http://www.eurekalert.org/pub_releases/2013-03/aaon-ctc032013.php

# Could that cold sore increase your risk of memory problems?

***Cold sore viruses may be associated with cognitive problems***

MINNEAPOLIS – The virus that causes cold sores, along with other viral or bacterial infections, may be associated with cognitive problems, according to a new study published in the March 26, 2013, print issue of Neurology®, the medical journal of the American Academy of Neurology.

The study found that people who have had higher levels of infection in their blood (measured by antibody levels), meaning they had been exposed over the years to various pathogens such as the herpes simplex type 1 virus that causes cold sores, were more likely to have cognitive problems than people with lower levels of infection in the blood.

"We found the link was greater among women, those with lower levels of education and Medicaid or no health insurance, and most prominently, in people who do not exercise," said author Mira Katan, MD, with the Northern Manhattan Study at Columbia University Medical Center in New York and a member of the American Academy of Neurology. The study was performed in collaboration with the Miller School of Medicine at the University of Miami in Miami, FL.

For the study, researchers tested thinking and memory in 1,625 people with an average age of 69 from northern Manhattan in New York. Participants gave blood samples that were tested for five common low grade infections: three viruses (herpes simplex type 1 (oral) and type 2 (genital), and cytomegalovirus), chlamydia pneumoniae (a common respiratory infection) and Helicobacter pylori (a bacteria found in the stomach).

The results showed that the people who had higher levels of infection had a 25 percent increase in the risk of a low score on a common test of cognition called the Mini-Mental State Examination.

The memory and thinking skills were tested every year for an average of eight years. But infection was not associated with changes in memory and thinking abilities over time.

"While this association needs to be further studied, the results could lead to ways to identify people at risk of cognitive impairment and eventually lower that risk," said Katan. "For example, exercise and childhood vaccinations against viruses could decrease the risk for memory problems later in life."

*The study was supported by the National Institutes of Neurological Disorders and Stroke (NINDS), the Swiss National Science Foundation and the Leducq Foundation.*

http://www.eurekalert.org/pub_releases/2013-03/zsol-wab032213.php

# What a bunch of dodos!

***A catastrophic mass extinction of birds in the Pacific Islands followed the arrival of the first people***

Research carried out by the Zoological Society of London (ZSL) and collaborators reveals that the last region on earth to be colonised by humans was home to more than 1,000 species of birds that went extinct soon after people reached their island homes.

The paper was published today (25th) in the journal Proceedings of the National Academy of Sciences USA.

Almost 4,000 years ago, tropical Pacific Islands were an untouched paradise, but the arrival of the first people in places like Hawaii and Fiji caused irreversible damage to these natural havens, due to overhunting and deforestation. As a result, birds disappeared. But understanding the scale and extent of these extinctions has been hampered by uncertainties in the fossil record.

Professor Tim Blackburn, Director of ZSL's Institute of Zoology says: "We studied fossils from 41 tropical Pacific islands and using new techniques we were able to gauge how many extra species of bird disappeared without leaving any trace."

They found that 160 species of non-passerine land birds (non-perching birds which generally have feet designed for specific functions, for example webbed for swimming) went extinct without a trace after the first humans arrived on these islands alone.

"If we take into account all the other islands in the tropical Pacific, as well as seabirds and songbirds, the total extinction toll is likely to have been around 1,300 bird species," Professor Blackburn added.

Species lost include several species of moa-nalos, large flightless waterfowl from Hawai'i, and the New Caledonian Sylviornis, a relative of the game birds (pheasants, grouse, etc) but which weighed in at around 30kg, three times as heavy as a swan.

Certain islands and bird species were particularly vulnerable to hunting and habitat destruction. Small, dry islands lost more species because they were more easily deforested and had fewer places for birds to hide from hunters. Flightless birds were over 30 times more likely to become extinct that those that could fly.

Bird extinctions in the tropical Pacific did not stop with these losses. Forty more species disappeared after Europeans arrived, and many more species are still threatened with extinction today.

http://www.eurekalert.org/pub_releases/2013-03/uoc--kss032513.php

## Kidney sparing surgery underutilized for patients who need it most

***Patients with chronic kidney insufficiency had an almost two-fold higher probability of undergoing total nephrectomy***

Researchers at the University of California, San Diego School of Medicine have released study results that show national treatment trends in the surgical management of patients with kidney disease. The study found that partial and complete kidney removal (total nephrectomy) and energy-based techniques to destroy tumors are all on the rise. Surprisingly, the patients most in need of kidney-sparing surgery are still more likely to undergo total nephrectomy. The findings recently published online in BJU International.

"While the overall proportion of patients receiving kidney preserving treatments for localized kidney tumors continues to grow, the most significant, and perhaps unsettling finding was that patients with kidney insufficiency still undergo complete kidney removal – even though kidney preserving treatment may be indicated," said senior author Ithaar Derweesh, MD, urologic oncologist at UC San Diego Moores Cancer Center.

The kidney is a vital organ which performs a variety of functions in addition to making urine. It controls blood pressure, bone health, and also makes a hormone to tell the bone marrow to produce red blood cells. Kidney insufficiency is characterized by a progressive decline in kidney function which may affect all of these actions.

"The study, which examined procedures over a 10-year period, found that patients with chronic kidney insufficiency had an almost two-fold higher probability of undergoing total nephrectomy than kidney preserving treatments, such as partial nephrectomy or tumor ablation," said Derweesh, a pioneer in minimally invasive kidney surgery.

Derweesh added that further investigation is needed to confirm these findings, and to examine what factors are responsible for patient and physician selection of treatment for kidney cancer. He noted that in the case of small renal masses less than four centimeters in size, partial nephrectomy has equivalent outcomes to total nephrectomy, and that ablation techniques, such as cryoablation or radiofrequency ablation, and observation are valid options for select patients.

While kidney insufficiency may result in total failure of the kidney, most patients do not progress to dialysis dependence. However, patients with worsening degrees of kidney insufficiency are at higher risk of cardiac events, such as heart attack and stroke, and osteoporosis and anemia.

The UC San Diego study utilized data from the Nationwide Inpatient Sample (NIS), the largest database of all annual hospital admissions in the United States. Approximately 443,850 procedures were included in the study.

Chronic kidney disease (CKD) is a condition characterized by a gradual loss of kidney function over time and affects more than 26 million American adults. The two main causes of chronic kidney disease are diabetes and high blood pressure, which are responsible for up to two-thirds of the cases. Renal cell carcinoma is a commonly diagnosed urological malignancy with an estimated 57,760 new cases and 12,908 deaths in the United States during 2009.

*Study contributors include: Jeffrey Woldrich, MD, Kerrin Palazzi, MPH, Sean Stroup, MD, Roger Sur, MD, J. Kellog Parsons, MD, and David Chang, PhD.*

http://www.sciencedaily.com/releases/2013/03/130325094032.htm?

## Nerve Regeneration Research and Therapy May Get Boost from New Discovery

***New mechanism for guiding the growth of nerves that involves cell-death machinery may bring advances in neurological medicine***

A new mechanism for guiding the growth of nerves that involves cell-death machinery has been found by scientists at the University of Nevada, Reno that may bring advances in neurological medicine and research. The team obtained the evidence in studies of fruit flies and reported their discovery in an article published in the publication Cell Reports. "Although the fly is a relatively simple organism, almost every gene identified in this species appears to be carrying out similar functions in humans," said Thomas Kidd, associate professor in the University's biology department in whose lab the work was performed.

The Kidd lab is part of a $10 million Center for Biomedical Research Excellence Project in Cell Biology of Signaling at the University, which is funded by the National Institute of Health's Institute of General Medical Sciences. The project is also funded by the National Science Foundation.

"Flies are useful because the neural mechanisms we are studying are similar to those in mammals," said Gunnar Newquist, lead author of the Cell Reports article and a post-doctoral neuroscience researcher in Kidd's lab. "We've found something no one has seen before, that blocking the cell-death pathway can make nerves deprived of guidance cues figure out the right way to connect with other neurons. This was completely unexpected and novel, but really exciting because it changes the way we look at nerve growth.

"Neurons have a natural ability to die, if they fail to make the right connections they usually die. Neurons, like most other cell types, have the capacity to commit suicide and many do so during the formation of the nervous system."
The wiring of nervous systems is composed of axons, specialized extensions of neurons that transmit electrical impulses. During development axons navigate long distances to their targets by using signals in their environment. Netrin-B is one of those signals. Kidd, Newquist and colleagues have shown that Netrin-B also keeps neurons alive. "Take away the Netrin-B and growth and cell death goes haywire," Newquist said.
This led them to the discovery that the cell-death machinery is active in growing nerves, and appears to be an integral part of the navigation mechanism.
"We use fruit fly genetics to study how these axons navigate these long distances correctly when developing," Kidd said. "Understanding the mechanisms they use to navigate is of great interest, not only for understanding how our brains form, but also as a starting point to devise ways to stimulate the re-growth of axons after injury, especially spinal cord injuries. "Our work suggests that therapeutics designed to keep neurons alive after injury may be able to stimulate neurons to start re-growing or sprouting new connections."
"I am very pleased to see Tom's and Gunnar's hard work come to fruition," said Chris von Bartheld, director of the University's cell-biology COBRE and a professor in the University of Nevada School of Medicine. "Linking axonal path finding and cell death signaling opens exciting new venues to better understand both topics. It also shows that our recently established center in cell biology is achieving its goals of producing top-level biomedical research."
The research featured intheCell Report article is a major focus of Kidd's lab, which is one of five research projects in the COBRE. The center is a strong stimulus to integrate the University's research in cell biology, developmental biology, signaling and neuroscience and to develop collaborations between basic scientists and clinicians.
*Gunnar Newquist, J. Michelle Drennan, Matthew Lamanuzzi, Kirsti Walker, James C. Clemens, Thomas Kidd. Blocking Apoptotic Signaling Rescues Axon Guidance in Netrin Mutants. Cell Reports, 2013; DOI: 10.1016/j.celrep.2013.02.017*

***http://phys.org/news/2013-03-sporting-groups-struggle.html***

## Sporting groups face struggle for coaches, study finds

***Australian sport faces a looming shortfall in experienced coaches as the workforce ages and less people are entering the system, a Deakin University study has found.***

In a first for Australian sport, researchers with Deakin's Centre for Exercise and Sports Science have examined the true nature of the Australian coaching workforce. They have found that more than 40 per cent of coaches are aged over 50 years and that more than half have less than 10 years experience.
"That we are seeing a drop in the number of next generation coaches is concerning for the future of Australian sports," said Deakin sports coaching expert Dr Andrew Dawson.
"As the population is growing so too is the growth in sports participation across the board. However, the number of new coaches coming into the system is not matching that growth. Couple this with an ageing workforce, and many coaches not staying beyond 10 years, and we could see sports organisations struggle to meet the demand for coaching services especially at the grass roots level where good quality coaching is seen by community sport organisations as integral to their viability and success," he said.
"If we are to ensure the future of sport in Australia, we need address the issues that are highlighted in our study and better support and nurture the enthusiasm, dedication and experience of at all levels of the performance spectrum.
"In particular, we need to find better ways to develop the volunteers (the mum and dad coaches) who are the backbone of sports in this country. The recent focus on coach development has been on the performance of our elite and professional coaches but this research reveals there is a much bigger problem emerging in the long-term development of Australia's coaching workforce."
The Profiling the Australian Coaching Workforce study involved interviews with 40 coaches and a survey of 1374 coaches from the grassroots of community and schools sport through to the professional level. The results provide an insight into the work coaches do, what motivates them, the rewards and costs of being a coach and what they believe are their developmental needs that will enhance their role and the performance of the athletes they train.
"As many as 659,000 coaches are working at all levels within the Australian sport system and each week they influence the lives of more than seven million Australians," Dr Dawson said.
"We found that the coaching workforce is made up of a diverse and dynamic group of dedicated individuals who give up their personal time and money to develop themselves and the athletes they coach.

"Overall, coaches enjoy their work. They began coaching because they wanted to give something back to their sport and continue to coach because of the intrinsic rewards such as seeing their athletes develop and succeed. "However, coaches did cite the stress of coaching can take a toll on their health and personal finances."
The results of the study highlighted a number of significant barriers to becoming a coach and few incentives to continue coaching.
"Key barriers to continuing coaching included the administrative demands and conflicts with key stakeholders such as administrators and parents," Dr Dawson said.
"Many of the issues the coaches highlighted could be addressed through national, state and community sport organisations providing better support for coaches to help them cover the financial costs of travel and further education and development, including opportunities to develop non-traditional coaching skills in areas such as leadership, counselling, conflict resolution and business development."
Dr Dawson said that what is missing is a nationally coordinated approach to coach development that is across the whole sport system.
"Two decades ago, Australia's coach education system was considered world class, but since the mid-1990s national sport policy changes led to coach development becoming decentralised and managed by each sport resulting in a gradual fragmentation of coach development in key areas such as mentoring and socio-cultural, ethical and health issues that are common among coaches across all sports," he said.
"The world leaders in long-term coach development are Canada and the UK who provide support for national, state and community sport organisations that is evidence-based and considered world's best practice. If we are serious about performing better in sport at all levels of participation, it is time we made coach development a national priority."
***Download a copy of the full report here.*** *Provided by Deakin University*

***http://phys.org/news/2013-03-skin-eating-amphibian.html***

## New skin-eating amphibian discovered

***Scientists have discovered a new species of caecilian - a worm-like amphibian - whose young peel off and eat their mother's skin.***

This new species, named Microcaecilia dermatophaga, is the first species of caecilian to be discovered in French Guiana for 150 years, and is one of only four species whose young are known to feed in this way. Its name, which means 'little skin-eating caecilian', refers to this unusual child-rearing strategy.
'What we've found is another species that's a skin-feeder, but most importantly, it's another species that's quite distantly related to other skin-feeders we've found, meaning that skin-feeding is probably an ancestral characteristic for caecilians,' says Dr Emma Sherratt from Harvard University, who discovered them during her PhD when she was working at the Natural History Museum, London.

