average 100 variants that disable genes - yet very few suffer ill effects*

Medical Xpress - Every person carries on average 100 variants that disable genes - yet very few suffer ill effects, an international team of researchers led by Yale University and Wellcome Trust Sanger Institute report in the Feb. 17 issue of the journal Science.

Scientists were surprised to find so many of these variants in healthy individuals because loss of gene functions leads to diseases such as cystic fibrosis and muscular dystrophy. The findings will allow researchers to better pinpoint new disease-causing mutations by helping them differentiate between frequently occurring but harmless genetic variants and rare dangerous ones, the authors say.

The study is the latest coming from the 1000 Genomes Project, a massive international personal genomics effort aiming to provide a comprehensive resource of human genetic variation that will help speed the development of personalized therapies based on the genetic makeup of patients.

The team analyzed the genomes of 185 individuals from Europe, Asia, and Africa looking for so-called loss-of-function variants, mutations that disable a gene's ability to make protein.

"Even though previous studies have shown that loss-of-function variants exist in the general population, their extent has been underappreciated. This is the first time we have a definite sense of variation in the numbers of functional genes between individuals," said Suganthi Balasubramanian, the lead Yale author in the paper.

The study shows no individual has a full complement of functional genes. On average, each individual has 20 genes where both copies of the gene are disabled.

"In total, this study identified 253 such genes. This means at least one percent of human genes can be shut down without causing serious disease," explains Mark Gerstein, Albert L. Williams Professor of Biomedical Informatics, co-senior author from Yale University.

The catalog of loss-of-function variants in healthy genomes will be invaluable to clinicians as they begin to use personalized genomic analysis to help diagnose and treat disease, the authors say.

"Our research will be beneficial for current DNA sequencing studies underway in disease patients," says Dr Chris Tyler-Smith, co-senior author from the Wellcome Trust Sanger Institute. "The common loss-of-function variants were typically in genes that can be shut down without causing serious effects."

Scientists also found a large number of extremely rare variants and "we believe these will be the most interesting cases in terms of a potential role in human disease," says Dr Daniel MacArthur from the Wellcome Trust Sanger Institute, lead author on the study.

The study also showed that as many as a quarter of the loss-of-function variants involve large stretches of DNA (so-called structural variants), rather than mutations of single base pairs, which were believed to be the primary source of genetic variation. Structural variants are not yet well characterized in the human population and represent a major Yale contribution to 1000 Genomes Project. The Yale team is also looking at variants outside of regions of DNA that code for genes, an area that constitutes the vast majority of the genome.

***More information:*** *"A Systematic Survey of Loss-of-Function Variants in Human Protein-Coding Genes," by D.G. MacArthur, Science, 2012.*

***http://www.eurekalert.org/pub_releases/2012-02/uom-bbo021712.php***

## Building blocks of early Earth survived collision that created moon

***Unexpected findings show that portions of the Earth's mantle formed when the planet was smaller, and that some of this early mantle survived Earth's formation, including a collision with a body that may have created the Moon.***

COLLEGE PARK, Md. - Unexpected new findings by a University of Maryland team of geochemists show that some portions of the Earth's mantle (the rocky layer between Earth's metallic core and crust) formed when the planet was much smaller than it is now, and that some of this early-formed mantle survived Earth's turbulent formation, including a collision with another planet-sized body that many scientists believe led to the creation of the Moon.

"It is believed that Earth grew to its current size by collisions of bodies of increasing size, over what may have been as much as tens of millions of years, yet our results suggest that some portions of the Earth formed within 10 to 20 million years of the creation of the Solar System and that parts of the planet created during this early stage of construction remained distinct within the mantle until at least 2.8 billion years ago." says UMD Professor of Geology Richard Walker, who led the research team.

Prior to this finding, scientific consensus held that the internal heat of the early Earth, in part generated by a massive impact between the proto-Earth and a planetoid approximately half its size (i.e., the size of Mars), would have led to vigorous mixing and perhaps even complete melting of the Earth. This, in turn, would have homogenized the early mantle, making it unlikely that any vestiges of the earliest-period of Earth history could be preserved and identified in volcanic rocks that erupted onto the surface more than one and a half billion years after Earth formed.

However, the Maryland team examined volcanic rocks that flourished in the first half of Earth's history, called komatiites, and found that these have a different type of composition than what they, or anyone, would have, expected. Their findings were just published in the journal Science: "182W Evidence for Long-Term Preservation of Early Mantle Differentiation Products," by Mathieu Touboul, Igor S. Puchtel, and Richard J. Walker, University of Maryland. Their laboratory and work are supported by funding from the National Science Foundation and NASA.

### An Isotopic Signature

"We have discovered 2.8 billion year old volcanic rocks from Russia that have a combination of isotopes of the chemical element tungsten that is different from the combination seen in most rocks -- different even from the tungsten filaments in incandescent light bulbs," says the first author, Touboul, a research associate in the University of Maryland's Department of Geology. "We believe we have detected the isotopic signature of one of the earliest-formed portions of the Earth, a building block that may have been created when the Earth was half of its current mass."

As with many other chemical elements, tungsten consists of different isotopes. All isotopes of an element are characterized by having the same number of electrons and protons but different numbers of neutrons. Therefore, isotopes of an element are characterized by identical chemical properties, but different mass and nuclear properties. Through radioactive decay, some unstable (radioactive) isotopes spontaneously transform from one element into another at a specific, but constant, rate. As a result, scientists can use certain radioactive isotopes to determine the age of certain processes that happen within the Earth, as well as for dating rocks.

For the Maryland team the tungsten isotope 182-tungsten (one of the five isotopes of tungsten) is of special interest because it can be produced by the radioactive decay of an unstable isotope of the element hafnium, 182-hafnium.

According to the UMD team, the radioactive isotope 182-hafnium was present at the time our Solar System formed, but is no longer present on Earth today. Indeed, decay of 182-hafnium into 182-tungsten is so rapid (~9 million year half-life) that variations in the abundance of 182-tungsten relative to other isotopes of tungsten can only be due to processes that occurred very early in the history of our Solar System, they say.

The Maryland geochemists found that the 2.8 billion year old Russian komatiites from Kostomuksha have more of the tungsten isotope 182-W than normal. "This difference in isotopic composition requires that the early Earth formed and separated into its current metallic core, silicate mantle, and perhaps crust, well within the first 60 million years after the beginning of our 4.57-billion-year-old Solar System," says Touboul.

***This is a photomicrograph of a small, thin section of komatiite lava. The "spinifex texture" is a hallmark and considered unequivocal evidence of their ancient origin as molten rock extruded from deep in the Earth.*** **Igor Puchtel, UMD**

"In itself this is not new," he says, "but what is new and surprising is that a portion of the growing Earth developed the unusual chemical characteristics that could lead to the enrichment in 182-tungsten; that this portion survived the cataclysmic impact that created our moon; and that it remained distinct from the rest of the mantle until internal heat melted the mantle and transported some of this material to the surface 2.8 billion years ago, allowing us to sample it today."

**Higher Precision Yields New Findings, Insights**

The UMD team explained that they were able to conduct this research because they have developed new techniques that allow the isotopic composition of tungsten to be measured with unprecedented precision. "We do this by chemically separating and purifying the tungsten from the rocks we study. We then use an instrument termed a mass spectrometer to measure the isotopic composition of the tungsten"

According to the researchers their new findings have far reaching implications for understanding how Earth formed; how it differentiated into a metallic core, rocky mantle and crust; and the dynamics of change within the mantle.

"These findings indicate that the Earth's mantle has never been completely melted and homogenized, and that convective mixing of the mantle, even while Earth was growing, was evidently very sluggish," says Walker. "Many questions remain. The rocks we studied are 2.8 billion years old. We don't know whether the portion of the Earth with this unusual isotopic composition or signature can be found in much younger rocks. We plan to analyze some modern volcanic rocks in the near future to assess this."

