http://www.sciencedaily.com/releases/2012/09/120903221037.htm

Less Ferocious Tasmanian Devils Could Help Save Species from Extinction

Evolving to become less aggressive could be key to saving the Tasmanian devil

Tasmanian devil. **(Credit: © redzaal / Fotolia)**

ScienceDaily - Evolving to become less aggressive could be key to saving the Tasmanian devil - famed for its ferocity - from extinction, research suggests. The species is being wiped out by Devil Facial Tumour Disease (DFTD), a fatal infectious cancer spread by biting. The new study, published in the British Ecological Society's Journal of Animal Ecology, found the less often a devil gets bitten, the more likely it is to become infected with the cancer.

According to lead author Dr Rodrigo Hamede of the University of Tasmania: "Our results - that devils with fewer bites are more likely to develop DFTD - were very surprising and counter-intuitive. In most infectious diseases there are so-called super-spreaders, a few individuals responsible for most of the transmission. But we found the more aggressive devils, rather than being super-spreaders, are super-receivers."

To find out whether biting frequency predicted acquiring DFTD, Dr Hamede and his colleagues set up dozens of devil traps at two sites for 10-day periods every three months between 2006 and 2010. They then recorded the pattern of injuries in the devils, and identified any tumours. One of the sites - West Pencil Pine - was selected because devils there seem to be less badly hit by the disease.

They made three discoveries: the level of bites was similar at both sites; devils with fewer bites were significantly more likely to develop DFTD; and most tumours occurred in devils' mouths. "This means that more aggressive devils do not get bitten as often, but they bite the tumours of the less aggressive devils and become infected," explains Dr Hamede.

Because there is no treatment for, or vaccine against, DFTD, the findings and the next stage of the research have important implications for saving the species from extinction. "Our next step is fascinating. First we need to explore the genetic differences that might be lessening the impact of DFTD in the West Pencil Pine devil population. Second, we need more detailed data on devil behaviour to define 'shy' or 'bold' types. We could then use this information to develop a management strategy to reduce the spread of the disease by boosting natural selection of less aggressive, and therefore more resilient, devils."

Understanding how infectious diseases spread is key to controlling them, but studying disease transmission in wild animals is often very difficult. And in DFTD, which is spread by biting, ecologists also need a better understanding of devil behaviour. Devils are solitary yet social animals. They do not live in groups but meet each other often, either during mating, establishing social hierarchies or when feeding around carcasses - all occasions when they bite each other.

Rodrigo K. Hamede, Hamish McCallum, Menna Jones. Biting injuries and transmission of Tasmanian devil facial tumour disease. Journal of Animal Ecology, 2012; DOI: 10.1111/j.1365-2656.2012.02025.x

http://www.gizmag.com/cellulose-nanocrystals-stronger-carbon-fiber-kevlar/23959/

Wood pulp extract stronger than carbon fiber or Kevlar

CNCs are stronger and stiffer than Kevlar or carbon fibers, putting CNC into composite materials results in high strength, low weight products costing ninety percent less than Kevlar fiber or carbon fiber

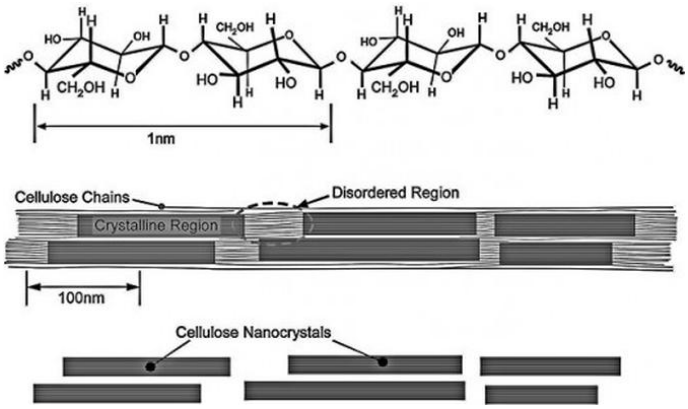
By Brian Dodson

The Forest Products Laboratory of the US Forest Service has opened a US$1.7 million pilot plant for the production of cellulose nanocrystals (CNC) from wood by-products materials such as wood chips and sawdust. Prepared properly, CNCs are stronger and stiffer than Kevlar or carbon fibers, so that putting CNC into composite materials results in high strength, low weight products. In addition, the cost of CNCs is less than ten percent of the cost of Kevlar fiber or carbon fiber. These qualities have attracted the interest of the military for use in lightweight armor and ballistic glass (CNCs are transparent), as well as companies in the automotive, aerospace, electronics, consumer products, and medical industries.

Cellulose is the most abundant biological polymer on the planet and it is found in the cell walls of plant and bacterial cells. Composed of long chains of glucose molecules, cellulose fibers are arranged in an intricate web that provides both structure and support for plant cells. The primary commercial source for cellulose is wood, which is essentially a network of cellulose fibers held together by a matrix of lignin, another natural polymer which is easily degraded and removed.

Wood pulp is produced in a variety of processes, all of which break down and wash away the lignin, leaving behind a suspension of cellulose fibers in water. A typical cellulose wood fiber is only tens of microns wide and about a millimeter long.

The cellulose in wood pulp, when dry, has the consistency of fluff or lint - a layer of wood pulp cellulose has mechanical properties reminiscent of a wet paper towel. Not what you might expect to be the source of one of the strongest materials known to Man. After all, paper is made from the cellulose in wood pulp, and doesn't show extraordinary strength or stiffness.



The upper figure shows the structure of the cellulose polymer; the middle figure shows a nanofibril containing both crystalline and amorphous cellulose; the lower figure shows the cellulose nanocrystals after the amorphous cellulose is removed by acid hydrolysis

Further processing breaks the cellulose fibers down into nanofibrils, which are about a thousand times smaller than the fibers. In the nanofibrils, cellulose takes the form of three-dimensional stacks of unbranched, long strands of glucose molecules, which are held together by hydrogen bonding. While not being "real" chemical bonds, hydrogen bonds between cellulose molecules are rather strong, adding to the strength and stiffness of cellulose nanocrystals.

Within these nanofibrils are regions which are very well ordered, in which cellulose chains are closely packed in parallel with one another. Typically, several of these crystalline regions appear along a single nanofibril, and are separated by amorphous regions which do not exhibit a large degree of order. Individual cellulose nanocrystals are then produced by dissolving the amorphous regions using a strong acid.

At present the yield for separating CNCs from wood pulp is about 30 percent. There are prospects for minor improvements, but the limiting factor is the ratio of crystalline to amorphous cellulose in the source material. A near-term goal for the cost of CNCs is $10 per kilogram, but large-scale production should reduce that figure to one or two dollars a kilo.

CNCs separated from wood pulp are typically a fraction of a micron long and have a square cross-section a few nanometers on a side. Their bulk density is low at 1.6 g/cc, but they exhibit incredible strength. An elastic modulus of nearly 150 GPa, and a tensile strength of nearly 10 GPa. Here's how its strength to compares to some better-known materials:

Material	**Elastic Modulus**		**Tensile Strength**
CNC	150 GPa	7	5 GPa
Kevlar 49	125 GPa	3	5 GPa
Carbon fiber	150 GPa	3	5 GPa
Carbon nanotubes	300 GPa		20 GPa
Stainless steel	200 GPa	0	5 GPa
Oak	10 GPa	0	1 GPa

The only reinforcing material that is stronger than cellulose nanocrystals is a carbon nanotube, which costs about 100 times as much. Stainless steel is included solely as a comparison to conventional materials. The relatively very low strength and modulus of oak points out how much the structure of a composite material can degrade the mechanical properties of reinforcing materials.