***Presumed mother with two hatchlings during the period of extended post-hatching parental care and maternal dermatophagy.***

Caecilians (pronounced siss-ee-lee-an) are amphibians, like frogs or toads, but are often mistaken for worms or snakes because they have no legs. Little is known about these strange creatures that have existed since before the dinosaurs. They live only in the moist tropics and most species live underground, so they are difficult to study.
Their colour ranges from pink to dark grey and they have ring-like ridges along their body, adding to their worm-like appearance. But unlike worms, caecilians have large mouths and sharp teeth that they use to eat invertebrates like worms and termites. Their eyes are covered by bone, so they are nearly blind and only see in black and white, but tentacles on the front of their head detect chemicals in the soil, giving them a 'sixth sense'.
This new species is unique because it has many fewer ridges along its body, and is more pink in colour than its closest relations, as it lacks the pigment that gives them a dark grey colour. It is also one of only four species whose young eat their mothers' skin.
To feed their young, females grow an extra layer of skin that's rich in fats. The young scrape this skin off with their teeth and eat it. To help them, they have a specialised set of teeth adapted to the job, which are replaced by more pointed adult teeth as they get older.
What's particularly surprising is that these amphibians feed their young in the same way as other, distantly-related caecilians. This may help scientists understand how caecilians as a group evolved.
'From an evolutionary perspective, finding another species that's a skin feeder gives us a better understanding of when this trait actually evolved,' says Sherratt, 'and perhaps whether it has evolved many times or whether it's a

key characteristic for a lot of species, in which case caecilians may have developed this specialised maternal care very early on in their evolution.'
The discovery of this new species may help us understand these enigmatic amphibians.
'They are still very poorly understood compared to most other creatures,' says Sherratt, 'so any information we do find out gives us that just that little bit more knowledge about the secret lives of these creatures that spend their entire lives underground, and seemingly have been around for a very long time.'
'Molecular estimates tell us they are probably around 250 million years old, which means caecilians survived whatever killed off dinosaurs, and many other creatures around the world, and for that reason I think they're quite extraordinary.'
*This research is published in PLoS One.*
*More information: Wilkinson, M. et al. (2013) A New Species of Skin-Feeding Caecilian and the First Report of Reproductive Mode in Microcaecilia (Amphibia: Gymnophiona: Siphonopidae). PLoS ONE 8(3): e57756. doi:10.1371/journal.pone.0057756*

***http://www.sciencedaily.com/releases/2013/03/130325124358.htm?***

## T-Cell Therapy Eradicates an Aggressive Leukemia in Two Children

***Two children with an aggressive form of childhood leukemia had a complete remission of their disease after treatment with a novel cell therapy***

Two children with an aggressive form of childhood leukemia had a complete remission of their disease -- showing no evidence of cancer cells in their bodies -- after treatment with a novel cell therapy that reprogrammed their immune cells to rapidly multiply and destroy leukemia cells.
A research team from The Children's Hospital of Philadelphia and the University of Pennsylvania published the case report of two pediatric patients Online First today in The New England Journal of Medicine. It will appear in the April 18 print issue.
One of the patients, 7-year-old Emily Whitehead, was featured in news stories in December 2012 after the experimental therapy led to her dramatic recovery after she relapsed following conventional treatment. Emily remains healthy and cancer-free, 11 months after receiving bioengineered T cells that zeroed in on a target found in this type of leukemia, called acute lymphoblastic leukemia (ALL).
The other patient, a 10-year-old girl, who also had a complete response to the same treatment, suffered a relapse two months later when other leukemia cells appeared that did not harbor the specific cell receptor targeted by the therapy.
"This study describes how these cells have a potent anticancer effect in children," said co-first author Stephan A. Grupp, M.D., Ph.D., of The Children's Hospital of Philadelphia, where both patients were treated in this clinical trial. "However, we also learned that in some patients with ALL, we will need to further modify the treatment to target other molecules on the surface of leukemia cells."
Grupp is the director of Translational Research for the Center for Childhood Cancer Research at The Children's Hospital of Philadelphia, and a professor of Pediatrics at the Perelman School of Medicine at the University of Pennsylvania. Michael Kalos, Ph.D., an adjunct associate professor in the department of Pathology and Laboratory Medicine in the Perelman School of Medicine at Penn, is co-first author on the study.
The current study builds on Grupp's ongoing collaboration with Penn Medicine scientists who originally developed the modified T cells as a treatment for B-cell leukemias. The Penn team reported on early successful results of a trial using this cell therapy in three adult chronic lymphocytic leukemia (CLL) patients in August of 2011. Two of those patients remain in remission more than 2½ years following their treatment, and as the Penn researchers reported in December 2012 at the annual meeting of the American Society of Hematology, seven out of ten adult patients treated at that point responded to the therapy. The team is led by the current study's senior author, Carl H. June, M.D., the Richard W. Vague Professor in Immunotherapy in the department of Pathology and Laboratory Medicine and the Perelman School of Medicine at the University of Pennsylvania and director of Translational Research in Penn's Abramson Cancer Center.
"We're hopeful that our efforts to treat patients with these personalized cellular therapies will reduce or even replace the need for bone marrow transplants, which carry a high mortality risk and require long hospitalizations," June said. "In the long run, if the treatment is effective in these late-stage patients, we would like to explore using it up front, and perhaps arrive at a point where leukemia can be treated without chemotherapy."
The research team colleagues adapted the original CLL treatment to combat another B-cell leukemia: ALL, which is the most common childhood cancer. After decades of research, oncologists can currently cure 85 percent of children with ALL. Both children in the current study had a high-risk type of ALL that stubbornly resists conventional treatments.

The new study used a relatively new approach in cancer treatment: immunotherapy, which manipulates the immune system to increase its cancer-fighting capabilities. Here the researchers engineered T cells to selectively kill another type of immune cell called B cells, which had become cancerous.
T cells are the workhorses of the immune system, recognizing and attacking invading disease cells. However, cancer cells fly under the radar of immune surveillance, evading detection by T cells. The new approach custom-designs T cells to "see" and attack the cancer cells.
The researchers removed some of each patient's own T cells and modified them in the laboratory to create a type of CAR (chimeric antigen receptor) cell called a CTL019 cell. These cells are designed to attack a protein called CD19 that occurs only on the surface of certain B cells.
By creating an antibody that recognizes CD19 and then connecting that antibody to T cells, the researchers created in CTL019 cells a sort of guided missile that locks in on and kills B cells, thereby attacking B-cell leukemia. After being returned to the patient's body, the CTL019 cells multiply a thousand times over and circulate throughout the body. Importantly, they persist for months afterward, guarding against a recurrence of this specific type of leukemia.
While the CTL019 cells eliminate leukemia, they also can generate an overactive immune response, called a cytokine release syndrome, involving dangerously high fever, low blood pressure, and other side effects. This complication was especially severe in Emily, and her hospital team needed to provide her with treatments that rapidly relieved the treatment-related symptoms by blunting the immune overresponse, while still preserving the modified T cells' anti-leukemia activity.
"The comprehensive testing plan that we have put in place to study patients' blood and bone marrow while they're undergoing this therapy is allowing us to be able to follow how the T cells are behaving in patients in real time, and guides us to be able to design more detailed and specific experiments to answer critical questions that come up from our studies," Kalos said.
The CTL019 therapy eliminates all B cells that carry the CD19 cell receptor: healthy cells as well as those with leukemia. Patients can live without B cells, although they require regular replacement infusions of immunoglobulin, which can be given at home, to perform the immune function normally provided by B cells.
The research team continues to refine their approach using this new technology and explore reasons why some patients may not respond to the therapy or may experience a recurrence of their disease. Grupp said the appearance of the CD19-negative leukemia cells in the second child may have resulted from her prior treatments. Unlike Emily, the second patient had received an umbilical cord cell transplant from a matched donor, so her engineered T cells were derived from her donor (transplanted) cells, with no additional side effects. Oncologists had previously treated her with blinatumomab, a monoclonal antibody, in hopes of fighting the cancer. The prior treatments may have selectively favored a population of CD19-negative T cells.
"The emergence of tumor cells that no longer contain the target protein suggests that in particular patients with high-risk ALL, we may need to broaden the treatment to include additional T cells that may go after additional targets," added Grupp. "However, the initial results with this immune-based approach are encouraging, and may later even be developed into treatments for other types of cancer."
*Funding from the National Institutes of Health (grants 1RO1 CA165206, R01 CA102646 and R01 CA116660), the Leukemia and Lymphoma Society, and the Alliance for Cancer Gene Therapy supported this study.*
*In August 2012, the University of Pennsylvania and Novartis announced an exclusive global research and licensing agreement to further study and commercialize these novel cellular immunotherapies using chimeric antigen receptor (CAR) technologies. As part of the transaction, Novartis acquired exclusive rights from Penn to CART-19, the therapy that was the subject of this clinical trial and which is now known as CTL019.*
*Stephan A. Grupp, Michael Kalos, David Barrett, Richard Aplenc, David L. Porter, Susan R. Rheingold, David T. Teachey, Anne Chew, Bernd Hauck, J. Fraser Wright, Michael C. Milone, Bruce L. Levine, Carl H. June. Chimeric Antigen Receptor–Modified T Cells for Acute Lymphoid Leukemia. New England Journal of Medicine, 2013; : 130325090025003 DOI: 10.1056/NEJMoa1215134*

***http://www.wired.com/wiredscience/2013/03/just-an-eye-drop-of-poison/***

## Just an (Eye) Drop of Poison

***Eye-drop poisoning is more routine you might think***

- **By Deborah Blum**

Earlier this month, California police arrested a man for spiking his girlfriend's drink with poison following a quarrel. He didn't deny it: the evidence included texts he'd sent to his buddies, bragging about his plan to make her pay for what she'd said to him.
But what poison did he admit to using? Dismiss any thoughts of the classic toxic elements such as arsenic, or murder mystery favorites such as cyanide. In this case, we're simply talking about a bottle of Visine eye drops.

Surprised? You shouldn't be. Eye-drop poisoning is more routine you might think. Remember the Ohio man arrested last year for sending his father to the hospital by putting two bottles of Visine into his milk? The Pennsylvania woman who'd been sneaking Visine into her boyfriend's drinking water for three years? (The poor man suffered all that time with nausea, breathing and blood pressure problems). Oh, and let's not forget the Wyoming teenager who was angry with her step-mother; the girl just pleaded no contest to aggravated assault charges this Friday.

Risky encounters with eyedrops have turned up on poison center roundups; the myth-busting website Snopes.com has tallied up even more. And those are lists of deliberate eye drop attacks. Let's not forget the hazards posed by accidental poisonings; the U.S. Food and Drug Administration has issued a warning to parents about leaving eye drops containers around where they might be found my children.

Snopes took up the question to debunk an apparent belief that sneaking eye drops into a drink would basically induce a hilarious case of diarrhea — a scenario portrayed in a prank scene in the 2005 movie *Wedding Crashers*. Did I mention that Snopes specializes in myth busting? The website labeled the diarrhea scenario false and more. It went on to issue this warning: "Ingestion of such a concoction is downright dangerous making this 'harmless' form of retaliation fraught with hazard."

Fraught with hazard? How, you may ask, does a formula that we trust enough to routinely drip it into our eyes suddenly sound so dangerous? There's a reason, actually, why this is an eyes-only formula, why the Visine labels state: "If swallowed get medical help or contact a poison control center right away." (As an aside, almost all the news stories mention Visine specifically because it's most often used due probably to popularity. But let's not forget there are others, including Murine Tears Plus, Eyesine and Tyzine).

Anyway, the active ingredient in these products is a compound called tetrahydrozoline, which turns to be a neat little arrangement of carbon, hydrogen, plus a little nitrogen. Or to be more specific it has the chemical formula: $C_{13}H_{16}N_2$. It belongs to a family of compounds known for their ability to induce chemical reactions that either relax or constrict blood vessels. The former tend to end up in medications used to reduce blood pressure. The latter, which includes tetrahydrozoline, often go into nasal sprays or in eye drop formulas that are designed to "get the red out."

This is not, by the way, a simple blood vessel squeeze. It derives from the way these compounds bind to receptors in the sympathetic nervous system, altering signals to the vascular system, triggering the change. It's this action on the nervous system which puts tetrahydrozoline in the "neurotoxic" category on the Material Safety Data Sheet required of all manufactured chemical compounds.

And this neurotoxicity tells us why eyedrops are indeed fraught with hazard if you swallow them — or if you sneakily induce others to swallow them. Used as directed, they may indeed give you that clear-eyed look but that's mostly due to the constriction of blood vessels in the eye. Internally they also induce vasoconstriction (as Toxnet calls it). The resulting symptoms are nothing, nothing at all like the Hollywood version of events. They include rapid heart beat, nausea, blurred vision, drowsiness, convulsions. The Toxnet entry, based partly on cases of children who swallowed a bottle of eyedrops or nosedrops left carelessly on a table or counter, notes that "drowsiness and mild coma" often alternate with periods of thrashing and hyperactivity.

What does this tell us, aside from the obvious home precautionary warnings (don't leave your eye drop bottles lying around the house and, by the way don't drink them)?

The record tells us that tetrahydrozoline while poisonous is not a top-of-line-lethal substance. According to the safety sheet, acute oral toxicity in lab mice stands at an LD50 of 345 mg/kg. (LD50 stands for lethal dose 50 percent, meaning the amount of a toxic substance that will kill half of a test population). For comparison, the LD50 of potassium cyanide in mice is 5 mg/kg. And that difference means that while people do end up the hospital, they tend to survive the stay. This is good news for victims and also for perpetrators, as so many of them end up arrested thanks in part to the very characteristic symptoms of eye drop poisoning.

Still as I also said earlier this is not really a tale of a classic homicidal poison like cyanide or one of devious plotting. This is more a tale of impulse, of anger, of grabbing a handy bottle. And, the California case I cited at the top of this story also reminds us that some people take movies like *Wedding Crashers* way too seriously; those incriminating texts of his indicated that idea was to make the girlfriend "crap for talking crap," basically a scenario right out of the movie.

We could make a case here that entertainment comedies aren't really the most reliable source of toxicology information. But for poisoners of the Visine caliber, here's another recommendation as well — go for more serious entertainment. Perhaps all this trouble could have been avoided if the angry boyfriend watched CSI instead. In an episode titled "Revenge Served Cold" an eye-drop-spiked drink doesn't just send the victim to the hospital, it kills him.

Over the top, sure. But take a note anyway.

http://bit.ly/17535SN

# Master gene helps weeds defy all weedkillers

***No wonder wheat and barley farmers in Europe and Australia have trouble killing ryegrass and black-grass. These plants have a master gene that makes some strains resistant to all major herbicides.***

**19:00 25 March 2013 by Andy Coghlan**

"This is a general detoxification mechanism, just like the way tumours detoxify anticancer drugs," says Robert Edwards of the University of York, UK. The good news is, if the gene can be neutralised, it would take out the weeds' resistance at a stroke.

That could be very desirable as black-grass and annual ryegrass pose a growing problem for cereal farmers. In Australia, for example, the plants can wreck entire harvests.

Most forms of herbicide resistance work by sabotaging the specific target against which a weedkiller acts. But the troublesome grasses have a gene called AmGSTF1, which makes an enzyme called a glutathione transferase. This causes the plant to make lots of protective antioxidants. These chemically neutralise many toxins in much the same way that human tumours – which also rely on glutathione transferases – can resist many drugs.

When Edwards transferred the AmGSTF1 gene from black-grass into thale cress, the cress became resistant to the herbicides.

However, Edwards's team also discovered a solution: a drug called 4-chloro-7-nitro-benzoxadiazole. It is sometimes given to people with cancers that are resistant to many drugs, because it blocks glutathione transferases. When Edwards sprayed it on the weedkiller-resistant cress, the cress became vulnerable again.

Edwards says the drug is too toxic for use on farms, but benign versions could be developed to switch off resistance, enabling farmers to kill weeds. His team is also trying to develop a test so farmers can quickly tell whether weeds have an active master gene. *Journal reference: PNAS, DOI: 10.1073/pnas.1221179110*

http://phys.org/news/2013-03-scientists-moon-asteroids-history.html

# Scientists find Moon, asteroids share history

***NASA and international researchers have discovered that Earth's moon has more in common than previously thought with large asteroids roaming our solar system.***

Phys.org - Scientists from NASA's Lunar Science Institute (NLSI) in Moffett Field, Calif., discovered that the same population of high-speed projectiles that impacted our lunar neighbor four billion years ago, also hit the giant asteroid Vesta and perhaps other large asteroids.

The research unveils an unexpected link between Vesta and the moon, and provides new means for studying the early bombardment history of terrestrial planets. The findings are published in the March issue of Nature Geoscience.

"It's always intriguing when interdisciplinary research changes the way we understand the history of our solar system," said Yvonne Pendleton, NLSI director. "Although the moon is located far from Vesta, which is in the main asteroid belt between the orbits of Mars and Jupiter, they seem to share some of the same bombardment history."

The findings support the theory that the repositioning of gas giant planets like Jupiter and Saturn from their original orbits to their current location destabilized portions of the asteroid belt and triggered a solar system-wide bombardment of asteroids billions of years ago, called the lunar cataclysm.

The research provides new constraints on the start and duration of the lunar cataclysm, and demonstrates that the cataclysm was an event that affected not only the inner solar system planets, but the asteroid belt as well.

The moon rocks brought back by NASA Apollo astronauts have long been used to study the bombardment history of the moon. Now the ages derived from meteorite samples have been used to study the collisional history of main belt asteroids. In particular, howardite and eucrite meteorites, which are common species found on Earth, have been used to study asteroid Vesta, their parent body. With the aid of computer simulations, researchers determined that meteorites from Vesta recorded high-speed impacts which are now long gone.

Researchers have linked these two datasets and found that the same population of projectiles responsible for making craters and basins on the moon were also hitting Vesta at very high velocities, enough to leave behind a number of telltale, impact-related ages.

The team's interpretation of the howardites and eucrites was augmented by recent close-in observations of Vesta's surface by NASA's Dawn spacecraft. In addition, the team used the latest dynamical models of early main belt evolution to discover the likely source of these high velocity impactors. The team determined that the population of projectiles that hit Vesta had orbits that also enabled some objects to strike the moon at high speeds.