***http://www.physorg.com/news/2012-02-science.html***

## Stealing for science

***Students at the University of Twente (The Netherlands) have stolen thirty laptops from various members of the university's staff.***

They were not prosecuted for this, so they could just get on with their studies. Indeed, these students even received ECTS credits for these thefts. UT researcher Trajce Dimkov asked the students to steal the machines as part of a scientific experiment. Stealing these laptops turned out to be a pretty simple matter.

Trajce Dimkov will be awarded his PhD at the University of Twente on 23 February. His doctoral research dealt with organizations' security policies. Under the pretext of conducting a user survey, Dimkov loaned laptops to thirty, randomly selected, university staff members. He then asked students to steal these laptops as part of a scientific experiment. The students made sixty attempts to steal these machines, thirty of which were successful. The study revealed that no matter how good an organization's security is, its effectiveness (or otherwise) is largely determined by human behaviour. Dimkov notes that "For instance, some people forgot to lock their door. In other cases, the students were able to think up a cover story that was sufficiently convincing to get a cleaner or caretaker to open the door for them. Other students were able to obtain the laptops by posing as technicians. Some claimed to have left their laptop in their supervisor's office, and that they needed it urgently, to complete an assignment. People tend to make an effort to be helpful, and a good cover story often does the trick."

The members of staff who had loaned the laptops were asked to make sure that these machines were always chained to their desks. They were also asked to lock the door when leaving their room, and to secure the laptop with a password. The university's security staff were informed in advance, to make sure that the students involved did not end up in jail.

To prevent such thefts in the future, Dimkov has developed a prototype model (a sort of navigation system) to identify ways in which laptops can be stolen. First, you have to enter data into this model, such as a map, information about members of staff, rules, locks, and security codes. The model uses special algorithms to link these items of data together, then generates scenarios that can be used to identify any "gaps" in the security system. Dimkov notes that "Without the input data, this system is of no use whatsoever to criminals. After all, what use is a navigation system without a map?"

***More information:*** *Trajce Dimkov conducted his doctoral research in the Department of Distributed and Embedded Security, at the CTIT research institute. He was supervised by Prof. Pieter Hartel and Dr Wolter Pieters. This research was partly funded by the STW Technology Foundation.*

***http://www.physorg.com/news/2012-02-properties-spiders-web.html***

## Researchers find the healing properties of a spider's web

***The study of spider webs has led to a discovery that will generate new kinds of medical sutures embedded with medication.***

PhysOrg.com - The University of Akron scientists have developed a novel synthetic material similar to a specific kind of silk spun by an orb spider. The specific web design is known as BOAS because it looks like beads-on-a-string in a circular web. The beads are glue droplets. The replication of this design can potentially be used as strong and flexible sutures that contain medication embedded in these bead-like structures.

The researchers developed the new biocompatible thread after meeting with physicians who specialize in wound healing and who expressed the need for better material-related solutions to medical problems.

**Novel material to aid healing**

The scientists published their findings in the American Chemical Society journal Langmuir in an article titled Spider Silk Inspired Functional Microthreads and will present at two upcoming scientific conferences. The next step is to apply for funding to speed the commercialization process to create medical materials that can help heal injured tendons or tissue.

"We have been very interested in architecture of adhesives produced by spiders and were very intrigued to understand why spiders use the BOAS structure," says Vasav Sahni, UA polymer science graduate student and lead author of the article published in Langmuir.

Sahni and research team members Dr. Ali Dhinojwala, chair of the Department of Polymer Science and Maurice Morton Professor of Polymer Science, and Disha Labhasetwar, an NSF-REU student with the Department of Polymer Science created the BOAS replica by copying techniques used by spiders when they spin silk.

"We used fluid mechanics concepts to vary the size and spacing of the glue beads and mimicked the spider silk threads by using commercially available materials, such as nylon," Sahni says, adding that he and his colleagues designed and fabricated a specialized piece of equipment to develop the threads.

**Glue releases medication**

Rather than place individual glue drops on a string, the researchers' novel technique coats threads uniformly with glue. The glue forms waves, which morph into beads that create greater-than-average contact areas and also release energy, or adhesive strength, when peeled. The beads can potentially also create a structure in which medication can be placed and released.

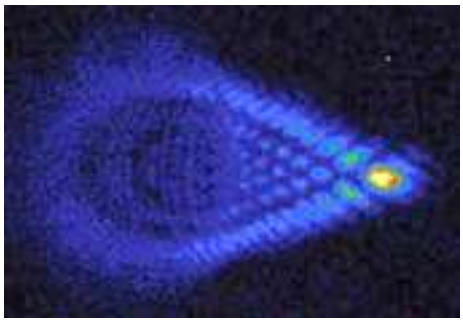
Dhinojwala, Sahni and their research colleagues developed the biocompatible thread to address one of the needs identified in a pilot program with the Austin BioInnovation Institute in Akron (ABIA). The scientists shadowed medical experts at the Akron General Hospital wound clinic, the Akron Children's Hospital burn unit and Summa Health System, assessing needs and developing solutions.

The researchers plan to apply for a $25,000 ABIA grant to fund development of a prototype and hope to see it commercialized locally. The researchers say their simple and scalable technique allows for rapid and large-scale fabrication of the new adhesives. *Provided by University of Akron*

http://www.physorg.com/news/2012-02-extreme-imaging-science.html

## Extreme imaging wins science praise

***A Griffith University PhD candidate has been highly awarded for his innovative image of the shadow of a single atom.***



Ben Norton, from the Kielpinski group in the Centre for Quantum Dynamics, was runner up in the CiSRA Extreme Imaging Competition following extensive work on high resolution imaging.

Run by Canon Australia and CiSRA, Canon Inc.'s Australian research centre, the Extreme Imaging competition aims to promote and celebrate local research at the intersection of imaging and technology.

"Atoms are the building blocks of matter. A human hair is a billion atoms wide," said Professor David Kielpinski, from Griffith's Centre for Quantum Dynamics. "So just manipulating and isolating a single atom is extremely difficult, let alone imaging it.

***Ben Norton's remarkable image of an atom was runner up in the CiSRA Extreme Imaging Competition in Australia.***
**Credit: Courtesy of Griffith University**

"Ben has had to use some very special tricks to do both. First of all he cools them down, to within a degree of 'absolute zero', the coldest temperature possible (about−273.15 °C), to keep them still. Then he traps them inside an ultra-high vacuum, holding them steady using electric fields. These techniques are very hard, but they have been done before. What is new is how Ben images them.

"To do this he uses a special flat lens made using concentric rings, which was originally developed for lighthouses. These lenses can be made so small and light that they can be put inside the vacuum chamber with the atoms, allowing Ben to collect as much light as possible. This last trick has allowed Ben to take some of the highest resolution images of atoms ever made, including the first ever image of the shadow of a single atom, by measuring how much light is absorbed when the atom is there."

Professor Kielpinski said imaging single atoms is important for understanding not just physics, but also in the new field of quantum computing. "The techniques developed in this project may have other applications too, such as ultra-high resolution imaging of biological cells.

"I really appreciate that this work has been considered worthy of the Canon prize – it's been an amazing opportunity. I am also very grateful for the strong support I have received from my supervisors at Griffith throughout my research there." *Provided by Griffith University*

http://www.newscientist.com/article/dn21495-usb-stick-can-sequence-dna-in-seconds.html

## USB stick can sequence DNA in seconds

***It may look like an ordinary USB memory stick, but a little gadget that can sequence DNA while plugged into your laptop could have far-reaching effects on medicine and genetic research.***

**18:29 17 February 2012 by Duncan Graham-Rowe**


The UK firm Oxford Nanopore built the device, called MinION, and claims it can sequence simple genomes – like those of some viruses and bacteria – in a matter of seconds. More complex genomes would take longer, but MinION could also be useful for obtaining quick results in sequencing DNA from cells in a biopsy to look for cancer, for example, or to determine the genetic identity of bone fragments at an archaeological dig.