As with most things, cellulose nanocrystals are not a perfect material. Their greatest nemesis is water. Cellulose is not soluble in water, nor does it depolymerize. The ether bonds between the glucose units of the cellulose molecule are not easily broken apart, requiring strong acids to enable cleavage reactions.

The hydrogen bonds between the cellulose molecules are also too strong in aggregate to be broken by encroaching water molecules. Indeed, crystalline cellulose requires treatment by water at 320° C and 250 atmospheres of pressure before enough water intercalates between the cellulose molecules to cause them to become amorphous in structure. The cellulose is still not soluble, just disordered from their near-perfect stacking in the crystalline structure.

But cellulose contains hydroxyl (OH) groups which protrude laterally along the cellulose molecule. These can form hydrogen bonds with water molecules, resulting in cellulose being hydrophilic (a drop of water will tend to spread across the cellulose surface). Given enough water, cellulose will become engorged with water, swelling to nearly double its dry volume.

Swelling introduces a large number of nano-defects in the cellulose structure. Although there is little swelling of a single CNC, water can penetrate into amorphous cellulose with ease, pushing apart the individual cellulose molecules in those regions. In addition, the bonds and interfaces between neighboring CNC will be disrupted, thereby significantly reducing the strength of any material reinforced with CNCs. To make matters worse, water can move easily over the surface/interfaces of the CNCs, thereby allowing water to penetrate far into a composite containing CNCs.

There are several approaches to make CNC composite materials viable choices for real world applications. The simplest, but most limited, is to choose applications in which the composite will not be exposed to water. Another is to alter the surface chemistry of the cellulose so that it becomes hydrophobic, or water-repelling. This is easy enough to do, but will likely substantially degrade the mechanical properties of the altered CNCs. A third approach is to choose a matrix material which is hydrophobic, and preferably that forms a hydrophobic interface with CNCs. While not particularly difficult from a purely chemical viewpoint, there is the practical difficulty that interfaces between hydrophobic and hydrophilic materials are usually severely lacking in strength. Perhaps the most practical approach will simply be to paint or otherwise coat CNC composite materials in some material that keeps water away. For such a prize - inexpensive strong and rigid materials - we can be sure that innovations will follow to make the theoretical practical. *Source: US Forest Service*

http://www.wired.com/dangerroom/2012/09/perfume/

Perfume of War: Iran Makes Musk to Conceal Troops

This week Iran revealed a perfume device that its inventor claims hides the smell of gunpowder.

By Robert Beckhusen

Call it the Dior of the Islamic Revolutionary Guards Corps. In one of the more bizarre military inventions from Iran, the U.S. arch-enemy has reportedly developed a perfume machine to hide troops during combat.

Iran's semi-official Fars News Agency reported that an Iranian inventor created a "fragrance making and spraying device to deceive enemies on the battlefield." The invention, called "Deceit Perfume" and jointly built as a "strategic project of the armed forces," is intended to camouflage the smell of gunpowder by spreading odors over "vast areas." Tehran's troops will also have a choice of four agreeable aromas: fresh air, rainy weather, seaside weather (for the navy) and tea, according to the news agency.

There's no telling how the device will deliver the perfume or how large it is - perhaps it's in the shape of a gun - but the machine is said to be a "highly effective and strategic weapon for civil defense, and for pushing back enemy threats, surprise attacks and offenses," inventor Mohammad Sadeh Pir-Tavana told Fars. Perhaps it could be used by Iran's 3,500-strong ninja army.

The device is mainly to be used during an unconventional war against a much larger foe, where the smell of cordite from a recent firefight could give away the location of Iranian insurgents. Instead of being alerted to the Iranians' presence by the smell, America's troops may be deceived into letting their guard down by the refreshing scent of an incoming rainstorm. On the other hand, if U.S. troops were actually chasing insurgents around in the middle of a hypothetical war with Iran, suddenly encountering the smell of tea - or the scent of rainy weather when it's not cloudy or raining - could be a dead giveaway that the insurgents are close after all. But never mind.

It wouldn't be the first time America's foes have attempted to militarize musk. In 2010, al-Qaida in the Arabian Peninsula was reported to have attempted to kill Saudi Arabian officials and clerics with poisoned perfume. The plot, though, apparently never made it past the planning stages, and which was to include robbing banks to pay for the plot. Iran's military fragrances are more defensive in nature.

The perfume machine is also just the latest - and weirdest - in a series of boasts and military projects announced by Tehran in recent days. The more conventional announcements include Iran building a new drone called the Shaparak, or Butterfly, which the Revolutionary Guards wants to equip with missiles. Minister of Defense Ahmad Vahidi reiterated on Monday that developing smart bombs constitutes "one of the main and important strategies of the defense ministry." Iran also announced on Monday that its drones are now operating with air defense units, and the the Islamic Republic is looking to boost its air defense grid with new missiles and an indigenous version of Russia's S-300 surface-to-air missile launcher.

The IAEA, meanwhile, believes Iran has doubled its number of nuclear centrifuges, but Iran is reportedly having trouble updating equipment needed to speed up uranium enrichment, which could be used to build a bomb.

Now for another far-out announcement, Admiral Habibollah Sayyari said Tuesday that Iran plans to send warships near U.S. coasts within "the next few years," according to the Associated Press. But, erm, Tehran regularly makes announcements like that. And actually sending a warship - while possible if Iran managed to

get the money together and secured a resupply base in a country like Venezuela - would be so pitiful a threat to the homeland that the U.S. military would instead "probably be grateful for the opportunity to study an Iranian ship close up," wrote National Post's Matt Gurney.
Suffice to say, if Iran ever does send a warship, somehow using a perfume machine to spray the scent of seaside weather doesn't sound like it will be very effective. Of course, the U.S. might not have reckoned with the ninja army. And we all know that ninjas will use every trick at hand - including scent - in order to sneak up and get you.

http://www.sciencedaily.com/releases/2012/09/120904170924.htm

Scientists Design Molecule That Reverses Some Fragile X Syndrome Defects

Scientists have designed a compound that shows promise as a potential therapy for a disease closely linked to fragile X syndrome

ScienceDaily - Scientists on the Florida campus of The Scripps Research Institute have designed a compound that shows promise as a potential therapy for one of the diseases closely linked to fragile X syndrome, a genetic condition that causes mental retardation, infertility, and memory impairment, and is the only known single-gene cause of autism.
The study, published online ahead of print in the journal ACS Chemical Biology September 4, 2012, focuses on tremor ataxia syndrome, which usually affects men over the age of 50 and results in Parkinson's like-symptoms - trembling, balance problems, muscle rigidity, as well as some neurological difficulties, including short-term memory loss and severe mood swings.
With fragile X syndrome, tremor ataxia syndrome, and related diseases, the root of the problem is a structural motif known as an "expanded triplet repeat" - in which a series of three nucleotides are repeated more times than normal in the genetic code of affected individuals. This defect, located in the fragile X mental retardation 1 (FMR1) gene, causes serious problems with the processing of RNA.
"While there is an abundance of potential RNA drug targets in disease, no one has any idea how to identify or design small molecules to target these RNAs," said Mathew Disney, a Scripps Research associate professor who led the study. "We have designed a compound capable of targeting the right RNA and reversing the defects that cause fragile X-associated tremor ataxia."