"It appears that the asteroidal meteorites show signs of the asteroid belt losing a lot of mass four billion years ago, with the escaped mass beating up on both the surviving main belt asteroids and the moon at high speeds" says lead author Simone Marchi, who has a joint appointment between two of NASA's Lunar Science Institutes, one at the Southwest Research Institute in Boulder, Colo., and another at the Lunar and Planetary Institute in Houston. "Our research not only supports the current theory, but it takes it to the next level of understanding."

***http://www.eurekalert.org/pub_releases/2013-03/uotm-pcv032513.php***

## Potential Chagas vaccine candidate shows unprecedented efficacy

***Scientists also identify simple vaccine delivery model in 'breakthrough' discovery; Novel vaccine could arrive in veterinary market in as few as 5 years***

Scientists are getting closer to a Chagas disease vaccine, something many believed impossible only 10 years ago. Research from the Sealy Center for Vaccine Development at the University of Texas Medical Branch at Galveston has resulted in a safe vaccine candidate that is simple to produce and shows a greater than 90 percent protection rate against chronic infection in mice.

In a paper published online in PLOS ONE, the researchers describe how they identified and tested potential Trypanosoma cruzi (also known as T. cruzi or Chagas disease) antigen candidates and delivery models to establish the safety and efficacy of a vaccine formulation known asTcVac3. This potential vaccine could halt the irreversible heart and organ damage that afflicts approximately 30 percent of those infected with Chagas.

"This signals a scientific breakthrough – unprecedented vaccine efficacy for a common parasitic disease with no cure for chronic sufferers," said lead author Nisha Garg, PhD, professor of microbiology, immunology and pathology at UTMB. "If this vaccine proves practical, it could be approved in as few as five years for use in canines, which are reservoir hosts of the disease. As many as 20 percent of dogs may be infected in Texas alone, developing the same heart conditions as humans but mistaken by vets for heartworm."

The study also provides further evidence that a human Chagas vaccine is possible, a topic of debate among some who still believe that Chagas heart disease is the result of an autoimmune disorder, she added.

T. cruzi, transmitted by the triatomine insect, or "kissing bug," is prevalent in almost all Latin American countries and is becoming more common in the U.S. The World Health Organization estimates that approximately 10 million people – mostly children – are infected worldwide. Approximately 13,000 die each year from the complications of Chagas-induced heart disease – a result of the chronic infection Garg and her team aim to vaccinate against. It is estimated that the global economic burden of Chagas is about $7 billion a year.

**TcVac3: The Path of Discovery**

TcVac3 is the result of rigorous computational/bioinformatics analysis and screening of the T. cruzi genome for potential candidate antigens over several years by Garg and her team. These analyses led the researchers to three potential antigens (TcG1, TcG2 and TcG4) for further investigation.

Next, they began testing these antigens and potential vaccine delivery models – how the components are arranged in the actual vaccine – to determine the best approaches.

Early experiments proved that delivery of the candidate antigens by a DNA-prime/protein boost approach, along with co-delivery of IL-12 and GM-CSF cytokine adjuvants meant to enhance the immune response, was effective in generating antibody and T cell responses capable of providing more than 90 percent control of acute infection and parasite burden in infected mice.

Recognizing, however, that this vaccine delivery model was quite complex, the scientists sought to simplify the vaccine using a DNA-prime/Modified Vaccinia Ankara (MVA)-boost approach – a delivery model that offers many advantages: it can accommodate multiple foreign genes in its genome; may be administered by a variety of routes; has an excellent safety record; and has been shown to generate immune responses to a variety of foreign antigens. MVA itself can act as an adjuvant since it provides a signal to the innate immune system and can boost T cells.

Based on preliminarystudies by the researchers that showed this delivery model to be potent, the scientists next tested the protective efficacy of TcVac3, constituted of just the TcG2 and TcG4 candidates and lacking the adjuvants, delivered by the DNA/MVA approach.

With two doses of the vaccine, the mice with TcVac3-induced antibodies exhibited 92 to 96 percent protection against chronic infection. They found that the DNA/MVA approach increased the vaccine efficacy enough to omit one of the antigens and the adjuvants, making it a much simpler but still highly effective vaccine.

"Because Chagas is most prevalent in developing countries, it is essential that a potential vaccine be inexpensive to develop and easy to deliver," said Garg. "TcVac3 accomplishes this goal, making it not just an effective candidate, but an ideal one."

Future research will determine if the vaccine composition can be simplified even further. In addition, the scientists are already conducting related trials in canines. Garg and her team are also working on pre-clinical trials of human patient samples, testing for immune response in patients that are already infected but not showing signs of chronic disease. Results of both studies are anticipated later this year.
*Shivali Gupta, Ph.D., also contributed to this study. Funding for the research was provided by National Institutes of Health and the American Heart Association.*

***http://www.eurekalert.org/pub_releases/2013-03/bmj-efl032613.php***

## Experts find link between low doses of vitamin D and adverse pregnancy outcomes

***Supplements may reduce these risks***

There is a link between vitamin D insufficiency and adverse health outcomes such as gestational diabetes and preeclampsia in mothers-to-be and low birth weight in newborns, suggests a paper published on bmj.com today.
Vitamin D insufficiency has been associated with a number of adverse health outcomes and has been recognised as a public health concern. Plus, observational data has suggested a link between low vitamin D and increased risk of adverse pregnancy outcomes (such as gestational diabetes, preeclampsia, risk of infections, caesarean section and foetal growth restriction). Knowledge of these associations is however limited.
Literature on this topic is growing rapidly. As such, researchers from the University of Calgary in Canada carried out a systematic review and meta-analysis of all existing evidence on the effect of vitamin D concentration on pregnancy and birth outcomes.
Data from 31 studies were included in the analysis - all published between 1980 and 2012 with between 95 and 1,100 participants. Differences in study design and quality were taken into account to minimise bias.
Results showed that pregnant women with low levels of 5-OH vitamin D were more likely to develop gestational diabetes (odds ratio of 0.49), had an increased chance of developing preeclampsia (odds ratio of 0. 79) and an increased chance of giving birth to a baby small for gestational age (odds ratio of 0.85). No significant differences were found in birth length and head circumference.
The researchers say these results are "concerning" given recent evidence that vitamin D insufficiency is common during pregnancy, especially among high risk women, particularly vegetarians, women with limited sun exposure and ethnic minorities with darker skin.
The researchers conclude that the findings identify a significant association, but there remains a need for large, well-designed randomized controlled trials to determine whether "strategies to optimize vitamin D concentration are effective in improving pregnancy and neonatal outcomes". They also suggest that future studies should look at the dose-response relationship between vitamin D supplements and adverse health outcomes.
In an accompanying editorial, Dr Lucas from the National Centre for Epidemiology and Population Health at the Australian National University says that the findings of this study support a goal of vitamin D sufficiency for all pregnant women. She says that "supplements, diet and sunlight exposure" are all influences which "should be used together, with care". Dr Lucas adds that a previous editorial called for large well designed controlled trials "to clarify the causal association" which she believes is needed to find the magnitude of importance between vitamin D and pregnancy.

***http://www.eurekalert.org/pub_releases/2013-03/aafc-ebi032013.php***

## Early-onset baldness in African-American men may be linked to prostate cancer

***Baldness was associated with an increased risk of prostate cancer among African-American men***

PHILADELPHIA — Baldness was associated with an increased risk of prostate cancer among African-American men, and risk for advanced prostate cancer increased with younger age and type of baldness, according to data published in Cancer Epidemiology, Biomarkers & Prevention, a journal of the American Association for Cancer Research.
"We focused on African-American men because they are at high risk for developing prostate cancer and are more than twice as likely to die from prostate cancer than other groups in the United States," said Charnita Zeigler-Johnson, Ph.D., research assistant professor at the Center for Clinical Epidemiology and Biostatistics at the University of Pennsylvania in Philadelphia. "Although this is a high-risk group for poor prostate cancer outcomes, no published study had focused on evaluating baldness as a potential risk factor in a sample of African-American men."
Zeigler-Johnson and her colleagues identified 318 men with prostate cancer and 219 controls among participants who enrolled in the Study of Clinical Outcomes, Risk and Ethnicity (SCORE) between 1998 and 2010. All of them were African-American and had varying degrees of baldness. They obtained information on type of baldness (none, frontal and vertex) and other medical history using a questionnaire.

The researchers found that any baldness was associated with a 69 percent increased risk of prostate cancer. In particular, African-American men with frontal baldness, and not vertex baldness, were more than twice as likely to have been diagnosed with advanced prostate cancer. This association was even stronger among men who were diagnosed when younger than 60, with a sixfold increase in high-stage prostate cancer and a fourfold increase in high-grade prostate cancer. In addition, among younger men with prostate cancer, those with frontal baldness were more likely to have a high prostate-specific antigen level at diagnosis.
"Early-onset baldness may be a risk factor for early-onset prostate cancer in African-American men, particularly younger men," said Zeigler-Johnson. "Pending future studies to confirm our results, there is a potential to use early-onset baldness as a clinical indicator of increased risk for prostate cancer in some populations of men."

***http://www.eurekalert.org/pub_releases/2013-03/uoc--rsp032613.php***

## Research suggests popular diabetes drugs can cause abnormal pancreatic growth in humans

***Individuals who had taken a type of drug commonly used to treat Type 2 diabetes showed abnormalities in the pancreas***

Individuals who had taken a type of drug commonly used to treat Type 2 diabetes showed abnormalities in the pancreas, including cell proliferation, that may be associated with an increased risk of neuroendocrine tumors, according to a new study by researchers from UCLA and the University of Florida. Their findings were published online March 22 in the journal Diabetes. The researchers, from the Larry L. Hillblom Islet Research Center at UCLA and the Diabetes Center at the University of Florida, found that cell mass was increased approximately 40 percent in the pancreases of deceased organ donors who had Type 2 diabetes and who had been treated with incretin therapy. This widely used type of treatment takes advantage of the action of a gut hormone known as glucagon-like peptide 1 (GLP-1) to lower blood sugar in the body.
Although there have been conflicting reports on the effects of the incretin class of drugs on the pancreas in animal studies, this is the first study to note such changes in the human pancreas. The research was made possible by a unique research consortium called nPOD (Network for Pancreatic Organ Donors with Diabetes), led by Dr. Mark Atkinson, a professor of pathology and pediatrics at the University of Florida. The network, which is funded by the Juvenile Diabetes Research Foundation, obtains pancreases from deceased organ donors, with permission of their next of kin, to better understand diabetes by investigating tissues of those with the disease.
"There is an increasing appreciation that animal studies do not always predict findings in humans," said Dr. Peter Butler, director of UCLA's Hillblom Islet Research Center and chief of the endocrinology, diabetes and hypertension unit. "The nPOD program is therefore a very precious resource." The researchers examined the pancreases of 20 deceased organ donors with Type 2 diabetes. Eight had been treated for at least a year with incretin therapy, while the other 12 had received therapies that didn't include incretin-based drugs. The researchers also evaluated 14 pancreases from a control group of non-diabetic individuals of similar age.
The pancreases of the individuals who had been on incretin therapy were larger than those of patients on other types of diabetes therapies, and this larger size was associated with increased cellular proliferation. Incretin-treated individuals showed an increase in pancreas dysplasia, an abnormal form of cell proliferation that is a risk factor for pancreatic cancer, as well as an expansion of alpha cells, endocrine cells that make the hormone glucagon.
This latter finding is likely a consequence of GLP-1–based therapies' suppression of the release of glucagon by alpha cells, since decreasing the availability or action of the hormone glucagon has been shown in a variety of prior studies to induce a proliferation of pancreatic alpha cells. This alpha-cell expansion has been associated with the development of pancreatic neuroendocrine tumors. Three of the eight incretin-treated individuals had microadenomas and one has a neuroendocrine tumor composed of alpha cells.
Of the eight donors who were on incretin therapy, seven had been taking sitagliptin, sold in pill form as Januvia and marketed by Merck, and one had been on exenatide, sold as Byetta by Bristol-Myers Squibb. These and similar drugs are currently under investigation by the U.S. Food and Drug Administration for their possible links to pancreatitis and pancreatic cancer.
"These findings lend additional weight to concerns regarding the effects of long term GLP-1–related therapy, with respect to both unintended proliferative actions on the exocrine pancreas and now also a possible increased risk of neuroendocrine tumors," the researchers write. "In addition to the surveillance previously recommended

for the potential association of GLP-1– based therapy and pancreatic cancer risk, the current data imply that surveillance for a possible increased risk of pancreatic neuroendocrine tumors is warranted."
Such surveillance approaches might include MRI imaging of the pancreas and screening for neuroendocrine tumors, Butler said. "The present studies are only from a small number of individuals, and while the findings do raise concerns, it will be important that other approaches are now used in a larger group of living individuals to further investigate these findings," he said.
A recent study led by Dr. Sonal Singh of Johns Hopkins University School of Medicine and Public Health and published in JAMA Internal Medicine suggested a doubling in the risk of hospitalization for acute pancreatitis with the GLP-1–based therapies and also recommended further research.
"Since most risk factors for acute pancreatitis are also linked to an increased risk of pancreatic cancer, these findings of changes in the human pancreas are very concerning," said Singh, an assistant professor of medicine and international health. "Now that GLP-1–based therapies have been shown to increase the risk of pancreatic inflammation and abnormal cell proliferation, further studies are needed to urgently clarify whether these linkages lead to pancreatic cancer with long-term use."
*Study co-authors, in addition to Butler and Atkinson, are Alexandra E. Butler, Tatyana Gurlo and David W. Dawson, all of UCLA, and Martha Campbell-Thompson of the University of Florida.*
*Grants from National Institute of Diabetes and Digestive and Kidney Diseases (DK059579, DK061539 and DK077967), the Hillblom Foundation, and the Peter and Valerie Kompaniez Foundation funded this study. The Juvenile Diabetes Research Foundation funds the nPOD program.*

***http://www.scientificamerican.com/article.cfm?id=how-to-ace-an-interview-feel-powerful***

# How to Ace an Interview: Feel Powerful

***Experiments show that simple psychological preparations make a big difference***

**By Cindi May | Tuesday, March 26, 2013 | 5**

In today's competitive job market, hopeful employees want to know what qualities lead one job candidate to prevail over dozens of other capable contenders. If we consider the recent appointment of Cardinal Jorge Bergoglio to the highest post in the Catholic Church, then humility, servility, and meekness may top the list. Numerous anecdotes about Pope Francis' unassuming nature have surfaced since his selection, including stories of him rejecting a chauffeur-driven car and images of him washing the feet of women. Perhaps the lesson here is that job seekers should reflect on their own relative insignificance, and strive to convey modesty, restraint, and vulnerability in the interview process.
This may be the right strategy — if you have a shot at the papacy. But if you are trying to secure a spot in the American business world, new research suggests that priming your powerful side is the way to go. A sense of power, it seems, increases your appeal both on paper and in person to those making hiring decisions.
It is already well established that people who feel empowered pay more attention to rewarding information, express themselves more freely when interacting with others, and experience more positive emotion. They also tend to be more persuasive, less susceptible to the influence of others, and more confident. Power breeds optimism, higher self-esteem, and action in pursuit of goals. By contrast, those lacking in perceived power experience a reduced sense of control and diminished access to resources or rewards, which in turn may lead to pessimism, depression, a withdrawal from activity, and poor health.
Joris Lammers and colleagues recently explored whether a sense of power, even if temporary, could improve success in the job interview process. In a series of studies, Lammers manipulated perceived power by asking participants to write about a prior personal experience in which they either had power (high power prime) or lacked power (low power prime). In one study, participants were primed for high or low power and then read a job ad for a sales analyst position. They were asked to assume that they possessed the necessary training and experience for the position, and to write an application letter. In a second study, participants were primed for high or low power, and then engaged in a 15 minute face-to-face mock interview for entrance into business school. This study also included a baseline group that received no prime manipulation before the interview.
In both studies, the candidates were evaluated by individuals who did not know about the power manipulation. In the first experiment, the raters had no interaction whatsoever with the participants, other than reading their applications. In that study, raters were significantly more inclined to offer the position to power-primed candidates than to those primed to feel powerless. In the second study, raters gave a yes or no judgment regarding acceptance of the candidates into business school, and also assessed how persuasive the candidates were during the interview. The high-power prime increased the likelihood of acceptance by 81 percent compared to the baseline (no prime) condition, and by 162 percent compared to the low-power prime condition.