**Image: Oxford Nanopore Technologies**

The company demonstrated today at the Advances in Genome Biology and Technology (AGBT) conference in Marco Island, Florida, that MinION has sequenced a simple virus called Phi X, which contains 5000 genetic base pairs.

**Proof of principle**

This is merely a proof of principle – "Phi X was the first DNA genome to be sequenced ever," says Nick Loman, a bioinformatician at the Pallen research group at the University of Birmingham, UK, and author of the blog Pathogens: Genes and Genomes. But it shows for the first time that this technology works, he says. "If you can sequence this genome you should be able to sequence larger genomes."

Oxford Nanopore is also building a larger device, GridION, for lab use. Both GridION and MinION operate using the same technology: DNA is added to a solution containing enzymes that bind to the end of each strand.

When a current is applied across the solution these enzymes and DNA are drawn to hundreds of wells in a membrane at the bottom of the solution, each just 10 micrometres in diameter.

Within each well is a modified version of the protein alpha hemolysin (AHL), which has a hollow tube just 10 nanometres wide at its core. As the DNA is drawn to the pore the enzyme attaches itself to the AHL and begins to unzip the DNA, threading one strand of the double helix through the pore. The unique electrical characteristics of each base disrupt the current flowing through each pore, enough to determine which of the four bases is passing through it. Each disruption is read by the device, like a tickertape reader.

**Long strands, and simple**

This approach has two key advantages over other sequencing techniques: first, the DNA does not need to be amplified - a time-consuming process that replicates the DNA in a sample to make it abundant enough to make a reliable measurement.

Second, the devices can sequence DNA strands as long as 10,000 bases continuously, whereas most other techniques require the DNA to be sheared into smaller fragments of at most a few hundred bases. This means that once they have been read they have to be painstakingly reassembled by software like pieces of a jigsaw. "We just read the entire thing in one go," as with Phi X, says Clive Brown, Oxford Nanopore's chief technology officer.

But Oxford Nanopore will face stiff competition. Jonathan Rothberg, a scientist and entrepreneur who founded rival firm 454 Life Sciences, also announced at the AGBT conference that his start-up company, Ion Torrent, will be launching a desktop sequencing machine. Dubbed the Ion Proton, it identifies bases by using transistors to detect hydrogen ions as they are given off during the polymerisation of DNA.

This device will be capable of sequencing a human genome in 2 hours for around $1000, Rothberg claims. Nanopores are an "elegant" technology, he says, but Ion Torrent already has a foot in the door. "As we saw last summer with the E. coli outbreak in Germany, people are already now using it," he says.

**Pocketful of DNA**

By contrast, the MinION would take about 6 hours to complete a human genome, Brown claims, though the company plans to market the device for use in shorter sequencing tasks like identifying pathogens, or screening for genetic mutations that can increase risk of certain diseases. Each unit is expected to cost $900 when it goes on sale later this year.

"The biggest strength of nanopore sequencing is that it generates very long reads, which has been a limitation for most other technologies," says Loman. If the costs, quality, ease of use and throughput can be brought in line with other instruments, it will be a "killer technology" for sequencing, he says.

As for clinical applications, David Rasko at the Institute for Genome Sciences at the University of Maryland in Baltimore, says the MinION could have huge benefits. "It may have serious implications for public health and it could really change the way we do medicine," he says. "You can see every physician walking around the hospital with a pocketful of these things." And it will likely increase the number of scientists generating sequencing data by making the technology cheaper and more accessible, he says.

***http://www.sciencedaily.com/releases/2012/02/120217145324.htm***

## New Braille-Like Texting App Lets You Text Without Looking

***Imagine if smartphone and tablet users could text a note under the table during a meeting without anyone being the wiser.***

ScienceDaily - Imagine if smartphone and tablet users could text a note under the table during a meeting without anyone being the wiser. Mobile gadget users might also be enabled to text while walking, watching TV or socializing without taking their eyes off what they're doing.

Georgia Tech researchers have built a prototype app for touch-screen mobile devices that is vying to be a complete solution for texting without the need to look at a mobile gadget's screen.

"Research has shown that chorded, or gesture-based, texting is a viable solution for eyes-free written communication in the future, making obsolete the need for users to look at their devices while inputting text on them," said Mario Romero, Postdoctoral Fellow in the School of Interactive Computing (IC) and the project's principal investigator.

The free open-source app, called BrailleTouch, incorporates the Braille writing system used by the visually impaired. It has been conceived as a texting tool for any of the millions of smartphone phone users worldwide.

Early studies with visually impaired participants proficient in Braille typing have demonstrated that users can input at least six times the number of words per minute when compared to other research prototypes for eyes-free texting on a touch screen. Users reach up to 32 words per minute with 92 percent accuracy with the prototype app for the iPhone.

"We are currently designing a study to formally evaluate BrailleTouch through both quantitative and qualitative methods," said Caleb Southern, an IC graduate student. "We will measure the typing speed and accuracy of visually impaired users and capture the feedback from study participants in areas such as comfort, ease of use and perceived value."

For sighted users, the research team is exploring how BrailleTouch could be a universal eyes-free mobile texting app that replaces soft QWERTY keyboards and other texting technologies.

"BrailleTouch is an out-of-the-box solution that will work with smartphones and tablets and allow users to start learning the Braille alphabet in a few minutes," said Romero. "It also reduces the need for expensive proprietary Braille keyboard devices, which typically cost thousands of dollars."

The researchers have designed BrailleTouch to address the limitations of soft keyboards, which do not provide tactile feedback, as well as physical keyboards, which often use small and numerous fixed buttons. BrailleTouch is the only iPhone app in existence that uses a six-finger chording process that replicates the traditional Braille keyboard.

The app uses a gesture-based solution by turning the iPhone's touchscreen into a soft-touch keyboard programmed for Braille and requiring only six keys, making it a practical solution for the limited screen real estate on smartphones.

The key feature of the BrailleTouch technology is the use of the six-key configuration so that the keyboard fits on the screen and users keep their fingers in a relatively fixed position while texting. This design allows users to hold their device with the screen facing away from them ¬– cradling the device with their palms or pinkies and thumbs – and to type with a majority of their fingers, identical to typing Braille on a standard keyboard.

The team behind BrailleTouch is led by Romero and IC Professor Gregory Abowd, co-principal investigator. Former IC affiliate Brian Frey conceived the original idea and developed the first prototype and Southern created an improved design. They are conducting usability studies together with James Clawson, a Ph.D. candidate in IC, and Kate Rosier, a master's graduate in Digital Media and bachelor's graduate in Computational Media.

The research group has developed iPhone and iPad versions of BrailleTouch and is currently working on Android versions. The app recently won the MobileHCI 2011 competition for design at the MobileHCI conference in Stockholm, Sweden.

*BrailleTouch will be demonstrated at the Abilities Expo-Atlanta 2012, taking place Feb. 17-19 at the Georgia World Congress Center. A video of BrailleTouch in action is available at the following link: http://www.youtube.com/watch?v=rIEO1bUFHsI*
*This project was supported in part by the Rehabilitation Engineering Research Center for Wireless Technologies (Wireless RERC), which is funded by the National Institute on Disability and Rehabilitation Research (NIDRR), United States Department of Education, under grant number H133E110002.*

***http://medicalxpress.com/news/2012-02-klebsiella-pneumoniae-superbug.html***

## Klebsiella pneumoniae 'superbug' is being studied

***University at Buffalo researchers are expressing concern about a new, under-recognized, much more potent variant of a common bacterium that has surfaced in the U.S.***

"Historically, in Western countries, classical strains of Klebsiella pneumoniae have caused infections mostly in sick, hospitalized patients whose host defense systems are compromised," says Thomas Russo, MD, professor in the Department of Medicine at the UB School of Medicine and Biomedical Sciences and head of its Infectious Disease Division. "But in the last 10 to 15 years, a new variant of it has begun causing community-acquired infection in young, healthy individuals," he says. "This variant causes serious, life-threatening, invasive infections and is able to spread to other organs from the initial site of infection."