Preventing Havoc

In tremor ataxia syndrome, the expanded triplet repeat leads to the expression of aberrant proteins that wreak widespread havoc. The repeats actually force the normal proteins that regulate RNA splicing - necessary for production of the right kind of proteins - into hiding. The compound designed by Disney and his colleagues not only improves the RNA splicing process, but also minimizes the ability of repeats to wreak havoc on a cell.
"It stops the repeat-associated defects in cell culture," Disney said, "and at fairly high concentrations, it completely reverses the defects. More importantly, the compound is non-toxic to the cells. It looks like a very good candidate for development, but we're still in the early stages of testing."
Overall, this study reinforces Disney's earlier findings showing it is possible to identify and develop small molecules that target these traditionally recalcitrant RNA defects. In March of this year, Disney published a study in the Journal of the American Chemical Society that described a small molecule that inhibited defects in myotonic dystrophy type 1 RNA in both cellular and animal models of disease.
"We've gotten very good at targeting RNA with small molecules, something a lot of people said couldn't be done," Disney pointed out. "Our approach is evolving into a general method that can be used to target any disease that is associated with an RNA, including, perhaps, fragile X syndrome itself." The new compound also works as a probe to better understand how these repeats cause fragile X syndrome and how they contribute to tremor ataxia, Disney added.

In addition to Disney, authors of the study, "Small Molecule That Targets r(CGG) and Improves Defects in 2 Fragile X-Associated Tremor Ataxia Syndrome," include Biao Liu, Wang-Yong Yang, Tuan Tran, and Jessica L. Childs-Disney of Scripps Research; and Nicolas Charlet-Berguerand and Chantal Sellier of the Institut de Génétique et de Biologie Moléculaire et Cellulaire (IGBMC), Institut National de la Santé et de la Recherche Médicale (INSERM), the Centre National de la Recherche Scientifique (CNRS), and University of Strasbourg, Illkirch, France.

The study was funded by the National Institutes of Health (award numbers 3R01GM079235-02S1 and 1R01GM079235-01A2), INSERM, the French National Research Agency, and The Scripps Research Institute.

Matthew D. Disney, Biao Liu, Wang-Yong Yang, Chantal Sellier, Tuan Tran, Nicolas Charlet-Berguerand, Jessica L. Childs-Disney. A Small Molecule That Targets r(CGG)expand Improves Defects in Fragile X-Associated Tremor Ataxia Syndrome. ACS Chemical Biology, 2012; 120904130429000 DOI: 10.1021/cb300135h

Raman Parkesh, Jessica L. Childs-Disney, Masayuki Nakamori, Amit Kumar, Eric Wang, Thomas Wang, Jason Hoskins, Tuan Tran, David Housman, Charles A. Thornton, Matthew D. Disney. Design of a Bioactive Small Molecule That Targets the

Myotonic Dystrophy Type 1 RNA via an RNA Motif–Ligand Database and Chemical Similarity Searching. Journal of the American Chemical Society, 2012; 134 (10): 4731 DOI: 10.1021/ja210088v

http://www.sciencedaily.com/releases/2012/09/120904183923.htm

Longer CPR Attempts Might Benefit Some Patients, Research Finds

New research shows longer attempts might be beneficial for some patients.

ScienceDaily - There isn't a hard and fast rule for how long doctors should perform CPR, but new research from the University of Michigan Health System shows longer attempts might be beneficial for some patients.

Most cardiac arrest patients are often successfully resuscitated after a short period of time - about 12 minutes on average. Practitioners are often reluctant to perform longer attempts - those that can last 30 minutes or longer - because if patients do not survive early on during cardiac arrest, their overall prognosis is poor.

The research from U-M, however, shows that for some patients, successful resuscitation only occurred after 30 minutes or more.

On a broader level, the researchers found that patients at hospitals where attempts were longer, on average, had a higher likelihood of immediate survival and survival to discharge, even after accounting for differences in overall patient characteristics. Importantly, patients who survived with longer arrest times did not appear to have substantially worse neurological function at discharge. The study, led by cardiologist Zachary D. Goldberger, M.D., M.S., an assistant professor at the University of Washington, and recently a Robert Wood Johnson Foundation cardiology fellow at U-M , was published online in The Lancet.

Each year, about 200,000 hospitalized patients will experience cardiac arrest, with only half of those surviving the initial arrest, and fewer than 20 percent surviving to discharge.

Goldberger says it's not surprising that there exists a wide variation among hospitals' average length of resuscitation attempts, given there's no firm evidence to guide practitioners when to stop their efforts once resuscitative efforts have started. "Our findings suggest an opportunity for improving care in this high-risk population. Overall, it may involve standardizing the time required for continuing resuscitation attempts prior to decisions regarding termination of efforts," Goldberger says.

After examining national data for more than 64,000 cardiac arrest patients between 2000 and 2008, the researchers found that while most patients were successfully resuscitated after a short period of time, about 15 percent of patients who survived needed at least 30 minutes to achieve a pulse.

While the study implies that longer attempts should be considered by medical professionals, U-M cardiologist Brahmajee K. Nallamothu, M.D., M.P.H. and senior author, says the research is only one piece of evidence to weigh during a cardiac arrest case. "We want to emphasize that our findings cannot identify an optimal duration for which to resuscitate patients," he says.

Steven L. Kronick, M.D., M.S., one of the paper's authors, U-M emergency department physician head of the U-M's CPR committee, agrees and says the research should be a part of ongoing efforts directed toward improving care for cardiac arrest patients. "The optimal resuscitation duration for any individual patient will continue to remain a bedside decision that relies on careful clinical judgment," he says. "Overall, we believe these findings present an opportunity to improve resuscitation care, especially at a systems-level."

Zachary D Goldberger MD,Paul S Chan MD,Prof Robert A Berg MD,Steven L Kronick MD,Colin R Cooke MD,Mingrui Lu MPH,Mousumi Banerjee PhD,Prof Rodney A Hayward MD,Prof Harlan M Krumholz MD,Dr Brahmajee K Nallamothu MD,for the American Heart Association Get With The Guidelines - Resuscitation (formerly the National Registry of Cardiopulmonary Resuscitation) Investigators. Duration of resuscitation efforts and survival after in-hospital cardiac arrest: an observational study. The Lancet, 5 September 2012 DOI: 10.1016/S0140-6736(12)60862-9

http://bit.ly/Q8Vnx4

Tutankhamun's death and the birth of monotheism

TUTANKHAMUN'S mysterious death as a teenager may finally have been explained.

05 September 2012 by Jessica Hamzelou

TUTANKHAMUN'S mysterious death as a teenager may finally have been explained. And the condition that cut short his life may also have triggered the earliest monotheistic religion, suggests a new review of his family history.

Since his lavishly furnished, nearly intact tomb was discovered in 1922, the cause of Tutankhamun's death has been at the centre of intense debate. There have been theories of murder, leprosy, tuberculosis, malaria, sickle-cell anaemia, a snake bite - even the suggestion that the young king died after a fall from his chariot.

But all of these theories have missed one vital point, says Hutan Ashrafian, a surgeon with an interest in medical history at Imperial College London. Tutankhamun died young with a feminised physique, and so did his immediate predecessors.