Not too surprisingly, high-power applicants were also perceived as more persuasive than either baseline or low-power applicants.
It may thus be wise to conjure a memory of a time when you were in charge and felt powerful as you prepare for your next interview. Indeed, it may even be tempting to engage in this type of exercise in a variety of arenas, for example, when buying a car or trying to talk your way out of a speeding ticket. Before you adopt personal power as your universal inner mantra, however, know that there are perils and pitfalls to power. As noted earlier, feelings of empowerment compel people to action. These actions may be socially altruistic, like donating to public radio, but they may also be self-serving, like turning off an annoying fan without permission, or even socially caustic, like consuming more than one's fair share of a common resource. Furthermore, feelings of power may make you less sensitive to the interests and positions of others, more likely to stereotype others, and less interested in seeking confirming evidence for your opinions or judgments.
In some cases, power can really go to your head. Empowered people are not only more confident that those with less power, they in fact exhibit hubristic overconfidence, both about events within their control and about events that are clearly outside their control. Individuals with a high sense of power, for example, are not only more likely to believe that their achievements will be celebrated by the press, they are also less likely to believe that they will experience turbulence on a plane or encounter a venomous snake. This overconfidence creates a sense of invulnerability, which in turn can result in a willingness to engage in risky behaviors, like having sex without a condom or taking bigger risks at the black jack table. In negotiations, this risk-tolerance leads empowered individuals to reveal more about their preferences and priorities, potentially leaving them vulnerable to their opponent's tactics.
Power as a pervasive personal philosophy clearly has its drawbacks. We have known this more than 100 years: Power corrupts and absolute power corrupts absolutely. Research on interpersonal relations certainly seems to support this premise. However, a small dose of power, right before your next interview, may be just what the doctor ordered to improve your odds of landing the job.

***http://www.sciencedaily.com/releases/2013/03/130326100839.htm***

## Scientists Form New Nerve Cells -- Directly in the Brain

***The field of cell therapy, which aims to form new cells in the body in order to cure disease, has taken another important step in the development towards new treatments.***

A new report from researchers at Lund University in Sweden shows that it is possible to re-programme other cells to become nerve cells, directly in the brain.
Two years ago, researchers in Lund were the first in the world to re-programme human skin cells, known as fibroblasts, to dopamine-producing nerve cells – without taking a detour via the stem cell stage. The research group has now gone a step further and shown that it is possible to re-programme both skin cells and support cells directly to nerve cells, in place in the brain.
“The findings are the first important evidence that it is possible to re-programme other cells to become nerve cells inside the brain”, said Malin Parmar, research group leader and Reader in Neurobiology.
The researchers used genes designed to be activated or de-activated using a drug. The genes were inserted into two types of human cells: fibroblasts and glia cells – support cells that are naturally present in the brain. Once the researchers had transplanted the cells into the brains of rats, the genes were activated using a drug in the animals’ drinking water. The cells then began their transformation into nerve cells.
In a separate experiment on mice, where similar genes were injected into the mice’s brains, the research group also succeeded in re-programming the mice’s own glia cells to become nerve cells. “The research findings have the potential to open the way for alternatives to cell transplants in the future, which would remove previous obstacles to research, such as the difficulty of getting the brain to accept foreign cells, and the risk of tumour development”, said Malin Parmar.
All in all, the new technique of direct re-programming in the brain could open up new possibilities to more effectively replace dying brain cells in conditions such as Parkinson’s disease.
“We are now developing the technique so that it can be used to create new nerve cells that replace the function of damaged cells. Being able to carry out the re-programming in vivo makes it possible to imagine a future in which we form new cells directly in the human brain, without taking a detour via cell cultures and transplants”, concluded Malin Parmar.
*Olof Torper, Ulrich Pfisterer, Daniel A. Wolf, Maria Pereira, Shong Lau, Johan Jakobsson, Anders Björklund, Shane Grealish, and Malin Parmar. Generation of induced neurons via direct conversion in vivo. PNAS, 2013 DOI: 10.1073/pnas.1303829110*

http://www.scientificamerican.com/article.cfm?id=tomatoes-peppers-strawberries-in-gr

# Tomatoes, Peppers, Strawberries Now Grow Well in Greenland's Arctic Valleys

***On the Arctic Circle, a chef is growing the kind of vegetables and herbs - potatoes, thyme, tomatoes, green peppers - more fitting for a suburban garden in a temperate zone than a land of Northern Lights, glaciers and musk oxen.***

**By Alistair Scrutton**

KANGERLUSSUAQ, Greenland (Reuters) - On the Arctic Circle, a chef is growing the kind of vegetables and herbs - potatoes, thyme, tomatoes, green peppers - more fitting for a suburban garden in a temperate zone than a land of Northern Lights, glaciers and musk oxen. Some Inuit hunters are finding reindeer fatter than ever thanks to more grazing on this frozen tundra, and for some, there is no longer a need to trek hours to find wild herbs. Welcome to climate change in Greenland, where locals say longer and warmer summers mean the country can grow the kind of crops unheard of years ago.

"Things are just growing quicker," said Kim Ernst, the Danish chef of Roklubben restaurant, nestled by a frozen lake near a former Cold War-era U.S. military base. "Every year we try new things," said Ernst, who even managed to grow a handful of strawberries that he served to some surprised Scandinavian royals. "I first came here in 1999 and no-one would have dreamed of doing this. But now the summer days seem warmer, and longer."

It was minus 20 degrees Centigrade in March but the sun was out and the air was still, with an almost spring feel. Ernst showed his greenhouse and an outdoor winter garden which in a few months may sprout again. Hundreds of miles south, some farmers now produce hay, and sheep farms have increased in size. Some supermarkets in the capital Nuuk sell locally grown vegetables during the summer.

Major commercial crop production is still in its infancy. But it is a sign of changes here that Greenland's government set up a commission this year to study how a changing climate may help farmers increase agricultural production and replace expensive imported foods. Change is already underway. Potatoes grown commercially in southern Greenland reached over 100 metric tons in 2012, double that of 2008. Vegetable production in the region may double this year compared with 2012, according to government data.

Some politicians hope global warming will allow this country a quarter the size of the United States to reduce its dependency on former colonial master Denmark for much of its food as political parties push for full independence.

Greenland, which is self-governing aside from defense and security, depends on an annual grant from Denmark of around $600 million, or half the island's annual budget. But the thawing of its enormous ice sheets have seen a boost in mining and oil exploration, as well as an interest in agriculture.

"I expect a lot of development in farming sheep and agriculture due to global warming," said Prime Minister Kuupik Kleist, whose government set up the commission. "It may become an important supplement to our economy."

Locals love recounting how Erik the Red first arrived in the southern fjords here in the 10th century and labeled this ice-covered island "Greenland" to entice others to settle. There is evidence that the climate was warmer then, allowing Viking settlements to grow crops for five centuries before mysteriously dying out.

**From cows to crops**

The scale of this new agriculture is tiny. There are just a few dozen sheep farms in southern Greenland, where most of the impact of climate change can be seen. Cows may number less than a hundred. But with 57,000 mostly Inuit human inhabitants, the numbers to feed are also small.

"You need to put this into perspective. We used to be high Arctic and now we are more sub Arctic," Kenneth Hoegh, an agronomist and former senior government advisor. "But we are still Arctic."

The symbolism is enormous, however, highlighting a changing global climate that has seen temperatures in the Arctic increase by about twice the global average - about 0.8 degrees Celsius since pre-industrial times.

"There are now huge areas in southern Greenland where you can grow things," said Josephine Nymand, a scientist at the Greenland Institute of Natural Resources in Nuuk. "Potatoes have most benefited. Also, cabbage has been very successful."

Sten Erik Langstrup Pedersen, who runs an organic farm in a fjord near Nuuk, first grew potatoes in 1976. Now he can plant crops two weeks earlier in May and harvest three weeks later in October compared with more than a decade ago. He grows 23 kinds of vegetables, compared with 15 a decade ago, including beans, peas, herbs and strawberries. He says he has sold some strawberries to top restaurants in Copenhagen.

But Pedersen is skeptical about how much it will catch on. "Greenlanders are impatient. They see a seal and they immediately just want to hunt it. They can never wait for vegetables to grow."

There is still potential. Hoegh estimates Greenland could provide half its food needs from home-grown produce which would be competitive with more expensive Danish imports.
But global change is not all about benefits. While summers are warmer, there is less rain. Some experts say that Greenland could soon need irrigation works - ironic for a country of ice and lakes.
"We have had dry summers for the last few years." said Aqqalooraq Frederiksen, a senior agricultural consultant in south Greenland, who said a late spring last year hurt potato crops.
On the Arctic circle, a flash flood last summer from suspected glacier melt water - which some locals here blamed on warm weather - swept away the only bridge connecting Ernst's restaurant to the airport. It came right in the middle of the tourist season, and the restaurant lost thousands of dollars.
It was an ominous reminder that global warming will bring its problems. Still, for Pedersen and his fjord in Nuuk, the future looks good. "The hotter, the better," Pedersen said. "For me."
*(Additional reporting by Katja Vahl in Nuuk; Editing by Sonya Hepinstall)*

***http://phys.org/news/2013-03-global-asteroid-impact-k-pg-extinction.html***

## Global fires after the asteroid impact probably caused the K-Pg extinction

***About 66 million years ago a mountain-sized asteroid hit what is now the Yucatan in Mexico at exactly the time of the Cretaceous-Paleogene (K-Pg) mass extinction.***

Evidence for the asteroid impact comes from sediments in the K-Pg boundary layer, but the details of the event, including what precisely caused the mass extinction, are still being debated.
Some scientists have hypothesized that since the ejecta from the impact would have heated up dramatically as it reentered the Earth's atmosphere, the resulting infrared radiation from the upper atmosphere would have ignited fires around the globe and killed everything except those animals and plants that were sheltered underground or underwater.
Other scientists have challenged the global fire hypothesis on the basis of several lines of evidence, including absence of charcoal-which would be a sign of widespread fires-in the K-Pg boundary sediments. They also suggested that the soot observed in the debris layer actually originated from the impact site itself, not from widespread fires caused by reentering ejecta.
Robertson et al. show that the apparent lack of charcoal in the K-Pg boundary layer resulted from changes in sedimentation rates: When the charcoal data are corrected for the known changes in sedimentation rates, they exhibit an excess of charcoal, not a deficiency. They also show that the mass of soot that could have been released from the impact site itself is far too small to account for the observed soot in the K-Pg layer. In addition, they argue that since the physical models show that the radiant energy reaching the ground from the reentering ejecta would be sufficient to ignite tinder, it would thereby spark widespread fires. The authors also review other evidence for and against the firestorm hypothesis and conclude that all of the data can be explained in ways that are consistent with widespread fires.
*More information: K/Pg extinction: Reevaluation of the heat/fire hypothesis, Journal of Geophysical Research-Planets, doi:10.1002/jgrg.20018 , 2013. http://onlinelibrary.wiley.com/doi/10.1002/jgrg.20018/abstract*

***http://www.sciencedaily.com/releases/2013/03/130326151125.htm***

## 2011 Oklahoma Temblor: Wastewater Injection Spurred Biggest Earthquake Yet, Study Says

***A new study in the journal Geology is the latest to tie a string of unusual earthquakes, in this case, in central Oklahoma, to the injection of wastewater deep underground.***

Researchers now say that the magnitude 5.7 earthquake near Prague, Okla., on Nov. 6, 2011, may also be the largest ever linked to wastewater injection. Felt as far away as Milwaukee, more than 800 miles away, the quake -- the biggest ever recorded in Oklahoma--destroyed 14 homes, buckled a federal highway and left two people injured. Small earthquakes continue to be recorded in the area.
The recent boom in U.S. energy production has produced massive amounts of wastewater. The water is used both in hydrofracking, which cracks open rocks to release natural gas, and in coaxing petroleum out of conventional oil wells. In both cases, the brine and chemical-laced water has to be disposed of, often by injecting it back underground elsewhere, where it has the potential to trigger earthquakes. The water linked to the Prague quakes was a byproduct of oil extraction at one set of oil wells, and was pumped into another set of depleted oil wells targeted for waste storage.
Scientists have linked a rising number of quakes in normally calm parts of Arkansas, Texas, Ohio and Colorado to below-ground injection. In the last four years, the number of quakes in the middle of the United States jumped 11-fold from the three decades prior, the authors of the Geology study estimate. Last year, a group at the U.S. Geological Survey also attributed a remarkable rise in small- to mid-size quakes in the region to

humans. The risk is serious enough that the National Academy of Sciences, in a report last year called for further research to "understand, limit and respond" to induced seismic events. Despite these studies, wastewater injection continues near the Oklahoma earthquakes.

The magnitude 5.7 quake near Prague was preceded by a 5.0 shock and followed by thousands of aftershocks. What made the swarm unusual is that wastewater had been pumped into abandoned oil wells nearby for 17 years without incident. In the study, researchers hypothesize that as wastewater replenished compartments once filled with oil, the pressure to keep the fluid going down had to be ratcheted up. As pressure built up, a known fault -- known to geologists as the Wilzetta fault--jumped. "When you overpressure the fault, you reduce the stress that's pinning the fault into place and that's when earthquakes happen," said study coauthor Heather Savage, a geophysicist at Columbia University's Lamont-Doherty Earth Observatory.

The amount of wastewater injected into the well was relatively small, yet it triggered a cascading series of tremors that led to the main shock, said study co-author Geoffrey Abers, also a seismologist at Lamont-Doherty. "There's something important about getting unexpectedly large earthquakes out of small systems that we have discovered here," he said. The observations mean that "the risk of humans inducing large earthquakes from even small injection activities is probably higher" than previously thought, he said.

Hours after the first magnitude 5.0 quake on Nov. 5, 2011, University of Oklahoma seismologist Katie Keranen rushed to install the first three of several dozen seismographs to record aftershocks. That night, on Nov. 6, the magnitude 5.7 main shock hit and Keranen watched as her house began to shake for what she said felt like 20 seconds. "It was clearly a significant event," said Keranen, the Geology study's lead author. "I gathered more equipment, more students, and headed to the field the next morning to deploy more stations."

Keranen's recordings of the magnitude 5.7 quake, and the aftershocks that followed, showed that the first Wilzetta fault rupture was no more than 650 feet from active injection wells and perhaps much closer, in the same sedimentary rocks, the study says. Further, wellhead records showed that after 13 years of pumping at zero to low pressure, injection pressure rose more than 10-fold from 2001 to 2006, the study says.

The Oklahoma Geological Survey has yet to issue an official account of the sequence, and wastewater injection at the site continues. In a statement responding to the paper, Survey seismologist Austin Holland said the study showed the earthquake sequence could have been triggered by the injections. But, he said, "it is still the opinion of those at the Oklahoma Geological Survey that these earthquakes could be naturally occurring. There remain many open questions, and more scientific investigations are underway on this sequence of earthquakes and many others within the state of Oklahoma."

The risk of setting off earthquakes by injecting fluid underground has been known since at least the 1960s, when injection at the Rocky Mountain Arsenal near Denver was suspended after a quake estimated at magnitude 4.8 or greater struck nearby -- the largest tied to wastewater disposal until the one near Prague, Okla. A series of similar incidents have emerged recently. University of Memphis seismologist Stephen Horton in a study last year linked a rise in earthquakes in north-central Arkansas to nearby injection wells. University of Texas, Austin, seismologist Cliff Frohlich in a 2011 study tied earthquake swarms at the Dallas-Fort Worth Airport to a brine disposal well a third of a mile away. In Ohio, Lamont-Doherty seismologists Won-Young Kim and John Armbruster traced a series of 2011 earthquakes near Youngstown to a nearby disposal well. That well has since been shut down, and Ohio has tightened its waste-injection rules.