Perhaps most important, says Russo, these hypervirulent strains of Klebsiella pneumoniae have the potential to become highly resistant to antibiotics, similar to Escherichia coli and classical Klebsiella pneumoniae.

"These hypervirulent strains are the next 'superbugs' –in-waiting," he says. "If they become resistant to antibiotics, they will become difficult, if not impossible to treat."

With recent funding from the National Institutes of Health under a program to fund high-risk, high-reward research, Russo and his UB colleagues are studying the microbiology of the new variant of Klebsiella pneumoniae in an effort to identify the genes that make it hypervirulent so they can figure out how to stop it in its tracks.

"Infections due to highly resistant bacteria are becoming increasingly problematic," says Russo. "We are continually threatened by a 'post-antibiotic' era. The combination of a bacterium that is both highly virulent and resistant to antimicrobials is double-trouble."

The researchers' concern stems from the fact that classical Klebsiella pneumoniae is one of the bacterial species that can easily acquire mobile genetic units, called plasmids, that contain multiple genes that confer high levels of antimicrobial resistance.

"That's in part why we're concerned," says Russo. "We know that this bacterium has the potential to acquire these plasmids and it almost certainly will." He notes that most bacteria that have proven to be resistant to most or all of the drugs currently available do not usually infect healthy members of the community.

"What is alarming about the hypervirulent Klebsiella pneumoniae is that they do possess the potential to infect healthy people," says Russo. "If this hypervirulent bacterium also becomes highly resistant to antimicrobials, we will have a significant problem to manage. We hope that our research and that of others can prevent this possibility."

While the new hypervirulent variant was first seen exclusively in in the Pacific Rim, it has now been found in several cities in North America, including Buffalo, and in Europe, Canada, Israel and South Africa as well. The UB researchers characterize it as "under-recognized" both by physicians and microbiology laboratories.

The disease most commonly presents as a liver abscess, which is not typical for otherwise healthy patients.

"This new variant presents with unique and scary features: first is its tendency to infect young, healthy people in the community and the second is its unique propensity for metastatic spread to other parts of the body," says Russo. "It spreads to sites beyond the initial source of the infection, such as the lungs, the central nervous system and the eye, potentially causing loss of vision. If infection spreads to the brain, there can be brain damage as well. Between 10 and 30 percent of cases are fatal."

In Buffalo, this hypervirulent variant of Klebsiella pneumoniae was identified in an otherwise healthy, young person several years ago. The patient, who was in his 20s, was hospitalized for several months before making a full recovery. Similar cases are causing concern throughout the international infectious disease community.

At the moment, most cases of hypervirulent Klebsiella pneumoniae resolve if treated aggressively with antibiotics and drainage of abscesses; however, some infections, despite optimal treatment, result in a persistent morbidity or death, Russo says.

He notes that the potential for the bug to acquire drug resistance is adding a sense of urgency to the research.

Russo says that microbiology labs should be aware that an important characteristic of these hypervirulent strains (also known as hypermucoviscous strains) is that when bacterial colonies grown on a solid surface in the laboratory are stretched by a common microbiology tool, called an inoculation loop, they form a viscous "string" greater than 5 millimeters in length.

Russo's team at UB is now beginning to develop a clearer picture of this formidable bacterial opponent.

In November, he and his colleagues published a PLoS ONE paper that showed that hypervirulent Klebsiella pneumoniae acquires iron more efficiently than the usual strains of K. pneumoniae. "With the NIH grant, we hope to further elucidate the precise details of the bacterial factors that are responsible for hypervirulent Klebsiella pneumoniae acquiring iron so much more efficiently," he says. "The goal of this line of research is that these iron-acquisition factors possessed by hypervirulent Klebsiella pneumoniae will then lend themselves as therapeutic or vaccine targets so that we can better treat or prevent infection." *Provided by University at Buffalo*

***http://www.eurekalert.org/pub_releases/2012-02/uol-urs021712.php***

## UofL research shows substituting with smokeless tobacco saves lives

### *Presentation at AAAS shows scientific foundation for tobacco harm reduction efforts*

VANCOUVER, British Columbia – Substituting smokeless tobacco products can save smokers' lives, and there is a scientific foundation that proves it. That is the message Brad Rodu, D.D.S., professor of medicine at the University of Louisville (UofL) School of Medicine and the Endowed Chair in Tobacco Harm Reduction at UofL's James Graham Brown Cancer Center, delivered at the Annual Meeting of the American Association for the Advancement of Science Feb. 18. Rodu spoke at the session, "Harm Reduction: Policy Change to Reduce the Global Toll of Smoking-Related Disease."

"Quit or die: That's been the brutal message delivered to 45 million American smokers, and it has helped contribute to 443,000 deaths per year, according to statistics from the Centers for Disease Control and Prevention," Rodu said. "The truth, however, is that total nicotine and tobacco abstinence is unattainable and unnecessary for many smokers."

Rodu's presentation, "Transforming Tobacco Use: The Potential of Tobacco Harm Reduction," was based on his almost 20 years of research. His work shows that smokers can greatly reduce their risk of disease and death by replacing smoking products with e-cigarettes or modern, spit-free smokeless tobacco. These products provide a much safer alternative for those smokers who are unable or unwilling to quit smoking because they continue to deliver nicotine without the harmful effect of smoking.

"Nicotine is addictive, but it is not the cause of any smoking-related disease. Like caffeine, nicotine can be used safely by consumers," Rodu said.

Decades of epidemiologic research bear out Rodu's findings. While no tobacco product is completely safe, smokeless products have been shown to be 98 percent safer than cigarettes. In the United Kingdom, the Royal College of Physicians reported in 2002 that smokeless tobacco is up to 1,000 times less hazardous than smoking, and in 2007, further urged world governments to seriously consider instituting tobacco harm reduction strategies as a means to save lives.

To see the proof of what tobacco harm reduction can do, look to Sweden, Rodu said. "Over the past 50 years, Swedish men have had Europe's highest per capita consumption of smokeless tobacco as well as Europe's lowest cigarette use. During the same time, they also have the lowest rate of lung cancer than men in any other European country."

In the United States., steps have been made to document the value of tobacco harm reduction. In 2006, a National Cancer Institute-funded study estimated that if tobacco harm reduction was "responsibly communicated" to smokers, 4 million would switch to smokeless tobacco. The American Council on Science and Health – which organized Rodu's session at the AAAS Annual Meeting – concluded in the same year that tobacco harm reduction "shows great potential as a public health strategy to help millions of smokers."

Rodu is well aware of the controversy his research findings generate. Opponents of any use of nicotine delivery products maintain that smokeless tobacco puts the user at great risk for oral cancer, a position not supported by research.

"The risk of mouth cancer among smokeless tobacco users is extremely low – certainly lower than the risk of smoking-related diseases among smokers," he said. "The annual mortality rate among long-term dry snuff users is 12 deaths per 100,000 and the rate among users of more popular snus, moist snuff and chewing tobacco is much lower. For perspective, the death rate among automobile users is 11 per 100,000 according to a 2009 report from the National Highway Traffic Safety Administration. Compare those to the rate among smokers: more than 600 deaths per 100,000 every year"

"The data clearly show that smokeless tobacco users have, at most, about the same risk of dying from mouth cancer as automobile users have of dying in a car wreck."