Paintings and sculptures show that Smenkhkare, an enigmatic pharaoh who may have been Tutankhamun's uncle or older brother, and Akhenaten, thought to have been the boy king's father, both had feminised figures, with unusually large breasts and wide hips. Two pharaohs that came before Akhenaten - Amenhotep III and Tuthmosis IV - seem to have had similar physiques. All of these kings died young and mysteriously, says Ashrafian. "There are so many theories, but they've focused on each pharaoh individually."

Ashrafian found that each pharaoh died at a slightly younger age than his predecessor, which suggests an inherited disorder, he says. Historical accounts associated with the individuals hint at what that disorder may have been.

"It's significant that two [of the five related pharaohs] had stories of religious visions associated with them," says Ashrafian. People with a form of epilepsy in which seizures begin in the brain's temporal lobe are known to experience hallucinations and religious visions, particularly after exposure to sunlight. It's likely that the family of pharaohs had a heritable form of temporal lobe epilepsy, he says.

This diagnosis would also account for the feminine features. The temporal lobe is connected to parts of the brain involved in the release of hormones, and epileptic seizures are known to alter the levels of hormones involved in sexual development. This might explain the development of the pharaohs' large breasts. A seizure might also be to blame for Tutankhamun's fractured leg, says Ashrafian (Epilepsy & Behavior, doi.org/h8s).

Tuthmosis IV had a religious experience in the middle of a sunny day, recorded in the Dream Stele - an inscription near the Great Sphinx in Giza. But his visions were nothing compared with those experienced by Akhenaten. They encouraged Akhenaten to raise the status of a minor deity called the "sun-disk", or Aten, into a supreme god - abandoning the ancient Egyptian polytheistic traditions to start what is thought to be the earliest recorded monotheistic religion. If Ashrafian's theory is correct, Akhenaten's religious experiment and Tutankhamun's premature death may both have been a consequence of a medical condition.

"People with temporal lobe epilepsy who are exposed to sunlight get the same sort of stimulation to the mind and religious zeal," says Ashrafian.

"It's a fascinating and plausible explanation," says Howard Markel, a medical historian at the University of Michigan in Ann Arbor. However, the theory is almost impossible to prove, he adds, given that there is no definitive genetic test for epilepsy.

Orrin Devinsky, a neurologist at the New York University Langone Medical Center, thinks the theory must remain speculative. "The exact timing of Akhenaten's religious conviction is not so clearly documented, and most cases of sudden religious conversion are not due to epilepsy," he says. "Monotheism could be related to epilepsy, or bipolar disorder, or schizophrenia, or drug intoxication from a fungus - but this paper does not sway me to any of these options."

Markel agrees: "Do we know that a seizure led to monotheism? It's a nice idea, but we don't know," he says. "It's a very interesting hypothesis, but it's just that - there's no definite proof."

http://www.eurekalert.org/pub_releases/2012-09/hu-tgs090512.php

Tough gel stretches to 21 times its length, recoils, and heals itself

Biocompatible material created at Harvard is much tougher than cartilage

Cambridge, Mass. - A team of experts in mechanics, materials science, and tissue engineering at Harvard have created an extremely stretchy and tough gel that may pave the way to replacing damaged cartilage in human joints. Called a hydrogel, because its main ingredient is water, the new material is a hybrid of two weak gels that combine to create something much stronger. Not only can this new gel stretch to 21 times its original length, but it is also exceptionally tough, self-healing, and biocompatible - a valuable collection of attributes that opens up new opportunities in medicine and tissue engineering.

The material, its properties, and a simple method of synthesis are described in the September 6 issue of Nature.

"Conventional hydrogels are very weak and brittle - imagine a spoon breaking through jelly," explains lead author Jeong-Yun Sun, a postdoctoral fellow at the Harvard School of Engineering and Applied Sciences (SEAS). "But because they are water-based and biocompatible, people would like to use them for some very challenging applications like artificial cartilage or spinal disks. For a gel to work in those settings, it has to be able to stretch and expand under compression and tension without breaking."

Sun and his coauthors were led by three faculty members: Zhigang Suo, Allen E. and Marilyn M. Puckett Professor of Mechanics and Materials at SEAS and a Kavli Scholar at the Kavli Institute for Bionano Science and Technology at Harvard; Joost J. Vlassak, Gordon McKay Professor of Materials Engineering and an Area Dean at SEAS; and David J. Mooney, Robert P. Pinkas Family Professor of Bioengineering at SEAS and a Core Faculty Member at the Wyss Institute for Biologically Inspired Engineering at Harvard.

To create the tough new hydrogel, they combined two common polymers. The primary component is polyacrylamide, known for its use in soft contact lenses and as the electrophoresis gel that separates DNA fragments in biology labs; the second component is alginate, a seaweed extract that is frequently used to thicken food. Separately, these gels are both quite weak - alginate, for instance, can stretch to only 1.2 times its length before it breaks. Combined in an 8:1 ratio, however, the two polymers form a complex network of crosslinked chains that reinforce one another. The chemical structure of this network allows the molecules to pull apart very slightly over a large area instead of allowing the gel to crack.

The alginate portion of the gel consists of polymer chains that form weak ionic bonds with one another, capturing calcium ions (added to the water) in the process. When the gel is stretched, some of these bonds between chains break - or "unzip," as the researchers put it - releasing the calcium. As a result, the gel expands slightly, but the polymer chains themselves remain intact. Meanwhile, the polyacrylamide chains form a grid-like structure that bonds covalently (very tightly) with the alginate chains.

Therefore, if the gel acquires a tiny crack as it stretches, the polyacrylamide grid helps to spread the pulling force over a large area, tugging on the alginate's ionic bonds and unzipping them here and there. The research team showed that even with a huge crack, a critically large hole, the hybrid gel can still stretch to 17 times its initial length.

Importantly, the new hydrogel is capable of maintaining its elasticity and toughness over multiple stretches. Provided the gel has some time to relax between stretches, the ionic bonds between the alginate and the calcium can "re-zip," and the researchers have shown that this process can be accelerated by raising the ambient temperature.

The group's combined expertise in mechanics, materials science, and bioengineering enabled the group to apply two concepts from mechanics - crack bridging and energy dissipation - to a new problem. "The unusually high stretchability and toughness of this gel, along with recovery, are exciting," says Suo. "Now that we've demonstrated that this is possible, we can use it as a model system for studying the mechanics of hydrogels further, and explore various applications." "It's very promising," Suo adds.

Beyond artificial cartilage, the researchers suggest that the new hydrogel could be used in soft robotics, optics, artificial muscle, as a tough protective covering for wounds, or "any other place where we need hydrogels of high stretchability and high toughness."

Additional coauthors included Xuanhe Zhao, a former Ph.D. student and postdoc at SEAS, now a faculty member at Duke University; Widusha R. K. Illeperuma, a graduate student at SEAS; Ovijit Chaudhuri, a postdoc in Mooney's lab; and Kyu Hwan Oh, Sun's former adviser and a faculty member at Seoul National University in Korea.