Wastewater injection is not the only way that people can touch off quakes. Evidence suggests that geothermal drilling, impoundment of water behind dams, enhanced oil recovery, solution salt mining and rock quarrying also can trigger seismic events. (Hydrofracking itself is not implicated in significant earthquakes; the amount of water used is usually not enough to produce substantial shaking.) The largest known earthquakes attributed to humans may be the two magnitude 7.0 events that shook the Gazli gas fields of Soviet Uzbekistan in 1976, followed by a third magnitude 7.0 quake eight years later. In a 1985 study in the Bulletin of the Seismological Society of America, Lamont-Doherty researchers David Simpson and William Leith hypothesized that the quakes were human-induced but noted that a lack of information prevented them from linking the events to gas production or other triggers. In 2009, a geothermal energy project in Basel, Switzerland, was canceled after development activities apparently led to a series of quakes of up to magnitude 3.4 that caused some $8 million in damage to surrounding properties.

In many of the wastewater injection cases documented so far, earthquakes followed within days or months of fluid injection starting. In contrast, the Oklahoma swarm happened years after injection began, similar to swarms at the Cogdell oil field in West Texas and the Fort St. John area of British Columbia.

The Wilzetta fault system remains under stress, the study's authors say, yet regulators continue to allow injection into nearby wells. Ideally, injection should be kept away from known faults and companies should be required to provide detailed records of how much fluid they are pumping underground and at what pressure,

said Keranen. The study authors also recommend sub-surface monitoring of fluid pressure for earthquake warning signs. Further research is needed but at a minimum, "there should be careful monitoring in regions where you have injection wells and protocols for stopping pumping even when small earthquakes are detected," said Abers. In a recent op-ed in the Albany (N.Y.) Times Union, Abers argued that New York should consider the risk of induced earthquakes from fluid injection in weighing whether to allow hydraulic fracturing to extract the state's shale gas reserves.

*The study was also coauthored by Elizabeth Cochran of the U.S. Geological Survey.*

*Katie M. Keranen, Heather M. Savage, Geoffrey A. Abers, and Elizabeth S. Cochran. Potentially induced earthquakes in Oklahoma, USA: Links between wastewater injection and the 2011 Mw 5.7 earthquake sequence. Geology, March 26, 2013 DOI: 10.1130/G33909.1*

***http://ars.to/10mNKXq***

## Fiber cables made of air move data at 99.7 percent the speed of light

***1.5 terabits per second, with only the laws of physics slowing it down.***

**by Jon Brodkin - Mar 27 2013, 6:55am TST**

Researchers say they have created fiber cables that can move data at 99.7 percent of the speed of light, all but eliminating the latency plaguing standard fiber technology. There are still data loss problems to be overcome before the cables could be used over long distances, but the research may be an important step toward incredibly low-latency data transmissions.

Although optic fibers transmit information using beams of light, that information doesn't actually go at "light speed." The speed of light, about 300,000 km/s, is the speed light travels in a vacuum. In a medium such as glass, it goes about 30 percent slower, a mere 200,000 km/s. "[L]ight propagates 31% slower in a silica glass fibre than in vacuum, thus compromising latency," notes a paper published Sunday in Nature Photonics, titled "Towards high-capacity fibre-optic communications at the speed of light in vacuum."

The research team from the University of Southampton in England solved this problem by taking the glass out of the glass fiber. This results in a "hollow-core photonic-bandgap fibre," which is made mostly of air yet still allows light to follow the path of the cable when it twists and turns.

The methods used by the researchers result in data loss of 3.5 dB/km, an impressively low number considering its incredibly low latency. However, that data loss is still too high for long-range communications. For now, these cables won't be used to wire up Internet Service Provider networks or for transatlantic cables.

The cable uses wide-bandwidth channels to send 37 streams of 40 gigabits per second each, with an aggregate transmission capacity of 1.48Tbps. Even with the current rate of data loss, the researchers say the cable technology is adequate for "short reach low-latency applications," such as future exaflop-speed supercomputers and "mega data centres." "For longer transmission distances, additional work is needed to further reduce surface scattering loss and to achieve the sub-0.2 dB km," the researchers wrote.

***UPDATE:*** *Although this wasn't described in the paper, one of the researchers told ExtremeTech that the cable's throughput actually goes up to 73.7Tbps, "using wave division multiplexing (WDM), combined with mode division multiplexing, to transmit three modes of 96 channels of 256Gbps."*

***http://www.eurekalert.org/pub_releases/2013-03/uoc-rsm032613.php***

## Researchers successfully map fountain of youth

***Researchers have for the first time mapped telomerase***

In collaboration with an international research team, University of Copenhagen researchers have for the first time mapped telomerase, an enzyme which has a kind of rejuvenating effect on normal cell ageing. The findings have just been published in Nature Genetics and are a step forward in the fight against cancer.

Mapping the cellular fountain of youth – telomerase. This is one of the results of a major research project involving more than 1,000 researchers worldwide, four years of hard work, DKK 55 million from the EU and blood samples from more than 200,000 people. This is the largest collaboration project ever to be conducted within cancer genetics.

Stig E. Bojesen, a researcher at the Faculty of Health and Medicial Sciences, University of Copenhagen, and staff specialist at the Department of Clinical Biochemistry, Copenhagen University Hospital, Herlev, has headed the efforts to map telomerase – an enzyme capable of creating new ends on cellular chromosomes, the so-called telomeres. In other words, a kind of cellular fountain of youth.

"We have discovered that differences in the telomeric gene are associated both with the risk of various cancers and with the length of the telomeres. The surprising finding was that the variants that caused the diseases were not the same as the ones which changed the length of the telomeres. This suggests that telomerase plays a far more complex role than previously assumed" says Stig E. Bojesen.

The mapping of telomerase is an important discovery, because telomerase is one of the very basic enzymes in cell biology. It relengthens the telomeres so that they get the same length as before embarking on cell division.
-The mapping of telomerase may, among other things, boost our knowledge of cancers and their treatment, and with the new findings the genetic correlation between cancer and telomere length has been thoroughly illustrated for the first time, says Stig E. Bojesen.

**Telomeres a cellular 'multi-ride ticket'**

The human body consists of 50,000,000,000,000 or fifty trillion cells, and each cell has 46 chromosomes which are the structures in the nucleus containing our hereditary material, the DNA. The ends of all chromosomes are protected by so-called telomeres. The telomeres serve to protect the chromosomes in much the same way as the plastic sheath on the end of a shoelace. But each time a cell divides, the telomeres become a little bit shorter and eventually end up being too short to protect the chromosomes.

Popularly speaking, each cell has a multi-ride ticket, and each time the cell divides, the telomeres (the chromosome ends) will use up one ride. Once there are no more rides left, the cell will not divide any more, and will, so to speak, retire. But some special cells in the body can activate telomerase, which again can elongate the telomeres.

Sex cells, or other stem cells which must be able to divide more than normal cells, have this feature. Unfortunately, cancer cells have discovered the trick, and it is known that they also produce telomerase and thus keep themselves artificially young. The telomerase gene therefore plays an important role in cancer biology, and it is precisely by identifying cancer genes that the researchers imagine that you can improve the identification rate and the treatment.

"A gene is like a country. As you map it, you can see what is going on in the various cities. One of the cities in what could be called Telomerase Land determines whether you develop breast cancer or ovarian cancer, while other parts of the gene determine the length of the telomeres. Mapping telomerase is therefore an important step towards being able to predict the risk of developing different cancers. In summary, our findings are very surprising and point in many directions. But as is the case with all good research, our work provides many answers but leaves even more questions" says Stig E. Bojesen.

**The international collaboration**

The large-scale COGS research collaboration has so far resulted in 14 articles which will be published simultaneously. Six of the articles will be published in the same issue of Nature Genetics and the remaining eight in other journals. All the articles from the many researchers involved in the project focus on the correlation between the environment, genetics and cancer, in particular breast cancer, ovarian cancer and prostate cancer.

***http://phys.org/news/2013-03-programmable.html***

## Making living matter programmable

***Thirty years ago, the future lay in programming computers. Today, it's programming cells.***

That was the message of panelists at an afternoon session yesterday (March 25) in Stanley Hall auditorium titled "Programming Life: the revolutionary potential of synthetic biology." Co-presented by UC Berkeley's Synthetic Biology Engineering Research Center (SynBERC) and Discover magazine, the panels brought together a dozen of synthetic biology's pioneers from academia and industry, in addition to ethicists focused on the societal impact of the technology.

Keynote speaker Juan Enriquez, a self-described "curiosity expert" and co-founder of the company Synthetic Genomics, compared the digital revolution spawned by thinking of information as a string of ones and zeros to the coming synthetic biology revolution, premised on thinking about life as a mix of interchangeable parts – genes and gene networks – that can be learned and manipulated like any language.

At the moment, this genetic manipulation, a natural outgrowth of genetic engineering, focuses on altering bacteria and yeast to produce products they wouldn't normally make, such as fuels or drugs. "To do with biology what you would do if you were designing a piece of software," according to moderator Corey Powell, editor at large of Discover, which plans to publish a story about the conference and post the video online.

UC Berkeley chemical engineer Jay Keasling has been a key player in developing the field of synthetic biology over the last decade. Enriquez introduced Keasling as someone "who in his spare time goes out and tries to build stuff that will cure malaria, and biofuels and the next generation of clean tech, all while mentoring students at this university and at the national labs and creating whole new fields of science."

Keasling, director of SynBERC, a UC Berkeley-led multi-institution collaboration that is laying the foundations for the field, expressed excitement about the newest development: the release next month by the pharmaceutical company sanofi aventis of a synthetic version of artemsinin, "the world's best antimalarial drug," he said.

Sparked by discoveries in Keasling's lab more than a decade ago, the drug is produced by engineered yeast and will be the first product from synthetic biology to reach the market. "There are roughly 300 to 500 million cases of malaria each year," he said. "Sanofi will initially produce about 100 million treatments, which will cover one-third to one-quarter of the need."

**Biofuels from yeast**

As CEO of the Joint BioEnergy Institute, Keasling is now focused on engineering microbes to turn "a billion tons of biomass that go unutilized in the U.S. on an annual basis … into fuel, producing roughly a third of the need in the U.S." But other advances are on the horizon, he said, such as engineering new materials and engineering "green" replacements for all the products now made from petroleum. "Some of these have the potential to significantly reduce our carbon footprint, by say, 80 percent," he said.

Virginia Ursin, Technology Prospecting Lead and Science Fellow at Monsanto Corp., noted that industry sees synthetic biology's triumphs as being 10-20 years down the road, but anticipated, for example, producing enzymes used in manufacturing or even engineering microbes that live on plants to improve plant growth.

"Engineering (microbes) to increase their impact on (plant) health or protection against disease is probably going to be one of the nearer term impacts of synthetic biology on agriculture," she said. Ultimately, she said, the field could have a revolutionary impact on agriculture similar to the green revolution sparked by the development of chemical fertilizers.

But the implications of being able to engineer cells go deeper, according to Enriquez.

"This isn't just about economic growth, this is also about where we are going as a human species," he said. Humans will no longer merely adapt to or adopt the environment, but "begin to understand how life is written, how life is coded, how life is copied, and how you can rewrite life."

**Scientists' moral choices**

Directly guiding "the evolution of microbes, bacteria, plants, animals and even ourselves," as Enriquez put it, sounds like science fiction. George Church, a biologist at Harvard Medical School, suggested that we might want to bring back extinct animals, such as the mammoth to help restore Arctic permafrost disintegrating under the impact of global warming.

In response, ethicist Laurie Zoloth of Northwestern University urged caution in exploiting the technology of synthetic biology. "You could change the world, and you have a powerful technology," she said. "I am more interested in what this technology makes of the women and men doing it. What sorts of interior moral choices they need to be making and how you create scientists who aren't only good at all these technical skills but very good at asking and thinking seriously about ethical and moral questions and coming to terms with the implications of their work." Given the current crises of climate change and ecological change, she added, "frankly, without this work, I don't think we have such great answers for (them)."

Church acknowledged that "we have an obligation to do it right. But because our environment, our world is changing, the decision to do nothing is a gigantic risk. The decision to do a particular new thing is a risk. We have to get better at risk assessment and safety engineering."

Drew Endy, a bioengineering professor at Stanford, summed up his hopes for synthetic biology. "What I would like to imagine as a longer-term encompassing vision is that humanity figured out how to reinvent the manufacturing of the things we need so that we can do it in partnership with nature; not to replace nature, but to dance better with it in sustaining what it means to be a flourishing human civilization."

*Provided by University of California - Berkeley*

***http://www.bbc.co.uk/news/science-environment-21958547***

## Neonicotinoid pesticides 'damage brains of bees'

***Commonly used pesticides are damaging honey bee brains, studies suggest.***

**By Rebecca Morelle Science reporter, BBC World Service**

Scientists have found that two types of chemicals called neonicotinoids and coumaphos are interfering with the insect's ability to learn and remember. Experiments revealed that exposure was also lowering brain activity, especially when the two pesticides were used in combination.

The research is detailed in two papers in Nature Communications and the Journal of Experimental Biology.

But a company that makes the substances said laboratory-based studies did not always apply to bees in the wild. And another report, published by the Defra's Food and Environment Research Agency (Fera), concluded that there was no link between bee health and exposure to neonicotinoids. The government agency carried out a study looking at bumblebees living on the edges of fields treated with the chemicals.

**Falling numbers**

Honey bees around the world are facing an uncertain future. They have been hit with a host of diseases, losses of habitat, and in the US the mysterious Colony Collapse Disorder has caused numbers to plummet.

Now researchers are asking whether pesticides are also playing a role in their decline.
To investigate, scientists looked at two common pesticides: neonicotinoids, which are used to control pests on oil seed rape and other crops, and a group of organophosphate chemicals called coumaphos, which are used to kill the Varroa mite, a parasite that attacks the honey bee.
Neonicotinoids are used more commonly in Europe, while coumaphos are more often employed in the United States. Work carried out by the University of Dundee, in Scotland, revealed that if the pesticides were applied directly to the brains of the pollinators, they caused a loss of brain activity.
Dr Christopher Connolly said: "We found neonicotinoids cause an immediate hyper-activation - so an epileptic type activity - this was proceeded by neuronal inactivation, where the brain goes quiet and cannot communicate any more. The same effects occur when we used organophosphates. "And if we used them together, the effect was additive, so they added to the toxicity: the effect was greater when both were present."
Another series of laboratory-based experiments, carried out at Newcastle University, examined the behaviour of the bees.
The researchers there found that bees exposed to both pesticides were unable to learn and then remember floral smells associated with a sweet nectar reward - a skill that is essential for bees in search of food.
Dr Sally Williamson said: "It would imply that the bees are able to forage less effectively, they are less able to find and learn and remember and then communicate to their hive mates what the good sources of pollen and nectar are."

**'No threat'**

She said that companies that are manufacturing the pesticides should take these findings into account when considering the safety of the chemicals. She explained: "At the moment, the initial tests for bee toxicity are giving the bees an acute dose and then watching them to see if they die.
"But because bees do these complex learning tasks, they are very social animals and they have a complex behavioural repertoire, they don't need to be killed outright in order not to be affected."
The European Commission recently called for a temporary moratorium on the use of neonicotinoids after a report by the European Food Safety Authority concluded that they posed a high acute risk to pollinators. But 14 out of the 27 EU nations - including the UK and Germany - opposed the ban, and the proposal has now been delayed.
Ian Boyd, chief scientist at Defra, said: "Decisions on the use of neonicotinoids must be based on sound scientific evidence."
He said that the results of the Fera bumblebee study suggested that the extent of the impact might not be as high as some studies had suggested - and called for "further data based on more realistic field trials is required".
Dr Julian Little, communications and government affairs manager at Bayer Crop Science Limited, which makes some of the pesticides, said the findings of laboratory-based studies should not be automatically extrapolated to the field. "If you take an insecticide and you give it directly to an insect, I can guarantee that you will have an effect - I am not at all surprised that this is what you will see," he explained. "What is really important is seeing what happens in real situations - in real fields, in real bee colonies, in real bee hives, with real bee keepers."