***About Brad Rodu*** *Rodu earned his dental degree from The Ohio State University. After an oral pathology residency program at Emory University, he completed fellowships at the University of Alabama at Birmingham sponsored by the American Cancer Society and the National Cancer Institute. He was on the UAB faculty from 1981 to 2005 with appointments in several departments in the schools of Medicine, Public Health and Dentistry. He joined the UofL faculty in 2005. His research is supported by unrestricted grants from tobacco manufacturers to the University of Louisville and by the Kentucky Research Challenge Trust Fund.*

***http://news.discovery.com/autos/driverless-cars-nevada-120218.html***

## Look Out! Driverless Cars Coming To Nevada

***Motorists of The Silver State, don't be alarmed if you soon glance over at the car next to you and see a passenger waving from a driverless car as you zoom across Interstate 15.***

**By Nic Halverson | Sat Feb 18, 2012 09:39 AM ET**

Nevada lays claim (mining pun intended) to some fairly brazen "firsts," morally suspect as they may be: The first U.S. state to legalize casino-style gambling; the first state to sanction the use of the gas chamber for executions; and, to top it off, the first (and only) state to legalize prostitution in the modern era.

Depending how you feel about sharing the road with driverless vehicles, Nevada's newest "first" sounds just as reckless as the aforementioned.

The Nevada Department of Motor Vehicles released a statement Wednesday saying that its Legislative Commission gave thumbs-up for regulations allowing for operation of self-driving cars on the state's highways and byways.

"Nevada is the first state to embrace what is surely the future of automobiles," Department of Motor Vehicles director Bruce Breslow said in a statement. "These regulations establish requirements companies must meet to test their vehicles on Nevada's public roadways as well as requirements for residents to legally operate them in the future."

Autonomous vehicles will display red license plates and, pending approval for public use, the cars will then have a green license plate.

Nevada said it collaborated with Google, car manufacturers, testing professionals, insurance companies, universities and law enforcement agencies to develop regulations.

Last June, the state passed a bill requiring the DMV to draft rules for autonomous vehicles.

Should an autonomous car be involved in an accident, there's always the human passenger to blame.

http://medicalxpress.com/news/2012-02-disease-confounds-experts-children.html

## Nodding disease confounds experts, kills children

***Patrick Anywar, 14, lies curled up naked in the dust and midday heat of a Ugandan village, struggling to look up at his younger brother and sister playing in front of the family home.***

After a minute's effort to face his siblings, Anywar's head slumps onto his chest and his emaciated body is gripped by convulsions. Anywar is one of more than 3,000 children in northern Uganda who are suffering from a debilitating mystery ailment known as nodding disease, which has touched almost every family in the village of Tumangu.

For several years, scientists have tried and failed to determine the cause of the illness, which locals say has killed hundreds of youngsters. What they do know is that the disease affects only children and gradually devastates its victims through debilitating seizures, stunted growth, wasted limbs, mental disabilities and sometimes starvation.

Anywar's mother, Rugina Abwoyo, has already lost one son, named Watmon, to the disease in 2010. Now she says she can do little but watch on helplessly as another child slips away. "Before he was walking and running like other children, but now someone always has to stay home to look after him," Abwoyo told AFP. "The disease is terrible -- it does not let him drink or eat by himself."

Walking along footpaths cut through the sorghum plantations, Joe Otto, a volunteer health worker, explains how nodding disease has ravaged Tumangu, about 450 kilometres (280 miles) north of the capital Kampala.

"There are 780 people living in this village and we have 97 cases of the disease. It has affected almost every family," Otto, 54, told AFP.

Whenever sporadic deliveries of medicine arrive at the local health centre several kilometres away, Otto pedals his bicycle to fetch the drugs. But he knows that they only offer a short-term solution. "We are giving out drugs for epilepsy, like carbamazepine, but this disease is different from epilepsy," Otto said.

Instead, as the disease has torn through their community, local residents have moved from fear to a grim acceptance, Otto says. "We started saying that the patient who had died was the one who had been cured, because finally they were at rest from this painful disease," Otto said. 'We hope that our youngest can be saved'

Scientists are trying to find a cure: since 2010, researchers ranging from epidemiologists to environmental experts, neurologists, toxicologists and psychiatrists have carried out a range of tests.

Investigations have looked at possible links between the disease and everything from a parasite that causes river blindness, to malnutrition and the after-effects of a civil war that ravaged northern Uganda for decades.

"We looked at all this, but unfortunately we were not able to pinpoint any significant contributing or risk factors," said Miriam Nanyunja, disease control and prevention officer at the World Health Organisation in Kampala. "The search for the causative agent is still ongoing," she added.

Often the results have thrown up more questions than answers. Scientists do not know if the disease is linked to similar outbreaks in neighbouring South Sudan and Tanzania. Efforts continue to understand if the disease is still spreading or has peaked -- and why it is seems confined only to certain communities. Last month, after pressure from lawmakers from affected areas, Uganda's health ministry produced an emergency response plan to try to identify and control the disease.

However, Nanyunja says that while the search for the cause and a possible cure goes on, for now, doctors can only focus on trying to alleviate the symptoms. "There are many diseases that we continue to treat symptomatically, without knowing the exact cause," Nanyunja said.

But for Patrick Anywar, any attempts to curb or cure the disease may come too late. "We are hoping that the doctors work very hard to get the cure for this disease," his mother Abwoyo says. "There is no future for us as so many children have already been affected, but we hope that our youngest can be saved."

http://www.sciencedaily.com/releases/2012/02/120219191244.htm

## Single-Atom Transistor Is End of Moore's Law; May Be Beginning of Quantum Computing

***The smallest transistor ever built - in fact, the smallest transistor that can be built -- has been created using a single phosphorus atom by an international team of researchers at the University of New South Wales, Purdue University and the University of Melbourne.***

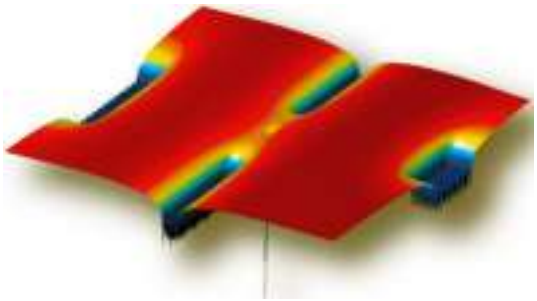
ScienceDaily - The single-atom device was described Sunday (Feb. 19) in a paper in the journal Nature Nanotechnology. Michelle Simmons, group leader and director of the ARC Centre for Quantum Computation and Communication at the University of New South Wales, says the development is less about improving current technology than building future tech.

"This is a beautiful demonstration of controlling matter at the atomic scale to make a real device," Simmons says. "Fifty years ago when the first transistor was developed, no one could have predicted the role that

computers would play in our society today. As we transition to atomic-scale devices, we are now entering a new paradigm where quantum mechanics promises a similar technological disruption. It is the promise of this future technology that makes this present development so exciting."

The same research team announced in January that it had developed a wire of phosphorus and silicon -- just one atom tall and four atoms wide -- that behaved like copper wire.

Simulations of the atomic transistor to model its behavior were conducted at Purdue using nanoHUB technology, an online community resource site for researchers in computational nanotechnology.



***A controllable transistor engineered from a single phosphorus atom has been developed by researchers at the University of New South Wales, Purdue University and the University of Melbourne. The atom, shown here in the center of an image from a computer model, sits in a channel in a silicon crystal. The atomic-sized transistor and wires might allow researchers to control gated qubits of information in future quantum computers.*** **Purdue University image**

Gerhard Klimeck, who directed the Purdue group that ran the simulations, says this is an important development because it shows how small electronic components can be engineered.