This work was supported by the U.S. Army Research Office, the National Science Foundation (NSF), the Defense Advanced Research Projects Agency, the National Institutes of Health, and the NSF-funded Materials Research Science and Engineering Center (MRSEC) at Harvard. The researchers also individually received support from the NSF Research Triangle MRSEC, a Haythornthwaite Research Initiation grant, the National Research Foundation of Korea, an Alexander von Humboldt Award, and Harvard University.

http://www.eurekalert.org/pub_releases/2012-09/miot-rib090512.php

Researchers identify biochemical functions for most of the human genome

1% of the human genome codes for proteins. What is the rest of the DNA doing.? 80% of the genome is likely involved in regulating the expression of nearby genes

Anne Trafton, MIT News Office

CAMBRIDGE, MA - Only about 1 percent of the human genome contains gene regions that code for proteins, raising the question of what the rest of the DNA is doing. Scientists have now begun to discover the answer: About 80 percent of the genome is biochemically active, and likely involved in regulating the expression of nearby genes, according to a study from a large international team of researchers.

The consortium, known as ENCODE (which stands for "Encyclopedia of DNA Elements"), includes hundreds of scientists from several dozen labs around the world. Using genetic sequencing data from 140 types of cells, the researchers were able to identify thousands of DNA regions that help fine-tune genes' activity and influence which genes are expressed in different kinds of cells.

Just as the sequencing of the human genome helped scientists learn how mutations in protein-coding genes can lead to disease, the new map of noncoding regions should provide some answers on how mutations in the regulatory elements lead to diseases such as lupus and diabetes, says Manolis Kellis, an associate professor of computer science at MIT, an associate member of the Broad Institute and an author of a paper describing the findings in the Sept. 5 online edition of Nature.

"Humans are 99.9 percent identical to each other, and you only have one difference in every 300 to 1,000 nucleotides," Kellis says. "What ENCODE allows you to do is provide an annotation of what each nucleotide of

the genome does, so that when it's mutated, we can make some predictions about the consequences of the mutation." Kellis, who leads MIT's Computational Biology Group, is one of the principal investigators involved in the Nature paper. The ENCODE collaboration is publishing about two dozen additional papers this week detailing the new results.

Mapping noncoding DNA

ENCODE was established in 2003 to extend our understanding of the human genome beyond protein-coding genes. One way to do that is by studying the chemical modifications of individual stretches of DNA, which control when genetic regions will be active. These modifications vary by cell type and can modify either DNA directly or the histone proteins that DNA wraps around.

To map these modifications, known collectively as the epigenome, the research groups had to collect many different kinds of data from different cell types. Some labs measured DNA or histone modifications, while others gauged the accessibility of different stretches of DNA by cutting it into fragments with enzymes.

Kellis and his group were among the computational scientists leading the effort to analyze and integrate the huge amount of data generated by different labs. "Given that we were getting more than 1,000 data sets, we had to figure out ways to automatically calibrate experiments," says Anshul Kundaje, a research scientist in MIT's Computational Biology Group. "We developed an almost purely automated system that did all of this."

The ENCODE researchers found that 80 percent of the genome experiences some kind of biochemical event, such as binding to proteins that regulate how often a neighboring gene is utilized. They also discovered that the same regulatory region can play different roles, depending on what type of cell it's acting in.

Human variation

The researchers also studied the conservation of nucleotides - the A, T, C and G "letters" of DNA - in the newly identified regulatory regions. Nucleotides are conserved if they remain the same over long evolutionary periods, which can be measured by analyzing the variability between species, or among individuals within a species.

A recent paper by Kellis and colleagues showed that 5 percent of noncoding DNA is conserved across mammals. In one of the ENCODE companion papers appearing online Sept. 5 in Science, Kellis and MIT postdoc Lucas Ward show that an additional 4 percent is conserved within the human lineage, suggesting that those elements control recently evolved traits, some of which are unique to humans.

When the researchers looked at the functions of genes near newly evolved regulatory regions, they found many genes that encode regulators that activate other genes. "Genes involved in the nerve growth pathway and color vision, both of which have been hypothesized to be recent innovations in the primate lineage, are enriched in human-constrained elements in non-conserved regions," Ward says.

The researchers found that the most highly conserved nucleotides were also the ones most likely to be associated with disease when mutated. They also showed that variants associated with autoimmune diseases such as lupus and rheumatoid arthritis are located in regions active only in immune cells, while variants linked to metabolic diseases are in regions active only in liver cells.

In their next phase, the ENCODE researchers hope to determine just how those variations lead to human disease. "What we've done over this series of papers is effectively paint a set of reference annotations of common human genome function," Kellis says. "Our next steps will be to personalize these maps - to basically ask how they vary naturally between individuals, by profiling different cell types from different people, and how their variation relates to human disease and complex human traits."

In one follow-up project, Kellis and colleagues are comparing activity levels of regulatory elements in different cell types from the same person, across many individuals. Another project is looking at DNA modification patterns across the entire genome of many individuals, in hopes of identifying how variation of specific elements relates to disease. *The research was funded by the National Human Genome Research Institute.*

http://www.eurekalert.org/pub_releases/2012-09/uom-eum090512.php

Exceptional upward mobility in the US is a myth, international studies show

The rhetoric is relentless: America is a place of unparalleled opportunity, where hard work and determination can propel a child out of humble beginnings into the White House, or at least a mansion on a hill.

ANN ARBOR, Mich.- But the reality is very different, according to a University of Michigan researcher who is studying inequality across generations around the world.

"Especially in the United States, people underestimate the extent to which your destiny is linked to your background. Research shows that it's really a myth that the U.S. is a land of exceptional social mobility," said

Fabian Pfeffer, a sociologist at the U-M Institute for Social Research and the organizer of an international conference on inequality across multiple generations being held Sept. 13-14 in Ann Arbor.
Pfeffer's own research illustrates this point based on data on two generations of families in the U.S. and a comparison of his findings to similar data from Germany and Sweden. The U.S. data come from the ISR Panel Study of Income Dynamics, a survey of a nationally representative sample that started with 5,000 U.S. families in 1968. He found that parental wealth plays an important role in whether children move up or down the socioeconomic ladder in adulthood. And that parental wealth has an influence above and beyond the three factors that sociologists and economists have traditionally considered in research on social mobility - parental education, income and occupation.
"Wealth not only fulfills a purchasing function, allowing families to buy homes in good neighborhoods and send their children to costly schools and colleges, for example, but it also has an insurance function, offering a sort of private safety net that gives children a very different set of choices as they enter the adult world," Pfeffer said. "Despite the widespread belief that the U.S. provides exceptional opportunities for upward mobility, these data show that parental wealth has an important role in shielding offspring from downward mobility and sustaining their upward mobility in the U.S. no less than in countries like Germany and Sweden, where parental wealth also serves as a private safety net that not even the more generous European public programs and social services seem to provide."
Pfeffer is now expanding the number of countries he is analyzing, and is also examining the influence of grandparents' wealth. Meanwhile, at the conference next week in Ann Arbor, participants from universities in Singapore, Sweden, Japan, Germany, Great Britain, South Africa and across the U.S. will share their ongoing research on different facets of intergenerational influences on inequality, from the timing of childbirth over generations to the impact grandparents, uncles and aunts have on educational attainment.
Information on Pfeffer's research: http://www-personal.umich.edu/~fpfeffer/cv_en.html
Working paper on Pfeffer's research, co-authored by Martin Hällsten, "Mobility Regimes and Parental Wealth: The United States, Germany, and Sweden in Comparison": www.psc.isr.umich.edu/pubs/abs/7676
Information on the conference, "Inequality across Multiple Generations": http://psidonline.isr.umich.edu/Publications/Workshops/Multigen2012_Agenda.pdf

http://www.eurekalert.org/pub_releases/2012-09/w-bbs090512.php

Brainy beverage: Study reveals how green tea boosts brain cell production to aid memory