***http://www.sciencedaily.com/releases/2013/03/130327132441.ht***

## Scientists Discover Driving Force Behind Prostate Cancer

***Research reveals the existence of a cancer inducing DNA re-alignment in stem cells from human prostate cancers***

Scientists at the University of York have discovered the driving force behind the development of prostate cancer. Their research, published in Nature Communications today and funded by the charity Yorkshire Cancer Research, reveals the existence of a cancer inducing DNA re-alignment in stem cells taken from human prostate cancers.
This opens the way to the development of drugs that target the stem cells, leading to more effective therapies that work against the root cause of the disease.
Professor Norman Maitland, Director of the YCR Cancer Research Unit, and his team in the University's Department of Biology were the first to isolate prostate cancer stem cells in 2005. While other cancer cells can be killed by current therapies, stem cells are able to evade their effects, resulting in cancer recurrence. The team has since been exploring the exact molecular properties that allow these cells to spread, survive and resist aggressive treatments such as radiation and chemotherapy.
Professor Maitland said: "This discovery marks a fundamental shift in our understanding of how solid cancers start. It is believed that 'root' cancer cells arise from healthy stem cells going wrong – for example certain controls can be turned off which allow the cells to keep growing and invade surrounding tissue.

"In blood cancers such as leukaemia, DNA is rearranged during an event known as chromosomal translocation, which results in a mutant protein that drives cancer progression. Although similar rearrangements have recently been discovered in solid cancers, until now, they have not been considered as stem cell functions. Our work has challenged this idea."
Professor Maitland's team has found these genetic accidents in prostate cancer stem cells and has shown that they result in a specific cancer-associated gene within the cells called ERG being inappropriately activated. It is believed that this activation triggers the stem cells to renew more often.
Professor Maitland continued: "The cells become selfish by surviving outside normal controls that exist in the prostate and thrive at the expense of their neighbours, ensuring that the genetic accident becomes permanent and passed from generation to generation. This process appears to be essential for the initiation of prostate cancer."
Yorkshire Cancer Research funded a £2.15m five year programme at the YCR Cancer Research Unit in August 2011 to allow scientists to continue their internationally-award winning research into prostate cancer.
Kathryn Scott, Head of Research Funding at the charity, said: "This exciting discovery is another step forward in our understanding of how prostate cancer begins. Professor Maitland has detected one of the earliest possible changes in the development of prostate cancer. The findings mean that new therapies can now be developed which specifically target the protein identified, killing the stem cells that remain after chemotherapy while leaving healthy cells untouched."
*Euan S. Polson, John L. Lewis, Hamza Celik, Vincent M. Mann, Michael J. Stower, Matthew S. Simms, Greta Rodrigues, Anne T. Collins, Norman J. Maitland. Monoallelic expression of TMPRSS2/ERG in prostate cancer stem cells. Nature Communications, 2013; 4: 1623 DOI: 10.1038/ncomms2627*

***http://www.sciencedaily.com/releases/2013/03/130327132552.htm***

## Genetic 'Spelling Mistakes' That Increase Risk of Common Cancers Determined

***More than 80 genetic 'spelling mistakes' that can increase the risk of breast, prostate and ovarian cancer have been found in a large, international research study within the framework of the EU network COGS.***

For the first time, the researchers also have a relatively clear picture of the total number of genetic alterations that can be linked to these cancers. Ultimately the researchers hope to be able to calculate the individual risk of cancer, to better understand how these cancers develop and to be able to generate new treatments.
The main findings are published in five articles in a special issue on genetic risk factors for cancer in the scientific journal Nature Genetics. The articles originate from COGS (Collaborative Oncological Gene-environment Study), an EU-based consortium where more than 160 research groups from all over the world are included. In the five COGS studies 100,000 patients with breast, ovarian or prostate cancer and 100,000 healthy individuals from the general population were included.
The scientists performed genetic analyses on all study participants. The composition of the nitrogen bases A, G, C and T was studied on 200,000 selected sections of the DNA strand. When cancer patients had significantly different compositions compared to healthy control subjects, the differences were considered to be relevant to risk of disease. The alterations can be described as a genetic 'spelling mistake', where A, G, C or T have been replaced with another letter. This spelling mistake is called Single Nucleotide Polymorphism (SNP) -- pronounced 'snip'.
For breast cancer the researchers found 49 genetic typos or SNPs, which is more than double the number previously found. In the case of prostate cancer, researchers have discovered another 26 deviations, which means that a total number of 78 SNPs may be linked to the disease. For ovarian cancer 8 new relevant SNPs were found.
"An equally important finding is that we identified how many additional SNPs that could influence the risk of breast cancer and prostate cancer, respectively. For breast cancer the number is 1,000 and for prostate cancer 2,000," says Per Hall, Professor at Karolinska Institutet and the coordinator of the COGS consortium. "We also have a picture of where in the genome we should look in future studies.
SNPs are part of our natural heritage, we all have them. How it affects the individual depends on where on the DNA strand the genetic deviation is found. The researchers now hope to be able to evaluate the importance of the identified deviations, so that it will be possible to more clearly predict which individuals are at high risk of developing one of these cancers.
"We're now on the verge of being able to use our knowledge to develop tests that could complement breast cancer screening and take us a step closer to having an effective prostate cancer screening programme," says Professor Doug Easton of the University of Cambridge, UK, who has led several of the presented studies.
At the same time as these five articles are published in Nature Genetics, the Nature Publishing Group publishes another two articles on studies emanating from the COGS collaboration in Nature Communications. An

additional number of COGS articles will be published simultaneously in other journals. The studies are financed by partly different funders; however the COGS project is mainly funded by the European Commission 7th Framework Programme. Other financial contributors to the COGS project are the Märit and Hans Rausing Initiative against Breast Cancer, the Swedish Research Council, Cancer Research UK and the Cancer Risk Prediction Center (CRisP).

"COGS is the largest genotyping project in the world targeting identification of genetic alterations that influence the risk of common cancers. The collaborative efforts have been tremendous and key to success," says COGS coordinator Per Hall.

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***http://www.scientificamerican.com/article.cfm?id=tiny-primitive-mammal-unearthed-japan***

# Tiny Primitive Mammal Unearthed in Japan

***The discovery of the jaw of a 112-million-year-old mammal from the early Cretaceous Period suggests that small creatures were already evolving quickly***

**By Tia Ghose and LiveScience**

Paleontologists in Japan have unearthed the jaw of a primitive mammal from the early Cretaceous period.

The pint-size creature, named Sasayamamylos kawaii for the geologic formation in Japan where it was found, is about 112 million years old and belongs to an ancient clade known as Eutherian mammals, which gave rise to all placental mammals. (A clade is a group of animals that share uniquely evolved features and therefore a common ancestry.)

The jaw sports pointy, sharp teeth and molars in a proportion similar to that found in modern mammals, said paleontologist Brian Davis of Missouri Southern State University, who was not involved in the study.

"This little critter, Sasayamamylos, is the oldest Eutherian mammal to demonstrate what paleontologists consider the modern dental formula in placental mammals," Davis told LiveScience.

The new mammal fossil, described today (March 26) in the journal Proceedings of the Royal Society B, suggests that these primitive creatures were already evolving quickly, with diverse traits emerging, at this point in the Cretaceous Era, he added.

***Eutherian mammal jaw Eutherian mammal jaw In 2007, scientists discovered a Eutherian mammal jaw that is about 112 million years old. The jaw shows that these primitive mammals had already started diversifying key characteristics at that time.*** **Image: Nao Kusuhashi**

## Tiny Creatures

Between 145 million and 66 million years ago, most mammals were tiny creatures that scampered underfoot as giant dinosaurs roamed the Earth. Scientists recently proposed that the first mammalian Eve, the mother to all placental mammals, lived about 65 million years ago, when dinosaurs went extinct. The first true mammal likely emerged at least 100 million years before that. But because the fossil record is spotty, determining exactly when mammals evolved their specific traits has been murkier.

Amateur fossil-hunters were searching through sediments in Hyogo, Japan in 2007 when they unearthed the skeletal fragments of an ancient mammalian jaw. They turned it over to a local museum, said study co-author Nao Kusuhashi, a paleontologist at Ehime University in Japan.

The jaw contained four sharp, pointy teeth known as pre-molars and three molars with complex ridges. That same pattern in the number of each type of tooth is found in placental mammals to this day, whereas earlier mammals have more of the sharp, pointy teeth. The teeth probably allowed Sasayamamylos to poke through the hard exoskeletons of beetles or other insects, Davis told LiveScience.

In general, molars probably allowed these primitive mammals to chew their food well, extracting as much energy as possible from it, Davis said.

http://bit.ly/ZuZnuf

# First Love Child of Human, Neanderthal Found

***The skeletal remains of an individual living in northern Italy 40,000-30,000 years ago are believed to be that of a human/Neanderthal hybrid, according to a paper in PLoS ONE***

**Mar 27, 2013 05:00 PM ET // by Jennifer Viegas**

If further analysis proves the theory correct, the remains belonged to the first known such hybrid, providing direct evidence that humans and Neanderthals interbred. Prior genetic research determined the DNA of people with European and Asian ancestry is 1 to 4 percent Neanderthal. The present study focuses on the individual's jaw, which was unearthed at a rock-shelter called Riparo di Mezzena in the Monti Lessini region of Italy. Both Neanderthals and modern humans inhabited Europe at the time.

"From the morphology of the lower jaw, the face of the Mezzena individual would have looked somehow intermediate between classic Neanderthals, who had a rather receding lower jaw (no chin), and the modern humans, who present a projecting lower jaw with a strongly developed chin," co-author Silvana Condemi, an anthropologist, told Discovery News.

Condemi is the CNRS research director at the University of Ai-Marseille. She and her colleagues studied the remains via DNA analysis and 3D imaging. They then compared those results with the same features from Homo sapiens. The genetic analysis shows that the individual's mitochondrial DNA is Neanderthal. Since this DNA is transmitted from a mother to her child, the researchers conclude that it was a "female Neanderthal who mated with male Homo sapiens."

By the time modern humans arrived in the area, the Neanderthals had already established their own culture, Mousterian, which lasted some 200,000 years. Numerous flint tools, such as axes and spear points, have been associated with the Mousterian. The artifacts are typically found in rock shelters, such as the Riparo di Mezzena, and caves throughout Europe.

The researchers found that, although the hybridization between the two hominid species likely took place, the Neanderthals continued to uphold their own cultural traditions. That's an intriguing clue, because it suggests that the two populations did not simply meet, mate and merge into a single group.

As Condemi and her colleagues wrote, the mandible supports the theory of "a slow process of replacement of Neanderthals by the invading modern human populations, as well as additional evidence of the upholding of the Neanderthals' cultural identity."

Prior fossil finds indicate that modern humans were living in a southern Italy cave as early as 45,000 years ago. Modern humans and Neanderthals therefore lived in roughly the same regions for thousands of years, but the new human arrivals, from the Neanderthal perspective, might not have been welcome, and for good reason. The research team hints that the modern humans may have raped female Neanderthals, bringing to mind modern cases of "ethnic cleansing."

Ian Tattersall is one of the world's leading experts on Neanderthals and the human fossil record. He is a paleoanthropologist and a curator emeritus at the American Museum of Natural History. Tattersall told Discovery News that the hypothesis, presented in the new paper, "is very intriguing and one that invites more research." Neanderthal culture and purebred Neanderthals all died out 35,000-30,000 years ago.

http://bit.ly/10peiHs

# Vaccine promises to cull foot and mouth slaughter

***Foot and mouth disease could be consigned to history, thanks to a new vaccine***

**Updated 16:29 28 March 2013 by Andy Coghlan**

Smouldering pyres of cattle sacrificed to halt foot and mouth disease could be consigned to history, thanks to a new vaccine. Crucially, it is the first that will allow vets to distinguish between animals have been vaccinated and those that have been infected by the virus.

That means it could overcome a key objection to the use of vaccines to deal with outbreaks of the disease.

In 2001, during a foot and mouth disease epidemic, the UK government was given the option of vaccinating cattle to contain outbreaks and prevent the virus from spreading further. It rejected the vaccine, choosing instead to slaughter 6 million animals at a cost of £8 billion.

They chose this option because the only vaccine available at the time was a whole, deactivated version of the foot and mouth virus. Cattle react to such vaccines by producing the same spectrum of protective antibodies as they would if they were infected by the live virus. In other words, so long as the virus is dormant, vaccinated cattle are impossible to distinguish from infected ones.

That's a big problem for countries that export cattle. They can only do so if they are declared free of foot and mouth, but to testers, a vaccinated herd looks infected. Now, Bryan Charleston of the Pirbright Institute in

Woking, UK, David Stuart of Diamond Light Source in Didcot, UK, and their colleagues have developed a synthetic vaccine that produces a different antibody signature in cattle.

**Core reasons**

The new vaccine consists of the inanimate outer shell of the virus, gutted of the genetic core that allows the live virus to infect cells, multiply and spread. To make it, Charleston inserted the genes coding for the outer shell and the enzymes that assemble it into moth cells. The cells pumped out empty viral shells.

Vaccinated herds only produce antibodies against this shell, whereas genuinely infected animals w uld produce antibodies against the core as well.

Rich and poor countries alike could benefit from the new vaccine. The old vaccine is in short supply and can only be made in secure labs – mostly in Europe and the US – and must be kept refrigerated to be functional. The synthetic one can be made without these precautions and stored at temperatures of up to almost 60 °C.

Charleston and his colleagues tested the vaccine on eight calves, which were protected from infection as efficiently as if they had been given the existing vaccine. "It induced protective antibodies that neutralise the virus for up to nine months," says Charleston.

The vaccine only protects against the "A" serotype of the virus, one of seven that exists worldwide, but new versions are almost ready to protect against two other serotypes. This includes the "O" strain, which accounts for 80 per cent of the world's outbreaks. It was to blame for the 2001 UK outbreak, and massive epidemics three years ago in Japan and South Korea.

The vaccine against each serotype will need to be tested successfully in herds of at least 17 animals to win European approval, and Charleston warns it could be seven years before the vaccines are available.

"Once available, vaccines of this type would have clear advantages over current technology as a possible option to help control the disease should we ever have another outbreak," says Nigel Gibbens, the UK's chief veterinary officer. *Journal reference: PLoS Pathogens, DOI: 10.1371/journal.ppat.1003255*

***http://www.eurekalert.org/pub_releases/2013-03/mcoq-smm032813.php***

## Surgical menopause may prime brain for stroke, Alzheimer's

***Women who abruptly and prematurely lose estrogen from surgical menopause have a two-fold increase in cognitive decline and dementia.***

"This is what the clinical studies indicate and our animal studies looking at the underlying mechanisms back this up," said Brann, corresponding author of the study in the journal Brain. "We wanted to find out why that is occurring. We suspect it's due to the premature loss of estrogen."

In an effort to mimic what occurs in women, Brann and his colleagues looked at rats 10 weeks after removal of their estrogen-producing ovaries that were either immediately started on low-dose estrogen therapy, started therapy 10 weeks later or never given estrogen.

When the researchers caused a stroke-like event in the brain's hippocampus, a center of learning and memory, they found the rodents treated late or not at all experienced more brain damage, specifically to a region of the hippocampus called CA3 that is normally stroke-resistant. To make matters worse, untreated or late-treated rats also began an abnormal, robust production of Alzheimer's disease-related proteins in the CA3 region, even becoming hypersensitive to one of the most toxic of the beta amyloid proteins that are a hallmark of Alzheimer's.

Both problems appear associated with the increased production of free radicals in the brain. In fact, when the researchers blocked the excessive production, heightened stroke sensitivity and brain cell death in the CA3 region were reduced. Interestingly the brain's increased sensitivity to stressors such as inadequate oxygen was gender specific, Brann said. Removing testes in male rats, didn't affect stroke size or damage.