"To me, this is the physical limit of Moore's Law," Klimeck says. "We can't make it smaller than this."

Although definitions can vary, simply stated Moore's Law holds that the number of transistors that can be placed on a processor will double approximately every 18 months. The latest Intel chip, the "Sandy Bridge," uses a manufacturing process to place 2.3 billion transistors 32 nanometers apart. A single phosphorus atom, by comparison, is just 0.1 nanometers across, which would significantly reduce the size of processors made using this technique, although it may be many years before single-atom processors actually are manufactured.

The single-atom transistor does have one serious limitation: It must be kept very cold, at least as cold as liquid nitrogen, or minus 391 degrees Fahrenheit (minus 196 Celsius).

"The atom sits in a well or channel, and for it to operate as a transistor the electrons must stay in that channel," Klimeck says. "At higher temperatures, the electrons move more and go outside of the channel. For this atom to act like a metal you have to contain the electrons to the channel.

"If someone develops a technique to contain the electrons, this technique could be used to build a computer that would work at room temperature. But this is a fundamental question for this technology."

Although single atoms serving as transistors have been observed before, this is the first time a single-atom transistor has been controllably engineered with atomic precision. The structure even has markers that allow researchers to attach contacts and apply a voltage, says Martin Fuechsle, a researcher at the University of New South Wales and lead author on the journal paper.

"The thing that is unique about what we have done is that we have, with atomic precision, positioned this individual atom within our device," Fuechsle says.

Simmons says this control is the key step in making a single-atom device. "By achieving the placement of a single atom, we have, at the same time, developed a technique that will allow us to be able to place several of these single-atom devices towards the goal of a developing a scalable system."

The single-atom transistor could lead the way to building a quantum computer that works by controlling the electrons and thereby the quantum information, or qubits. Some scientists, however, have doubts that such a device can ever be built.

"Whilst this result is a major milestone in scalable silicon quantum computing, it does not answer the question of whether quantum computing is possible or not," Simmons says. "The answer to this lies in whether quantum coherence can be controlled over large numbers of qubits. The technique we have developed is potentially scalable, using the same materials as the silicon industry, but more time is needed to realize this goal."

Klimeck says despite the hurdles, the single-atom transistor is an important development. "This opens eyes because it is a device that behaves like metal in silicon. This will lead to many more discoveries."

The research project spanned the globe and was the result of many years of effort.

"When I established this program 10 years ago, many people thought it was impossible with too many technical hurdles. However, on reading into the literature I could not see any practical reason why it would not be possible," Simmons says. "Brute determination and systemic studies were necessary -- as well as having many outstanding students and postdoctoral researchers who have worked on the project."

Klimeck notes that modern collaboration and community-building tools such as nanoHUB played an important role.

"This was a trans-Pacific collaboration that came about through the community created in nanoHUB. Now Purdue graduate students spend time studying at the University of New South Wales, and their students travel to Purdue to learn more about nanotechnology. It has been a rewarding collaboration, both for the scientific discoveries and for the personal relationships that were formed."

***Journal Reference:***

*Martin Fuechsle, Jill A. Miwa, Suddhasatta Mahapatra, Hoon Ryu, Sunhee Lee, Oliver Warschkow, Lloyd C. L. Hollenberg, Gerhard Klimeck, Michelle Y. Simmons. A single-atom transistor. Nature Nanotechnology, 2012; DOI: 10.1038/nnano.2012.21*

***http://www.eurekalert.org/pub_releases/2012-02/aaon-snt020712.php***

## Study: New treatment for traumatic brain injury shows promise in animals

### *A new drug is showing promise in shielding against the harmful effects of traumatic brain injury in rats*

NEW ORLEANS – A new drug is showing promise in shielding against the harmful effects of traumatic brain injury (TBI) in rats, according to a study that was released today and will be presented at the American Academy of Neurology's 64th Annual Meeting in New Orleans April 21 to April 28, 2012.

"There are currently no primary treatments for TBI, so this research provides hope that effective treatments can be developed," said study author Michael Kaufman, a second year medical student at Wayne State University School of Medicine in Detroit and a member of the American Academy of Neurology. The principal investigator on the study is Christian Kreipke, MD, also with Wayne State University School of Medicine.

Traumatic brain injury causes a decrease in blood flow in the cerebrum of the brain, which if prolonged, can cause permanent cell dysfunction and death. A receptor in the brain called endothelin receptor A (ETrA) contributes to the restriction of blood flow as early as four hours after a brain injury. The new drug, called clazosentan, is thought to specifically block these receptors.

Researchers gave brain-injured rats the drug clazosentan through an intravenous (IV) line at several different points in time after the injury. Next, they measured the rat's blood flow in the hippocampus and sensory motor cortex with an MRI brain scan and tested their behavior in learning a maze.

Preliminary data from the study found that clazosentan decreased the effects of the traumatic brain injury on blood flow to the hippocampus by 25 percent at four hours and 23 percent at 48 hours after TBI. However, giving the rats the drug at 12 hours post-injury caused some to improve, while others worsened or remained the same. In the trial, the drug was most effective when given at two hours post-injury and again at 24 hours after the trauma. The rats also performed better on the maze test when given the drug at two and 24 hours post-injury.

"This research is the foundation for future clinical trials that will investigate the possibilities of using clazosentan in the treatment of TBI," said Kaufman.

*Learn more about traumatic brain injury at http://www.aan.com/patients.*

*The study was supported by the American Academy of Neurology, the National Institutes of Health, and the U.S. Department of Veterans Affairs.*

***http://www.eurekalert.org/pub_releases/2012-02/bc-dcm021712.php***

## Deadly carbon monoxide prevents miscarriage

### *Low dose carbon monoxide therapy is able to restore placental function and prevent fetal death in mice, without any detrimental effects.*

Heme oxygenase-1 is essential for the growth of blood vessels in the placenta and in establishing blood flow in the umbilical cord. Too little HO-1 can lead to a restriction in the growth of the fetus and even in fetal death and miscarriage. New research published in BioMed Central's open access journal Medical Gas Research has shown that low dose carbon monoxide therapy is able to restore placental function and prevent fetal death in mice, without any detrimental effects.

Intrauterine growth restriction due to problems in placental function and blood flow can result in a 'small for gestational age' baby, miscarriage or perinatal death. Both miscarriage and pre-eclampsia are associated with low levels of HO-1 in the placenta, however research suggests that carbon monoxide can mimic the effects of HO-1. Researchers from the Otto-von-Guericke University, Germany tested carbon monoxide therapy on intrauterine growth restriction in mice. They found that an extended course of low dose (50ppm) carbon monoxide was able to reduce fetal loss from 30% to zero – all the babies survived.

Prof Ana Claudia Zenclussen, who led the research explained, "At the levels used to prevent fetal death we found that inhaled low dose carbon monoxide was anti-inflammatory. It reduced the amount of cell death (apoptosis), and increased levels of the anti-apoptotic molecule BAG-1, in the placenta and additionally increased the level of vascular endothelial growth factor (VEGF), which is associated with angiogenesis and blood vessel repair."

Intrauterine growth restriction is a serious complication of pregnancy. Surviving babies have a lifelong increased risk of hypertension, cardiovascular disease and renal disease. In the face of these fears carbon monoxide therapy may provide a lifeline to mothers at risk. However there is a cautionary note - higher doses of carbon monoxide were able to improve placental function but were damaging to the fetus, shorter treatment at low dose was not enough to prevent fetal death. Prof Zenclussen warned, "It is very important, given the inherent dangers in using carbon monoxide, that the dose and length of treatment are tightly controlled."