Researchers have discovered how the chemical properties of China's favorite drink affect the generation of brain cells

It has long been believed that drinking green tea is good for the memory. Now researchers have discovered how the chemical properties of China's favorite drink affect the generation of brain cells, providing benefits for memory and spatial learning. The research is published in Molecular Nutrition & Food Research.
"Green tea is a popular beverage across the world," said Professor Yun Bai from the Third Military Medical University, Chongqing, China. "There has been plenty of scientific attention on its use in helping prevent cardiovascular diseases, but now there is emerging evidence that its chemical properties may impact cellular mechanisms in the brain."
Professor Bai's team focused on the organic chemical EGCG, (epigallocatechin-3 gallate) a key property of green tea. While EGCG is a known anti-oxidant, the team believed it can also have a beneficial effect against age-related degenerative diseases. "We proposed that EGCG can improve cognitive function by impacting the generation of neuron cells, a process known as neurogenesis," said Bai. "We focused our research on the hippocampus, the part of the brain which processes information from short-term to long-term memory."
The team found that EGCG boosts the production of neural progenitor cells, which like stem cells can adapt, or differentiate, into various types of cells. The team then used laboratory mice to discover if this increased cell production gave an advantage to memory or spatial learning. "We ran tests on two groups of mice, one which had imbibed EGCG and a control group," said Bai. "First the mice were trained for three days to find a visible platform in their maze. Then they were trained for seven days to find a hidden platform."
The team found that the EGCG treated mice required less time to find the hidden platform. Overall the results revealed that EGCG enhances learning and memory by improving object recognition and spatial memory.
"We have shown that the organic chemical EGCG acts directly to increase the production of neural progenitor cells, both in glass tests and in mice," concluded Bai. "This helps us to understand the potential for EGCG, and green tea which contains it, to help combat degenerative diseases and memory loss."

This paper is published as part of a collection of articles bringing together high quality research on the theme of food science and technology with particular relevance to China. Browse free articles from Wiley's food science and technology publications including the Journal of Food Science, Journal of the Science of Food and Agriculture and Molecular Nutrition & Food Research.

http://www.scientificamerican.com/article.cfm?id=biomarker-predicts-recovery-from-a-type-of-depression

Biomarker Predicts Recovery from a Type of Depression

A new study signifies the beginning of the end of psychiatrists' guess-work in figuring out which antidepressants work best for individual patients

By Amy Maxmen | Wednesday, September 5, 2012 | 1

People who live with clinical depression must also suffer the 'trial and error' approach that psychiatrists take when prescribing antidepressants. Now, a study published this week signifies the beginning of the end of guesswork. In it, a blood test predicts who will respond well to a novel treatment for depression, and who might even fare worse."We haven't had a test like this in psychiatry before," says Andy Miller, a professor of psychiatry at Emory University and an author on the study in Archives of General Psychiatry. "There is no brain scan, no physiological measure that tells you whether a patient will respond to one drug more than another."

The test identifies an inflammatory protein in blood, C-reactive protein or CRP, that indicates internal inflammation. Whereas 62% of depressed participants with high CRP levels responded well to the new treatment, only 33% of participants with low CRP levels did.

The correlation was not entirely unexpected, because the drug suppresses inflammation, and Miller thinks that inflammation underlies depression in some people. To test whether a potent anti-inflammatory could soothe the malady, his team recruited 60 people who had lived with major depression for more than a decade and had received no relief from antidepressants.

Half of the participants received monthly treatments of the rheumatoid arthritis drug, Janssen's Infliximab, and half received a placebo. Overall, Infliximab did not seem to work. However, when Miller's team analyzed how the subset of participants with high CRP faired, it turns out they responded well to the drug, with a relief from sadness, suicidal thoughts, anxiety and other symptoms.

Since the late 1980s, researchers have sporadically hypothesized that inflammation can lead to depression. The theory is that depressed behavior might be beneficial in the short term because it reserves an injured animal's energy for healing rather than romping around in the sunlight. Although the hypothesis has never received widespread support, researchers have found that some depressed patients indeed bear elevated levels of inflammatory proteins.

On the basis of the results from this relatively small study, a biologic drug such as Infliximab might be a better option in the anti-inflammatory realm than Cox-2 inhibitors such as aspirin, which come with unwanted side effects, says Miller. Although he knows of no Infliximab-like drug in development for depression, he says that companies might be encouraged by his team's results. What's more, with a biomarker to predict a response, companies will have a better chance of success.

Robert Dantzer, a neuroimmunologist at MD Anderson Cancer Center in Houston, Texas, notes that some of the participants in the low-CRP group fared worse on Infliximab than on placebo. Thus, the CRP test could be as important a tool for excluding depressed patients from taking anti-inflammatory therapies as for predicting responders.

http://bit.ly/Qs2FQC

Evolution could explain the placebo effect

ON THE face of it, the placebo effect makes no sense.

06 September 2012 by Colin Barras

Someone suffering from a low-level infection will recover just as nicely whether they take an active drug or a simple sugar pill. This suggests people are able to heal themselves unaided - so why wait for a sugar pill to prompt recovery? New evidence from a computer model offers a possible evolutionary explanation, and suggests that the immune system has an on-off switch controlled by the mind.

It all starts with the observation that something similar to the placebo effect occurs in many animals, says Peter Trimmer, a biologist at the University of Bristol, UK. For instance, Siberian hamsters do little to fight an infection if the lights above their lab cage mimic the short days and long nights of winter. But changing the lighting pattern to give the impression of summer causes them to mount a full immune response.

Likewise, those people who think they are taking a drug but are really receiving a placebo can have a response which is twice that of those who receive no pills (Annals of Family Medicine, doi.org/cckm8b). In Siberian hamsters and people, intervention creates a mental cue that kick-starts the immune response.

There is a simple explanation, says Trimmer: the immune system is costly to run - so costly that a strong and sustained response could dangerously drain an animal's energy reserves. In other words, as long as the infection is not lethal, it pays to wait for a sign that fighting it will not endanger the animal in other ways.

Nicholas Humphrey, a retired psychologist formerly at the London School of Economics, first proposed this idea a decade ago, but only now has evidence to support it emerged from a computer model designed by Trimmer and his colleagues. According to Humphrey's picture, the Siberian hamster subconsciously acts on a cue that it is summer because food supplies to sustain an immune response are plentiful at that time of year. We subconsciously respond to treatment - even a sham one - because it comes with assurances that it will weaken the infection, allowing our immune response to succeed rapidly without straining the body's resources.

Trimmer's simulation is built on this assumption - that animals need to spend vital resources on fighting low-level infections. The model revealed that, in challenging environments, animals lived longer and sired more offspring if they endured infections without mounting an immune response. In more favourable environments, it was best for animals to mount an immune response and return to health as quickly as possible (Evolution and Human Behavior, doi.org/h8p). The results show a clear evolutionary benefit to switching the immune system on and off depending on environmental conditions.

"I'm pleased to see that my theory stands up to computational modelling," says Humphrey. If the idea is right, he adds, it means we have misunderstood the nature of placebos. Farming and other innovations in the past 10,000 years mean that many people have a stable food supply and can safely mount a full immune response at any time - but our subconscious switch has not yet adapted to this. A placebo tricks the mind into thinking it is an ideal time to switch on an immune response, says Humphrey.