Although exactly how it works is unknown, estrogen appears to help protect younger females from problems such as stroke and heart attack. Their risks of the maladies increase after menopause to about the same as males. Follow up studies are needed to see if estrogen therapy also reduces sensitivity to the beta amyloid protein in the CA3 region, as they expect, Brann noted.

Brann earlier showed that prolonged estrogen deprivation in aging rats dramatically reduces the number of brain receptors for the hormone as well as its ability to prevent strokes. Damage was forestalled if estrogen replacement was started shortly after hormone levels drop, according to the 2011 study in the journal Proceedings of the National Academy of Sciences.

The surprising results of the much-publicized Women's Health Initiative – a 12-year study of 161,808 women ages 50-79 – found hormone therapy generally increased rather than decreased stroke risk as well as other health problems. Critics said one problem with the study was that many of the women, like Brann's aged rats, had gone years without hormone replacement, bolstering the case that timing is everything.

*The research was supported by the National Institute of Neurological Disorders and Stroke, an American Heart Association Scientist Development grant and a National Natural Science Foundation grant.*

***http://phys.org/news/2013-03-organisation-trumps-size-primate-brain.html***

## Organisation trumps size in primate brain evolution

***The evolution of anthropoid primates, including monkeys, apes and humans, over the past 40 million years was largely driven by brain reorganization, and not brain size, according to new research from UCL.***

Phys.org - The study, which is published in Proceedings of the Royal Society B, found that around three quarters of differences between the brains of species of monkeys and apes are due to internal reorganization that is independent of size, dispelling the idea that variation in size is the primary factor characterising anthropoid primate brain evolution.

Dr Jeroen Smaers (UCL Anthropology and UCL Genetics, Evolution & Environment), lead author of the study said: "The brain is central to how animals adapt and modify their behaviour in a changing environment. "What we've found is that in relation to the brain, species differences are mainly explained by how the brain is organized and wired internally, not how large the brain is. This suggests that brain reorganization, not size, may have been the principal force driving brain evolution."

To trace the evolutionary history of the anthropoid brain, the team collected the overall size of the brain and its internal structures for 17 anthropoid primate species. They then mapped and compared the evolutionary changes in these structures to get an insight into how the brains of each of the species specialised while adapting to their respective environments.

Dr Christophe Soligo (UCL Anthropology) said: "Changes in the overall size of the brain have often been proposed as the main solution to producing a more complex brain that supports more complex behaviour. "A bigger brain is, however, energetically very expensive and may not always be an option. Sometimes animals do not have the choice to increase their energetic input but are still faced with a pressure to adapt. This is when reorganisation may come into play."

By analysing the overall and relative size of 20 brain structures, the team also found that the prefrontal cortex – a brain area that synthesizes information processed in other parts of the brain to produce complex judgements and behaviours - plays the biggest role in explaining the evolutionary changes in anthropoid primate brain organisation.

"We've known for a while that brain reorganization is important. But we had no idea it would explain as much variation between species as it does. We've also been further able to characterize evolutionary specializations allowing us to pinpoint what makes certain species special - such as motor learning in great apes and humans - and how far back in time specific evolutionary lineages started to evolve differently from other lineages," added Dr Soligo.

Dr Smaers said: "As the principal base of behaviour, the brain lies at the heart of the adaptive profile of any animal. By mapping detailed patterns of how the brain evolved, we can increase our understanding of the selective pressures that have shaped a species' natural history. This not only adds to our understanding of the natural world, but also helps us understand the evolutionary history of brain systems that underlie human brain disorders." *More information: rspb.royalsocietypublishing.org/content/280/1759/20130269.full*

***http://www.wired.com/wiredscience/2013/03/neutral-biodiversity/***

## Something Other Than Adaptation Could Be Driving Evolution

**What explains the incredible variety of life on Earth? It seems obvious. Evolution, of course! But perhaps not the evolution most people grew up with.**

**By Brandon Keim**

Some ecologists say the theory needs an update. They've proposed a new dynamic driving the emergence of new species, one that doesn't involve adaptations or survival of the fittest.

Give evolution enough time and space, they say, and new species can just happen. Speciation might not only be an evolutionary consequence of fitness differences and natural selection, but a property intrinsic to evolution, just as all matter has gravity.

"Our work shows that evolution wants to be diverse," said Yaneer Bar-Yam, president of the New England Complex Systems Institute. "It's enough for organisms to be spread out in space and time."

In a March 13 Proceedings of the National Academy of Sciences paper, Bar-Yam and his co-authors, Brazilian ecologists Ayana Martins at the University of Sao Paulo and Marcus Aguiar at the University of Campinas, modeled the evolution of greenish warblers living around the Tibetan plateau.

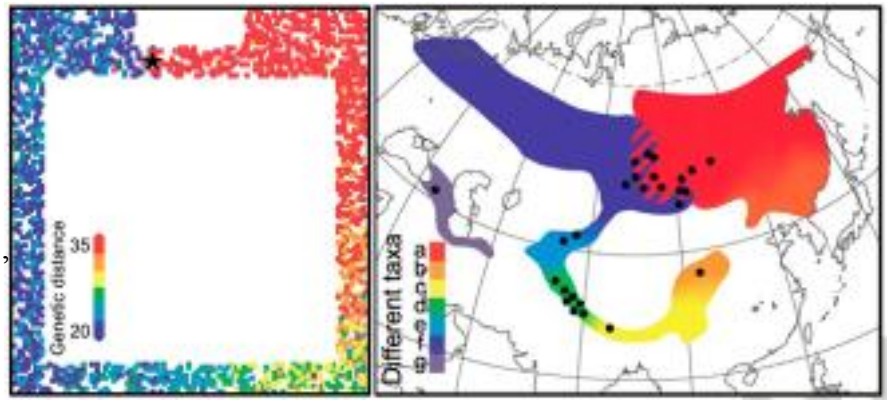
The warblers are what's known as a ring species, a rare phenomenon that occurs when species inhabit a horseshoe-shaped range. Genes flow around the ring, passing between neighboring populations — yet at the ring's tips, the animals no longer interbreed with one another.

By the usual standards, these end populations have become new species. According to the researchers' model of the process, no special adaptations or differences in reproductive fitness are needed to explain — or at least to computationally replicate — the greenish warblers' divergence.

**'An alternative hypothesis to adaptation and selection of new species.'**

"This sounds kind of crazy, right? We normally think of species as being adapted for particular functions. They have their own role to play in a community. That's the standard wisdom," said theoretical ecologist James O'Dwyer of the Santa Fe Institute, who was not involved in the study.

Instead, over 2,000 modeled generations, a time frame that fits with the 10,000 years that greenish warblers have ringed the Tibetan plateau's slopes since their exposure by retreating glaciers, random genetic mutations drifted through the birds' populations, ultimately clustering in diversity patterns resembling what's seen in reality.




***A computational model of greenish warbler evolution (left) fits real-world patterns of the species (right). Color corresponds to degrees of genetic difference.*** **Image: Martins et al./PNAS**

Adaptation and natural selection certainly played a part in the warblers' evolution, said Bar-Yam, but they weren't necessarily the driving forces. And though geography is involved, it's very different from the population-isolating physical separation created by mountain ranges or islands. "The plateau plays an important role in the formation of the ring species, but it does not block gene flow," said Aguiar. "No barriers and no specific selection processes are required." Rather than adaptation, distance is the driver.

That notion falls under the umbrella of neutral biodiversity theory, a dry-sounding name for a dramatic challenge to the notion that adaptation is biological diversity's wellspring. First articulated by University of California, Los Angeles ecologist Stephen Hubbell, who in 2001 published The Unified Neutral Theory of Biodiversity and Biogeography, it's a challenge occasioned by the surprising difficulty of explaining biodiversity, or why life is arranged the way it is.

Neutral biodiversity doesn't reject the usual evolutionary drivers of adaptation and geographic isolation, which are clearly at work in shaping species traits and generating diversity. But these drivers don't seem to explain many big-picture patterns. It's not just ring species that are perplexing. Tropical forests, which originally inspired Hubbell's theory, seemingly have far more species than there are niches to adaptively inhabit. Common patterns of species distribution also occur in disparate places, such as rain forests and coral reefs. The usual evolutionary models didn't fit these phenomena.

Some under-appreciated forces seemed to be operating, which Hubbell identified as neutral genetic drift: the flow, at landscape-level scales, of random genetic variations that emerge in individuals and spread through populations, but are 'neutral,' having no biological function.

That most mutations are neutral isn't a new idea. It was first proposed in the late 1960s by Japanese geneticist Mootoo Kimura, and is an established dynamic in population genetics. That it might actually drive diversity on its own, though, accounting for substantial differences between species, was new.

How exactly this might work and how important it could be has been hotly debated ever since, at least in ecological circles. Some ecologists reject the idea altogether. Other researchers, including Bar-Yam's group, have built on Hubbell's original ideas.

Their work "offers an alternative hypothesis to adaptation and selection of new species," said O'Dwyer, but he warned that it's hard to tell whether neutral processes really occur. Computational models of neutral biodiversity often seem to predict real-world patterns, as with the greenish warblers, but that doesn't mean they're right.

Datasets necessary to test neutral explanations need to span hundreds if not thousands of years, and should encompass not just a few species but entire ecosystems, said O'Dwyer. He thinks some combination of neutral and non-neutral processes likely shape biodiversity, and teasing their contributions apart will be difficult.

Ecologist Rampal Etienne of the University of Groningen, whose own research suggests that sexual reproduction makes evolution speed up, echoed O'Dwyer's point. "The major question is what data will be able to distinguish neutral from non-neutral explanations," said Etienne, who cautioned against jumping to conclusions with Bar-Yam's model.

Like any model, it's based on assumptions and only imperfectly imitates reality, he said. Its more fundamental value, as with other work on neutral biodiversity, is that it critically examines whether adaptation really explains the natural world's richness. In other words, the theory of evolution is still evolving.
*Citation: "Evolution and stability of ring species." By Ayana B. Martins, Marcus A. M. de Aguiar and Yaneer Bar-Yam. Proceedings of the National Academy of Sciences, March 11, 2013.*
*Update 3/28: Text modified to emphasize that neutral biodiversity theory does not exclude 'traditional' evolutionary mechanisms, but would be an addition to them.*

***http://www.eurekalert.org/pub_releases/2013-03/sri-nva032813.php***

## New vaccine-design approach targets HIV and other fast-mutating viruses

***Technique for vaccine design that could be particularly useful against HIV and other fast-changing viruses***

LA JOLLA, CA - A team led by scientists from The Scripps Research Institute (TSRI) and the International AIDS Vaccine Initiative (IAVI) has unveiled a new technique for vaccine design that could be particularly useful against HIV and other fast-changing viruses.
The report, which appears March 28, 2013, in Science Express, the early online edition of the journal Science, offers a step toward solving what has been one of the central problems of modern vaccine design: how to stimulate the immune system to produce the right kind of antibody response to protect against a wide range of viral strains. The researchers demonstrated their new technique by engineering an immunogen (substance that induces immunity) that has promise to reliably initiate an otherwise rare response effective against many types of HIV.
"We're hoping to test this immunogen soon in mice engineered to produce human antibodies, and eventually in humans," said team leader William R. Schief, who is an associate professor of immunology and member of the IAVI Neutralizing Antibody Center at TSRI.

**Seeking a Better Way**

For highly variable viruses such as HIV and influenza, vaccine researchers want to elicit antibodies that protect against most or all viral strains—not just a few strains, as seasonal flu vaccines currently on the market.
Vaccine researchers have identified several of these broadly neutralizing antibodies from long-term HIV-positive survivors, harvesting antibody-producing B cells from blood samples and then sifting through them to identify those that produce antibodies capable of neutralizing multiple strains of HIV. Such broadly neutralizing antibodies typically work by blocking crucial functional sites on a virus that are conserved among different strains despite high mutation elsewhere.
However, even with these powerful broadly neutralizing antibodies in hand, scientists need to find a way to elicit their production in the body through a vaccine. "For example, to elicit broadly neutralizing antibodies called VRC01-class antibodies that neutralize 90 percent of known HIV strains, you could try using the HIV envelope protein as your immunogen," said Schief, "but you run into the problem that the envelope protein doesn't bind with any detectable affinity to the B cells needed to launch a broadly neutralizing antibody response."
To reliably initiate that VRC01-class antibody response, Schief and his colleagues therefore sought to develop a new method for designing vaccine immunogens.

**From Weak to Strong**

Joseph Jardine, a TSRI graduate student in the Schief laboratory, evaluated the genes of VRC01-producing B cells in order to deduce the identities of the less mature B cells—known as germline B cells—from which they originate. Germline B cells are major targets of modern viral vaccines, because it is the initial stimulation of these B cells and their antibodies that leads to a long-term antibody response.
In response to vaccination, germline B cells could, in principle, mature into the desired VRC01-producing B cells—but natural HIV proteins fail to bind or stimulate these germline B cells so they cannot get the process started. The team thus set out to design an artificial immunogen that would be successful at achieving this.
Jardine used a protein modeling software suite called Rosetta to improve the binding of VRC01 germline B cell antibodies to HIV's envelope protein. "We asked Rosetta to look for mutations on the side of the HIV envelope protein that would help it bind tightly to our germline antibodies," he said.
Rosetta identified dozens of mutations that could help improve binding to germline antibodies. Jardine then generated libraries that contained all possible combinations of beneficial mutations, resulting in millions of mutants, and screened them using techniques called yeast surface display and FACS. This combination of computational prediction and directed evolution successfully produced a few mutant envelope proteins with high affinity for germline VRC01-class antibodies.
Jardine then focused on making a minimal immunogen—much smaller than HIV envelope—and so continued development using the "engineered outer domain (eOD)" previously developed by Po-Ssu Huang in the Schief

lab while Schief was at the University of Washington. Several iterative rounds of design and selection using a panel of germline antibodies produced a final, optimized immunogen—a construct they called eOD-GT6.

**A Closer Look**

To get a better look at eOD-GT6 and its interaction with germline antibodies, the team turned to the laboratory of Ian A. Wilson, chair of the Department of Integrative Structural and Computational Biology and a member of the IAVI Neutralizing Antibody Center at TSRI.

Jean-Philippe Julien, a senior research associate in the Wilson laboratory, determined the 3D atomic structure of the designed immunogen using X-ray crystallography—and, in an unusual feat, also determined the crystal structure of a germline VRC01 antibody, plus the structure of the immunogen and antibody bound together.

"We wanted to know whether eOD-GT6 looked the way we anticipated and whether it bound to the antibody in the way that we predicted—and in both cases the answer was 'yes'," said Julien. "We also were able to identify the key mutations that conferred its reactivity with germline VRC01 antibodies."

**Mimicking a Virus**

Vaccine researchers know that such an immunogen typically does better at stimulating an antibody response when it is presented not as a single copy but in a closely spaced cluster of multiple copies, and with only its antibody-binding end exposed. "We wanted it to look like a virus," said Sergey Menis, a visiting graduate student in the Schief laboratory.

Menis therefore devised a tiny virus-mimicking particle made from 60 copies of an obscure bacterial enzyme and coated it with 60 copies of eOD-GT6. The particle worked well at activating VRC01 germline B cells and even mature B cells in the lab dish, whereas single-copy eOD-GT6 did not.

"Essentially it's a self-assembling nanoparticle that presents the immunogen in a properly oriented way," Menis said. "We're hoping that this approach can be used not just for an HIV vaccine but for many other vaccines, too."

The next step for the eOD-GT6 immunogen project, said Schief, is to test its ability to stimulate an antibody response in lab animals that are themselves engineered to produce human germline antibodies. The difficulty with testing immunogens that target human germline antibodies is that animals typically used for vaccine testing cannot make those same antibodies. So the team is collaborating with other researchers who are engineering mice to produce human germline antibodies. After that, he hopes to learn how to drive the response, from the activation of the germline B cells all the way to the production of mature, broadly neutralizing VRC01-class antibodies, using a series of designed immunogens.

Schief also hopes they will be able to test their germline-targeting approach in humans sooner rather than later, noting "it will be really important to find out if this works in a human being."