***Notes to Editors*** *Exploring the potential of low doses carbon monoxide as therapy in pregnancy complications. Tarek El-Mousleh, Pablo A Casalis, Ivonne Wollenberg, Maria L Zenclussen, Hans D Volk, Stefanie Langwisch, Federico Jensen and Ana C Zenclussen Medical Gas Research (in press)*

***http://www.bbc.co.uk/news/science-environment-16972761***

## Lab-grown meat is first step to artificial hamburger

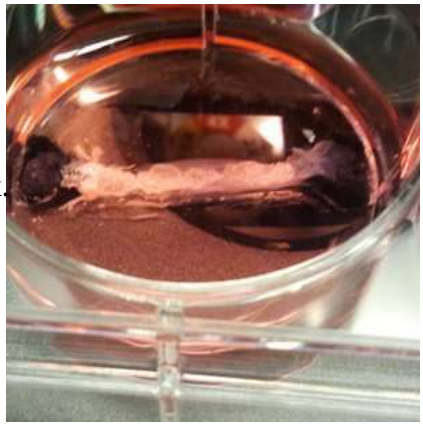
***Dutch scientists have used stem cells to create strips of muscle tissue with the aim of producing the first lab-grown hamburger later this year.***

**By Pallab Ghosh Science correspondent, BBC News, Vancouver**

The aim of the research is to develop a more efficient way of producing meat than rearing animals. At a major science meeting in Canada, Prof Mark Post said synthetic meat could reduce the environmental footprint of meat by up to 60%. "We would gain a tremendous amount in terms of resources," he said.

Professor Post's group at Maastricht University in the Netherlands has grown small pieces of muscle about 2cm long, 1cm wide and about a mm thick. They are off-white and resemble strips of calamari in appearance. These strips will be mixed with blood and artificially grown fat to produce a hamburger by the autumn.

The cost of producing the hamburger will be £200,000 but Professor Post says that once the principle has been demonstrated, production techniques will be improved and costs will come down.



***The first strips of muscle have been grown in a project to develop a new way to produce meat***

At a news conference, Prof Post said he was even planning to ask celebrity chef Heston Blumenthal to cook it. "The reason we are doing this is not to show a viable product but to show that in reality we can do this," he told BBC News. "From then on, we need to spend a whole lot of work and money to make the process efficient and then cost effective."

So why use such high tech methods to produce meat when livestock production methods have done the job effectively for thousands of years? It is because most food scientists believe that current methods of food production are unsustainable. Some estimate that food production will have to double within the next 50 years to meet the requirements of a growing population. During this period, climate change, water shortages and greater urbanisation will make it more difficult to produce food.

Prof Sean Smukler from the University of British Columbia said keeping pace with demand for meat from Asia and Africa will be particularly hard as demand from these regions will shoot up as living standards rise. He thinks that lab grown meat could be a good solution. "It will help reduce land pressures," he told BBC News. "Anything that stops more wild land being converted to agricultural land is a good thing. We're already reaching a critical point in availability of arable land," he said.

Lab-grown meat could eventually become more efficient than producing meat the old fashioned way, according to Prof Post. Currently, 100g of vegetable protein has to be fed to pigs or cows to produce 15g of animal protein, an efficiency of 15%. He believes that synthetic meat could be produced with an equivalent energy efficiency of 50%. So what is the synthetic burger likely to taste like?

"In the beginning it will taste bland," says Prof Post. "I think we will need to work on the flavour separately by trying to figure out which components of the meat actually produce the taste and analyse what the composition of the strip is and whether we can change that." Prof Post also said that if the technology took off, it would reduce the number of animals that were factory farmed and slaughtered.

But David Steele, who is president of Earthsave Canada, said that the same benefits could be achieved if people ate less meat. "While I do think that there are definite environmental and animal welfare advantages of this high-tech approach over factory farming, especially, it is pretty clear to me that plant-based alternatives... have substantial environmental and probably animal welfare advantages over synthetic meat," he said.

Dr Steele, who is also a molecular biologist, said he was also concerned that unhealthily high levels of antibiotics and antifungal chemicals would be needed to stop the synthetic meat from rotting.

http://medicalxpress.com/news/2012-02-dieting-wrong-scientists.html

# Everything you know about dieting is wrong: scientists

***Everything you know about dieting is wrong, say US scientists who have devised a new formula for calculating calories and weight loss that they hope will revolutionize the way people tackle obesity.***

Obesity rates have doubled worldwide in the past 30 years, coinciding with a growing food surplus, and the ensuing epidemic has sparked a multibillion dollar weight loss industry that has largely failed to curb the problem.

Current standards in the United States, where two thirds of people are overweight or obese, advise people that cutting calories by a certain amount will result in a slow and steady weight loss over time.

But that advice fails to account for how the body changes as it slims down, burning less energy and acquiring a slower metabolism, researchers told the American Association for the Advancement of Science meeting in Vancouver. The result is a plateau effect that ends up discouraging dieters and sending them back into harmful patterns of overeating.

As an example, researcher Kevin Hall offered up his large vanilla latte, purchased at a popular coffee shop. When he asked, the barista told him it contained about 240 calories.

"The notion was if I drank one of these every day and then I replaced it with just black coffee no sugar, then over the course of a year I should lose about 25 pounds, and that should just keep going," Hall told reporters.

"People have used this sort of rule of thumb to predict how much people should lose for decades now, and it turns out to be completely wrong."

Hall, a scientist with the US National Institutes of Health, said his work aims to "come up with better rules and better predictions of what is going to happen when an individual changes their diet."

He and colleagues said their scientific model is aimed to help doctors and policymakers, while a "back-of-the-envelope calculation" for consumers means cutting small amounts of daily calories, but expecting to cut more over time. "If I want to lose 10 pounds of weight eventually, I have to cut 100 calories per day out of my diet," Hall explained. "You'll get halfway there in about a year, and then you will eventually plateau, (reaching the goal) after about three years," he added. "For folks abroad that works out to about 100 kilojoules per day per kilogram. The contrast is the old rule of thumb predicts twice as much weight loss after a year, and it gets worse after that."

The new model gives dieters one calorie goal for short term weight loss and another for permanent weight loss. Exercise is also calculated in to help set realistic goals.

Tests on small numbers of adults who were fed strictly controlled diets showed the model was accurate, though real-life situations are harder to predict.

Study co-author Carson Chow, also with NIH, said the daily calorie cut needed for weight loss was actually smaller than researchers anticipated. "It is essentially one cookie different a day, so a 150 calorie cookie leads to a seven kilogram (15 pound) difference in weight. That is huge in my opinion," Chow said.

Their model was first published in The Lancet in August 2011, and a link is available at ***http://bwsimulator.niddk.nih.gov*.**

"People can plug in some information about their initial age, their height, their weight, some estimate of their physical activity level," Hall said.

Add in a goal weight and the "model will simulate what changes of diet or exercise that person would have to do to achieve that goal weight, and then even more importantly what they need to do permanently maintain that weight loss."

Since The Lancet article appeared, the notion has not exactly taken the world by storm, in part because it's not primed for public use, but is mainly aimed at doctors and researchers with adult American patients for now.

Also, if a dieter enters an extreme weight goal, the number of calories the model returns may be much too low to be realistic or healthy, so it needs an expert's interpretation.

"It's not particularly user friendly... but it is still relatively informative," said Hall, who maintains hope that some day his message will be heard.

"There is a lot of inertia behind these old rules of thumb," he said, adding that he was heartened by an editorial in December in the journal of the American Dietetic Association that commented on the idea of a weight loss plateau and mentioned the new simulator.