Paul Enck at the University of Tübingen in Germany says it is an intriguing idea, but points out that there are many different placebo responses, depending on the disease. It is unlikely that a single mechanism explains them all, he says.

http://www.eurekalert.org/pub_releases/2012-09/uab-ftr090612.php

Favorite TV reruns may have restorative powers, says UB researcher

What if watching TV under specific conditions could actually provide a mental boost?

BUFFALO, N.Y. -- We hear all the time that we need to get off the couch, stop watching TV and get moving. But what if watching TV under specific conditions could actually provide the mental boost you need to tackle a difficult task?

A new paper that describes two studies by Jaye Derrick, PhD, research scientist at the University at Buffalo's Research Institute on Addictions, found that watching a rerun of a favorite TV show may help restore the drive to get things done in people who have used up their reserves of willpower or self-control.

"People have a limited pool of these valuable mental resources," explains Derrick. "When they use them on a task, they use up some of this limited resource. Therefore, they have less willpower and self-control for the next task. "With enough time, these mental resources will return. However, there may be ways to more quickly restore them."

One of these ways is to re-watch your favorite TV show, Derrick's research found. Doing so, she says, taps into the surrogate relationship people form with the characters in their favorite shows. We find it comforting, mainly because we already know what the characters are going to say and do. All we have to do is sit back and enjoy it.

"When you watch a favorite re-run, you typically don't have to use any effort to control what you are thinking, saying or doing. You are not exerting the mental energy required for self-control or willpower," Derrick explains. "At the same time, you are enjoying your 'interaction,' with the TV show's characters, and this activity restores your energy."

In the first of her two studies published in the journal Social Psychological and Personality Science, Derrick asked half of the participants to complete a structured task which required concentrated effort. The other half were asked to complete a similar but less structured task that allowed them more freedom and required much less effort. Then half of the participants were asked to write about their favorite television show while the other half listed items in their room (a "neutral" task). Following this, the participants were tested to measure any reduction or renewal of willpower.

Those who wrote about their favorite television show (rather than listing items in their room) wrote for longer if they had done the structured task than if they had done the less-structured task. This, Derrick says, indicates these participants were seeking out their favorite TV shows and they wanted to spend more time thinking about

them. And writing about their favorite television show restored their energy levels and allowed them to perform better on a difficult puzzle.
In the second study, participants kept a daily diary. They reported on their effortful tasks, media consumption and energy levels each day. If they had to do effortful tasks, they were more likely to seek out a re-run of their favorite television show, to re-watch a favorite movie or to re-read a favorite book. Doing so, then restored their energy levels. "In other words, there was a measurable restorative effect from a familiar fictional world," Derrick finds. But that doesn't mean people should veg-out in front of any TV show.
"The restorative effect I found is specific to re-watching favorite television shows (or re-watching favorite movies or re-reading favorite books)," Derrick says. "Just watching whatever is on television does not provide the same benefit. And perhaps surprisingly, watching a new episode of a favorite television show for the first time does not provide the same benefit."
Derrick explains that there is something special and comfortable about a "relationship" in which you already know what the other person is going to say and do, and all you have to do is sit there and enjoy it.
In fact, the effects of this fictional "social surrogacy" may work better than actual social interaction with real people under some circumstances. "Although there are positive outcomes to social interaction such as a sense of feeling of being energized," says Derrick, "human exchanges can also produce a sense of rejection, exclusion and ostracism, which may diminish willpower."
Derrick's findings may dispel some notions that watching TV is bad for us.
"Based on my research, I would argue that watching television is not all bad. While there is a great deal of research demonstrating that violent television can increase aggression, and watching television may be contributing to the growing obesity epidemic, watching a favorite television show can provide a variety of benefits, which may enhance overall wellbeing," she says.
Derrick's new research will expand on these findings and examine other social consequences of television.
"I have found, for example, that favorite television shows can actually increase people's pro-social behavior. Specifically, after thinking about a favorite television show, people are more willing to forgive others, are more willing to help a stranger and are more willing to sacrifice for their romantic partner," she says.

http://www.eurekalert.org/pub_releases/2012-09/cshl-so090612.php

Storm of 'awakened' transposons may cause brain-cell pathologies in ALS, other illnesses

Important progress in an effort to understand the relationship between transposons – sequences of DNA that can jump around within the genome, potentially causing great damage

Cold Spring Harbor, NY – A team of neuroscientists and informatics experts at Cold Spring Harbor Laboratory (CSHL) reports important progress in an effort to understand the relationship between transposons – sequences of DNA that can jump around within the genome, potentially causing great damage – and mechanisms involved in serious neurodegenerative disorders including ALS (amyotrophic lateral sclerosis, also known as Lou Gehrig's disease), FTLD (frontotemporal lobar degeneration) and Alzheimer's disease.
A close analysis of previously unanalyzed genome data has led CSHL Associate Professor Joshua Dubnau, Ph.D., and colleagues to discover a signature of disease that may help explain these and other neural pathologies. As reported by the team September 6 in the journal PloS One, this signature leads them to hypothesize that dormant transposons awaken in the genome, setting off the equivalent of a transposon storm in some brain cells, capable of causing cell death.
Transposons – often called transposable elements, or TEs, by scientists – are understood collectively to occupy a large fraction (~40%) of the genetic material of multicellular organisms, including humans. Most TEs are genomic fossils, effectively inactive. A minority of TEs capable of activation are ordinarily suppressed by a variety of cellular defense mechanisms that have evolved along with life over eons of time to prevent sudden rearrangements (i.e., mutations) of the genetic material.
Dubnau's team, in collaboration with the team of CSHL Assistant Professor Molly Hammell, Ph.D., a bioinformatics expert and co-senior author of the new paper, were intrigued by recent research indicating that some transposable elements are active in brain cells during neurogenesis, the process in which new neurons are born, as well as during normal brain development. They postulate that mobile chunks of DNA somehow evade the mechanisms that normally restrain them, in certain neuropathologies. For this reason they decided to comb vast datasets for evidence of a particular kind of interaction: the binding of a protein called TDP-43 to RNA "messages" copied from portions of the genome occupied by TEs.
TDP-43 is a versatile protein, known to bind to both DNA and RNA in cells. Accumulations of proteins and RNAs that prominently include TDP-43 are known to have a causal role in ALS pathology. TDP-43 is also known to be associated with FTLD, a condition in which frontal and temporal lobes of the brain atrophy.

"Accumulations of TDP-43 in what we call 'inclusion bodies' within the cell's cytoplasm is a shared pathological hallmark in a broad spectrum of neurodegenerative disorders including in ALS and FTLD," Dubnau says.

The CSHL scientists noted that TDP-43 was bound to transposon-derived RNAs across three independent, publicly archived genomic datasets from rat, mouse and human samples. Separate research revealed that this association of the binding protein and TE-derived transcripts was less common in the brain cells of FTLD patients, relative to healthy people. They also observed in two different mouse models with TDP-43 dysfunction that the number of observed TE-derived RNA messages was significantly elevated.

"Putting all these observations together," Dubnau says, "we are now suggesting that TDP-43 normally functions to silence or repress the expression of potentially harmful transposons. When TDP-43 function is compromised, these mobile elements become overexpressed. And this, we hypothesize, may contribute to toxic effects underlying pathologies we see in ALS, FTLD, and perhaps other neurodegenerative conditions."