*The first authors of the paper, "Rational HIV immunogen design to target specific germline B cell receptors," were Jardine, Julien and Menis. Co-authors were Takayuki Ota and Devin Sok of the Nemazee and Burton laboratories at TSRI, respectively; Travis Nieusma of the Ward laboratory at TSRI; John Mathison of the Ulevitch laboratory at TSRI; Oleksandr Kalyuzhniy and Skye MacPherson, researchers in the Schief laboratory from IAVI and TSRI, respectively; Po-Ssu Huang and David Baker of the University of Washington, Seattle; Andrew McGuire and Leonidas Stamatatos of the Seattle Biomedical Research Institute; and TSRI principal investigators Andrew B. Ward, David Nemazee, Ian A. Wilson, and Dennis R. Burton, who is also head of the IAVI Neutralizing Center at TSRI.*

*The project was funded in part by IAVI; the National Institutes of Health (AI84817, AI081625 and AI33292); and the Ragon Institute.*

***http://bit.ly/16bq2aH***

## Kansas was unbearably hot 270 million years ago

***Temperatures soared to nearly 74 degrees Celsius, research suggests***

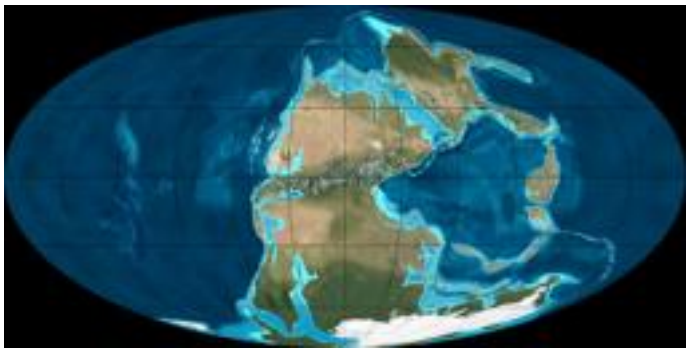
**By Erin Wayman**

The Permian period was hot, hot, hot: Around 270 million years ago, air temperatures near the equator may have soared to almost 74º Celsius or 165º Fahrenheit, scientists report March 18 in Geology. That's far hotter than anywhere on Earth today.

"I can't even imagine what it would have been like," says Neil Tabor, a sedimentary geochemist at Southern Methodist University in Dallas, who wasn't involved in the research. The intense heat may explain why plants and animals vanished from parts of the tropics at this time, he says, a disappearance that preceded the mass extinction that ended the Permian period 252 million years ago. Only microbes that thrive under extreme conditions could have survived such temperatures.

Evidence for the sweltering heat comes from Kansas, which was near the equator during the Permian, when the continents fused to form Pangaea. Previous work showed that, in the middle Permian, western Kansas was a desert where lakes of brine repeatedly formed, evaporated and left behind salt deposits. Geologist Kathleen

Benison of West Virginia University in Morgantown and a colleague had determined that air temperatures there reached 50º C, no hotter than California's Death Valley today.
Benison and Jay Zambito, also of West Virginia, were surprised to find the even hotter temperatures while investigating another site in western Kansas. They looked at crystals of halite, or rock salt, which act as natural thermometers. As the crystals grow in evaporating lakes of brine, they trap microscopic bubbles of saltwater. In the lab, scientists can estimate the temperature at which the bubbles formed, thereby taking the temperature of the ancient brine itself. Each bubble, Benison says, "is a very specific snapshot for a single time and place." Since the brine pools were only tens of centimeters deep, the bubbles are good proxies for air temperature, she adds.



***HOT TIMES During the Permian period, the continents came together to form Pangaea (map of supercontinent's configuration 280 million years ago shown). Air temperatures near the equator might have been as high as nearly 74º Celsius.*** **© Ron Blakey/NAU Geology**

The team collected almost 400 temperature readings across 15 layers in sediments buried roughly 600 to 800 meters deep. The whole record corresponds to about 270 million years ago, but how much time elapsed across the layers is unknown. Near the beginning and end of the record, average air temperatures were in the 20s and 30s Celsius.Temperatures spiked in the middle layers to an average of almost 45º C, with daytime temperatures ranging from about 25º C to almost 74º C. Benison and Zambito plan to investigate temperatures over a larger geographic area to see whether the sizzling heat was a regional phenomenon.
But some researchers are not convinced that the team actually found such extreme temperatures. "It's a question of whether or not they've measured the air temperature," says geochemist Ron Spencer of the University of Calgary in Canada. In some modern salt pans, even shallow brine can be much hotter than the air because it retains heat better.
If the air temperatures are valid, they create a mystery. "It's unclear at the moment how we could get such extreme conditions at this place," Tabor says. Climate simulations can't yet reconcile how the area could be that hot and still have liquid water at the surface.
*J.J. Zambito IV and K.C. Benison. Extremely high temperatures and paleoclimate trends recorded in Permian ephemeral lake halite. Geology. Published online March 18, 2013. doi:10.1130/G34078.1. [Go to]*
*K.C. Benison and R.H. Goldstein. Permian paleoclimate data from fluid inclusions in halite. Chemical Geology. Vol. 154, February 1999, p. 113. [Go to]*

***http://www.sciencedaily.com/releases/2013/03/130328152544.htm***

## Everything You Know About Osmosis Is (Probably) Wrong

***Though the concept is important to physiology, osmosis is understood in a much simpler - and often incorrect - way***

Osmosis - the flow of a solvent across a semipermeable membrane from a region of lower to higher solute concentration - is a well-developed concept in physics and biophysics. The problem is that, even though the concept is important to plant and human physiology, osmosis is understood in biology and chemistry in a much simpler - and often incorrect - way.
"A range of surprising misconceptions about osmosis continue to appear in papers, web sites and textbooks," says Eric Kramer, professor of physics at Bard College at Simon's Rock in Great Barrington, Mass. "Wrong ideas about osmosis are especially common in educational materials aimed at students of chemistry and biology. Once learned, these errors influence the thinking of professionals throughout their careers."
The thermodynamic theory of osmosis was published by J.W. Gibbs in 1897, and during the next half-century dozens of other scientists published explanations for it in terms of interactions between the solute and solvent molecules. "Many of the greatest scientists of the 20th century took a turn at it," says Kramer, "A textbook in 1951 offered the first coherent telling of the whole theory."
Though physicists have had this complete and correct explanation since the 50s, chemistry and biology never caught up. Why? One reason is because the incorrect theory is much easier. "The thermodynamic explanation can be pretty dense, and features entropy, which can be scary for people," he says. "The correct theory would be harder to teach at an introductory level, although I'm working with a textbook author who plans to spread the word."

Reach back into your memory for your first science lesson on osmosis. It probably involved a demonstration with a bag of sugar with holes poked in it. When dunked into water, the water rushed into the bag. Using this example of osmosis, Kramer lays out the common misconceptions:

***(1) "The first misconception is that osmosis is limited to liquids," he says. "But it works just fine for gases, too."***

***(2) "Another misconception that osmosis requires an attractive force," he says. "It doesn't. When water fills the bag of sugar, it's not because the sugar is pulling the water in. That's not part of the explanation."***

***(3) "A misconception is that osmosis always happens down a concentration gradient," he says. "When you dissolve something in water, the water doesn't necessarily get more diluted. Depending on the substance, it can get more concentrated."***

***(4) "Anther misconception is that you don't need to invoke a force to explain why the water flows into the bag. It's thought that, like diffusion, it's a spontaneous process," he says. "But, in fact, there is a force. It's complicated how it happens, but it turns out that the membrane - or the bag, in the familiar lab demonstration - exerts a force that pushes the water in."***

"These misconceptions are surprisingly robust," says Kramer. "Nearly all have been discussed by other authors during the long history of osmotic research, and yet they continue to find believers in each generation of professionals. While authors in physics and biophysics have generally settled on the correct understanding of osmosis, these ideas have not penetrated into the fields of chemistry and biology. It's very surprising that, in 60 years, no physicist talked to a chemist long enough to figure this out."

Kramer is co-author, with colleague and chemist David Myers, of the article, "Osmosis is not driven by water dilution," in the April issue of Trends in Plant Science. They have authored a previous article, "Five popular misconceptions about osmosis," in the American Journal of Physics (August, 2012).

*Eric M. Kramer, David R. Myers. Osmosis is not driven by water dilution. Trends in Plant Science, 2013; DOI: 10.1016/j.tplants.2012.12.001*

***http://www.sciencedaily.com/releases/2013/03/130329085938.htm***

## Innate Immune System Can Kill HIV When a Viral Gene Is Deactivated

***Human cells have an intrinsic capacity to destroy HIV. However, the virus has evolved to contain a gene that blocks this ability.***

When this gene is removed from the virus, the innate human immune system destroys HIV by mutating it to the point where it can no longer survive.

This phenomenon has been shown in test tube laboratory experiments, but now researchers at the University of North Carolina School of Medicine have demonstrated that the same phenomenon occurs in a humanized mouse model, suggesting a promising new target for tackling the virus, which has killed nearly 30 million people worldwide since it first appeared three decades ago.

A family of human proteins called APOBEC3 effectively restrict the growth of HIV and other viruses, but this action is fully counteracted by the viral infectivity factor gene (vif) in HIV. In the study, researchers intravenously infected humanized mice with HIV. They found that the most commonly transmitted strains of HIV are completely neutralized by APOBEC3 proteins when vif is removed from the virus.

"Without the vif gene, HIV can be completely destroyed by the body's own immune system," said J. Victor Garcia, PhD, professor of medicine at the UNC School of Medicine and senior author on the study. "These results suggest a new target for developing drugs fully capable of killing the virus."

Garcia and his colleagues pioneered the humanized mouse model used for these studies. The aptly named "BLT" mouse is created by introducing human bone marrow, liver and thymus tissues into animals without an immune system of their own. The mice have a fully functioning human immune system and can be infected with HIV in the same manner as humans. In previous research, Garcia and his team have effectively prevented intravenous, rectal, vaginal and oral transmission of HIV in the mice with pre-exposure prophylaxis (PrEP).

For the current study, Garcia and his colleagues also infected BLT mice with another, highly harmful strain of the virus. The results show that this strain of HIV does continue to replicate, even without vif, but at a much slower rate and without harming the human immune system. Further, the researchers found that virus replication in this case was limited to one tissue -- the thymus -- in the entire body.

"These findings demonstrate a fundamental weakness in HIV," said John F. Krisko, PhD, lead author on the study. "If this weakness can be exploited, it might eventually lead to a cure for HIV/AIDS," Krisko said.

The study appears March 28 in the online journal PloS Pathogens.

In addition to Garcia and Krisko, other study authors are Francisco Martinez-Torres, PhD, and John L. Foster, PhD, all of the Center for AIDS Research at the University of North Carolina School of Medicine.

*John F. Krisko, Francisco Martinez-Torres, John L. Foster, J. Victor Garcia. HIV Restriction by APOBEC3 in Humanized Mice. PLoS Pathogens, 2013; 9 (3): e1003242 DOI: 10.1371/journal.ppat.1003242*

http://www.sciencedaily.com/releases/2013/03/130330130531.htm

# Artificial Spleen to Treat Bloodstream Infections: Sepsis Therapeutic Device Under Development

***A DARPA contract further advances a blood-cleansing technology and accelerate its translation to humans as a new type of sepsis therapy***

The Wyss Institute for Biologically Inspired Engineering at Harvard University announced today that it was awarded a $9.25 million contract from the Defense Advanced Research Projects Agency (DARPA) to further advance a blood-cleansing technology developed at the Institute with prior DARPA support, and help accelerate its translation to humans as a new type of sepsis therapy.

The device will be used to treat bloodstream infections that are the leading cause of death in critically ill patients and soldiers injured in combat.

To rapidly cleanse the blood of pathogens, the patient's blood is mixed with magnetic nanobeads coated with a genetically engineered version of a human blood 'opsonin' protein that binds to a wide variety of bacteria, fungi, viruses, parasites, and toxins. It is then flowed through microchannels in the device where magnetic forces pull out the bead-bound pathogens without removing human blood cells, proteins, fluids, or electrolytes -- much like a human spleen does. The cleansed blood then flows back to the patient.

***The Spleen-on-a-chip, developed at the Wyss Institute, will be used to treat bloodstream infections that are the leading cause of death in critically ill patients and soldiers injured in combat.*** **(Credit: Wyss Institute)**

"In just a few years we have been able to develop a suite of new technologies, and to integrate them to create a powerful new device that could potentially transform the way we treat sepsis," said Wyss founding director and project leader, Don Ingber, M.D., Ph.D. "The continued support from DARPA enables us to advance our device manufacturing capabilities and to obtain validation in large animal models, which is precisely what is required to enable this technology to be moved towards testing in humans."

The team will work to develop manufacturing and integration strategies for its core pathogen-binding opsonin and Spleen-on-a-Chip fluidic separation technologies, as well as a novel coating technology called "SLIPS," which is a super-hydrophobic coating inspired from the slippery surface of a pitcher plant that repels nearly any material it contacts. By coating the inner surface of the channels of the device with SLIPS, blood cleansing can be carried out without the need for anticoagulants to prevent blood clotting.

In addition to Ingber, the multidisciplinary team behind this effort includes Wyss core faculty and Harvard School of Engineering and Applied Science faculty member Joanna Aizenberg, Ph.D., who developed the SLIPS technology; Wyss senior staff member Michael Super, Ph.D., who engineered the human opsonin protein; and Mark Puder, M.D., Ph.D., Associate Professor of Pediatric Surgery at Boston Children's Hospital and Harvard Medical School who will be assisting with animal studies.

http://www.space.com/20426-mercury-meteorite-discovery-messenger.html

# Green Meteorite May Be from Mercury, a First

***Scientists may have discovered the first meteorite from Mercury.***

**by Miriam Kramer, SPACE.com Staff Writer**

The green rock found in Morocco last year may be the first known visitor from the solar system's innermost planet, according to meteorite scientist Anthony Irving, who unveiled the new findings this month at the 44th annual Lunar and Planetary Science Conference in The Woodlands, Texas. The study suggests that a space rock called NWA 7325 came from Mercury, and not an asteroid or Mars. NWA 7325 is actually a group of 35 meteorite samples discovered in 2012 in Morocco. They are ancient, with Irving and his team dating the rocks to an age of about 4.56 billion years.

***This green meteorite that landed in Morocco in 2012 could be from Mercury. Stefan Ralew/sr-meteorites.de***

"It might be a sample from Mercury, or it might be a sample from a body smaller than Mercury but [which] is like Mercury," Irving said during his talk. A large impact could have shot NWA 7325 out from Mercury to Earth, he added.

Irving is an Earth and Space Sciences professor at the University of Washington and has been studying meteorites for years. But the NWA 7325 meteorite is unlike anything found on Earth before, he told SPACE.com.

Meteorites from Mars are imbued with some Martian atmosphere, making them somewhat simple to tell apart from other rocks. Space rocks from Vesta, one of the largest asteroids in the solar system, are also chemically distinct, but NWA 7325 does not resemble any space rock documented by scientists today.

Irving thinks that the meteoritewas created and eventually ejected from a planet or other body that had flowing magma on its surface at some point in its history. Evidence suggests that the rock could have been formed as "scum" on the top of the magma, Irving said.

NWA 7325 has a lower magnetic intensity — the magnetism passed from a cosmic body's magnetic field into a rock — than any other rock yet found, Irving said. Data sent back from NASA's Messenger spacecraft currently in orbit around Mercury shows that the planet's low magnetism closely resembles that found in NWA 7325, Irving said.

Messenger's observations also provided Irving with further evidence that could support his hypothesis. Scientists familiar with Mercury's geological and chemical composition think that the planet's surface is very low in iron. The meteorite is also low in iron, suggesting that wherever the rock came from, its parent body resembles Mercury.

While Messenger's first extended mission just finished, the team has put in a request to continue researching the planet with the orbiter for the next two years. If the mission is extended until 2015, the science returned by the spacecraft could help further validate or invalidate Irving's ideas about the origin of the meteorite.Although finding meteorites on Earth that came from Mercury is less likely than finding Martian meteorites, it could be possible, Irving said.