"It's going to take some time to get the public and the professional community aware that there is a new way of doing things, and we actually have some tools that weren't available before."

http://news.discovery.com/animals/blacktip-reef-sharks-122002.html

## Even Sharks Make Friends

***They have a reputation for being ruthless, solitary predators, but the opposite may be closer to the truth.***

**By Jennifer Viegas | Mon Feb 20, 2012 07:00 AM ET**

Sharks have a reputation for being ruthless, solitary predators, but evidence is mounting that certain species enjoy complex social lives that include longstanding relationships and teamwork. A new study, published in the latest Animal Behaviour, documents how one population of blacktip reef sharks is actually organized into four communities and two subcommunities. The research shows for the first time that adults of a reef-associated shark species form stable, long-term social bonds. The image contrasts with usual reports on this species, which mistakenly sinks its sharp teeth into surfers and swimmers from time to time.

Lead author Johann Mourier told Discovery News that "other species, such as grey reef sharks and scalloped hammerheads form polarized groups where individuals have a specific place, and such species may also have complex social organization."

Mourier, a scientist at the Center for Island Research and Environmental Study (CNRS-EPHE), and colleagues Julie Vercelloni and Serge Planes conducted the study at Moorea Island in the Society archipelago, French Polynesia. A total of seven sites were surveyed on a regular basis along just over 6 miles of the north shore of Moorea. The surveys included nearly hour-long dives at a depth close to 50 feet, with the diver photographing nearby sharks.

Analysis of the gathered data determined that the sharks were not within non-random collections, but rather had organized themselves into meaningful social groups. "The four main communities are mixed-sex communities that use a specific home range, however, within these communities individuals tend to associate more often with others of the same sex and length," Mourier said.

In a prior study, he determined that length is proportional to a shark's age, with male blacktip reef sharks being mature at about the age of 7 and measuring around 3.6 feet long. Females are slightly larger than males.

Mourier suspects the sharks join together in communities for protection and to avoid aggression with each other. He and his colleagues also observed a remarkable feat, "when a group of about four or five blacktip reef sharks herded a school of fishes around a coral structure." This suggests they can cooperate with each other to hunt as a team. Yet another perk to organizing could be that each shark becomes a comforting landmark for others in the group. As Mourier said, "Using a home range and knowing all individuals may help individuals to have a better knowledge of their environment."

The researchers point out that sharks' relative brain mass-body ratios have been found to be comparable to those of mammals, indicating that they are capable of complex social behaviors on par with those demonstrated in birds and mammals. It could just be that the highly mobile nature of sharks, combined with the difficulty of following individuals in the open sea, has kept their social interactions hidden away from human eyes until recent years.

In another study, led by Demian Chapman, researchers showed that lemon sharks at the Bimini islands, Bahamas, tended to stay near their coastal birthplace for many years. "We were very surprised to see that many lemon sharks lingered for years around the island where they were born -- often more than half of their development to adulthood," said Chapman, a shark scientist with the Institute for Ocean Conservation Science at Stony Brook University.

In both studies, age therefore seems to help shape a shark's social life. Family ties may also be important to sharks, a possibility that Mourier and his colleagues are investigating now. The scientists clipped the fins of 70 percent of the sharks involved in this latest study and are analyzing the bits for DNA. He said, "This will soon reveal if they tend to group with relatives, as is the case in other social animals, such as for some mammals."

http://medicalxpress.com/news/2012-02-eu-skin-cancer-drug-roche.html

## EU approves skin cancer fighting drug: Roche

***Swiss drug giant Roche said on Monday it had been given European Union approval for its treatment to fight a highly aggressive form of skin cancer.***

The European Commission gave the green light to Zelboraf, a drug used to treat adults with BRAF V600 mutation-positive unresectable or metastatic melanoma, the company announced in a statement.

If diagnosed early melanoma is generally curable but when it spreads to other parts of the body it is the deadliest form of skin cancer. Currently only one in four people lives for more than a year following diagnosis.

US health authorities approved Zelboraf in August last year, with Roche describing initial sales as "very encouraging." The group recorded Zelboraf sales worth 31 million Swiss francs (25.7 million euros) in 2011.

The treatment, already approved by Switzerland, Brazil and Canada among others, has "revolutionised melanoma treatment," said analysts at the Zurich Cantonal Bank (ZKB).
Roche said the drug allows people to live significantly longer, with trials showing the risk of death was reduced by 63 percent for people who received Zelboraf compared to those who received standard first-line treatment.

***http://www.physorg.com/news/2012-02-archaeologists-jordan-earliest.html***

## Archaeologists discover Jordan's earliest buildings

***Some of the earliest evidence of prehistoric architecture has been discovered in the Jordanian desert, providing archaeologists with a new perspective on how humans lived 20,000 years ago.***

PhysOrg.com - Archaeologists working in eastern Jordan have announced the discovery of 20,000-year-old hut structures, the earliest yet found in the Kingdom. The finding suggests that the area was once intensively occupied and that the origins of architecture in the region date back twenty millennia, before the emergence of agriculture.

The research, published 15 February, 2012 in PLoS One by a joint British, Danish, American and Jordanian team, describes huts that hunter-gatherers used as long-term residences and suggests that many behaviours that have been associated with later cultures and communities, such as a growing attachment to a location and a far-reaching social network, existed up to 10,000 years earlier.

***Two Early Epipalaeolithic structures.*** **Credit: Lisa Maher**

Excavations at the site of Kharaneh IV are providing archaeologists with a new perspective on how humans lived 20,000 years ago. Although the area is starkly dry and barren today, during the last Ice Age the deserts of Jordan were in bloom, with rivers, streams, and seasonal lakes and ponds providing a rich environment for hunter-gatherers to settle in.

“What we witness at the site of Kharaneh IV in the Jordanian desert is an enormous concentration of people in one place,” explained Dr. Jay Stock from the Department of Archaeology and Anthropology at the University of Cambridge and co-author of the article.

“People lived here for considerable periods of time when these huts were built. They exchanged objects with other groups in the region and even buried their dead at the site. These activities precede the settlements associated with the emergence of agriculture, which replaced hunting and gathering later on. At Kharaneh IV we have been able to document similar behaviour a full 10,000 years before agriculture appears on the scene.”

The archaeologists, who were funded by a grant from the Arts and Humanities Research Council UK, spent three seasons excavating at the large open-air site covering two hectares. They recovered hundreds of thousands of stone tools, animal bones and other finds from Kharaneh IV, which today appears as little more than a mound 3 m high rising above the desert landscape.

Based on the size and density of the site, the researchers had long suspected that Kharaneh IV was frequented by large numbers of people for long periods of time; these latest findings now confirm their theory. “It may not look very impressive to the untrained eye, but it is one of the densest and largest Palaeolithic open-air sites in the region,” said Dr. Lisa Maher, from the University of California, Berkeley, who spearheads the excavations.

“The stone tools and animal bone vastly exceed the amounts recovered from most other sites of this time period in southwest Asia.” In addition, the team also recovered rarer items, such as shell beads, bones with regularly incised lines and a fragment of limestone with geometric carved patterns.

So far, the team has fully excavated two huts; but there may be several more hidden beneath the desert’s sands. “They’re not large by any means. They measure about 2–3 m in maximum length and were dug into the ground. The walls and roof were made of brush wood, which then burnt and collapsed leaving dark coloured marks,” described Dr. Tobias Richter from the University of Copenhagen and one of the project’s co-directors.

Radiocarbon dating suggests that the hut is between 19,300 and 18,600 years old. Although a team of archaeologists working at Ohalo II on the shore of the Sea of Galilee in 1989 found the region’s oldest hut structures, which date from 23,000 years ago, the team working at the Kharaneh IV site believe their discovery is no less significant, as Dr. Maher explained:

“Inside the huts, we found intentionally burnt piles of gazelle horn cores, clumps of red ochre pigment and a cache of hundreds of pierced marine shells. These shell beads were brought to the site from the Mediterranean and Red Sea over 250 km away, showing that people were very well linked to regional social networks and exchanged items across considerable distances.” *Provided by University of Cambridge*