Current research by the CSHL team aims to flesh out these relationships, and particularly to determine, in Dubnau's words, "whether the unleashing of transposons is a cause or a consequence of the neurodegeneration that we know to be associated with TDP-43 accumulation, in these devastating illnesses."

"Transposable Elements in TDP-43-mediated Neurodegenerative Disorders" appears September 6, 2012 in PloS One. The authors are: W. Li, Y. Lin, L. Prazak, M. Hammell and J. Dubnau. The paper can be read online at: http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0044099

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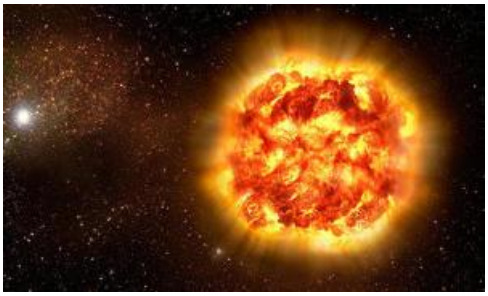
http://www.sciencedaily.com/releases/2012/09/120906074025.htm

In Quest of the Cosmic Origins of Silver: Silver and Gold Materialized in Different Stellar Explosions

Silver can only have materialised during the explosion of clearly defined types of stars which are different from the kind of stars producing gold



At the end of their lives, stars with ten times the mass of our sun explode as so-called supernovae. In the process, elements like silver are either hurled out into the universe or produced in the first place. The illustration is an artist's impression of the first moments of such an explosion before the star is completely torn apart. **European Southern Observatory/ESO**

ScienceDaily - In the quest for the cosmic origins of heavy elements, Heidelberg scientist Dr. Camilla Hansen has established that silver can only have materialised during the explosion of clearly defined types of star. These are different from the kind of stars producing gold when they explode. The evidence for this comes from the measurement of various high-mass stars with the help of which the stepwise evolution of the components of all matter can be reconstructed.

The findings from the investigations conducted by Dr. Hansen of Heidelberg University's Centre for Astronomy (ZAH) in conjunction with other scientists in Germany and fellow astronomers in Japan and Sweden have been published in the journal Astronomy & Astrophysics.

The lightweight elements hydrogen, helium and traces of lithium came into being a few minutes after the Big Bang. All heavier elements materialised later in the interior of stars or during star explosions, with each generation of stars contributing a little to enriching the universe with chemical elements. The elements a star can generate in its lifetime depend largely on its mass. At the end of their lives, stars about ten times the size of our sun explode as so-called supernovae, producing elements sometimes heavier than iron that are released by the explosion. Depending on how heavy the star originally was, silver and gold can also materialise in this way. When various stars of the same mass explode, the ratio of elements generated and hurled out into the universe is identical. This constant relation is perpetuated in the subsequent generations of stars forming from the remnants of their predecessors. The investigations by Dr. Hansen and her associated scientists have now demonstrated that the amount of silver in the stars measured is completely independent of the amounts of other heavy elements like gold. These observations indicate clearly for the first time that during a supernova silver takes shape in an entirely different fusion process from that in which gold forms. Accordingly, the scientists contend that silver cannot have originated together with gold. The elements must have materialised from stars of different masses.

"This is the first incontrovertible evidence for a special fusion process taking place during the explosion of a star," says Dr. Hansen. "Up to now this had been mere speculation. After this discovery, we must now use simulations of these processes in supernova explosions to investigate more precisely when the conditions for

the formation of silver are present. That way we can find out how heavy the stars were that could produce silver during their dramatic demise."

http://www.sciencedaily.com/releases/2012/09/120906084306.htm

Plants Cry for Help When an Attack Can Be Expected

Eggs of insect pests deposited on plants trigger the production of scents by plants that affect different plant community members probably helping the plant to get rid of the pest before it becomes harmful.

ScienceDaily - These results are reported the journal PLoS ONE by researchers, of the Laboratory of Entomology of Wageningen University and the Netherlands Institute of Ecology (NIOO-KNAW).

The research team, led by Nina Fatouros, tested how parasitic wasps, natural enemies of a common cabbage pest, the large cabbage white butterfly, and gravid butterfly females respond to black mustard, a cabbage relative, emitting scents during the initial phase of herbivore attack, when eggs are laid.

They show that butterfly egg deposition triggers highly specific chemical and structural changes in the plant that attract different parasitic wasps attacking either butterfly eggs or caterpillars but repel egg-laying butterflies. However, egg deposition by a less common pest, the cabbage moth, does not trigger such changes. A specific plant response to butterfly egg deposition might help the plant defending itself before actual damage by hatching caterpillars starts.

Nina E. Fatouros, Dani Lucas-Barbosa, Berhane T. Weldegergis, Foteini G. Pashalidou, Joop J. A. van Loon, Marcel Dicke, Jeffrey A. Harvey, Rieta Gols, Martinus E. Huigens. Plant Volatiles Induced by Herbivore Egg Deposition Affect Insects of Different Trophic Levels. PLoS ONE, 2012; 7 (8): e43607 DOI: 10.1371/journal.pone.0043607

http://blogs.scientificamerican.com/guest-blog/2012/09/06/tapeworm-in-her-brain/

Brain Parasites, California's Hidden Health Problem

Sara Alvarez was afraid. The doctors told her she needed surgery - brain surgery.

By Mollie Bloudoff-Indelicato | September 6, 2012

Operations on such a complex organ are never simple, but this procedure was exceptionally difficult. There was a high risk of complications, of debilitation, of post-op problems. Alvarez might wake up paralyzed. She might wake up legally blind. Worse still, there was a chance she might not wake up at all.

Her mad dash to the emergency room had all begun with a walk in the park four days earlier. It was December 20, 2010, in Sunnyvale, Calif., a town that lives up to its name. The West Coast winter, not as long or as harsh as seasons in the East, gave her the opportunity to take her youngest child out for an afternoon stroll. In the fading light of dusk, Alvarez, too, began to fade. She lost the feeling in her right leg. Her right foot followed suit. She couldn't lift or move her right hand. She was weak, and her body was numb. There was fear then, too. At 10:15 p.m., Alvarez says her husband drove her to Redwood City. That night she became a patient at Kaiser Permanente Redwood City Hospital. She says the doctors batted diagnoses back and forth. It was a tumor. No, it was cancer.

It was Christmas, and Alvarez's children cried and prayed, terrified that an unknown affliction would steal their mother away. Finally a CT scan revealed the malady. Alvarez had neurocysticercosis - a calcified tapeworm lodged in her brain.

Neurocysticercosis, which is common around the world but is not recognized as a major health concern in the U.S., has taken root in California, some health officials say. The disease is easy to prevent and relatively inexpensive to treat if caught early on. But once in the advanced stages, these brain parasites are costly to both patient and government.

The problem is that, due to a lack of education, most of the population doesn't know that there's a parasite wriggling within them, says Patricia Wilkins, a scientist with the Center for Disease Control and Prevention (CDC). Latinos, the community most afflicted by the disease, do not receive outreach or education about how to avoid or treat the potentially life-threatening organism, Wilkins adds.

Neurocysticercosis "primarily exists in marginalized populations, Hispanic immigrants," Wilkins adds.

The National Institutes of Health classifies neurocysticercosis as the leading cause of epilepsy worldwide, and the World Health Organization (WHO) estimates that tapeworms infect 50 million people globally. The CDC says an estimated 1,900 people are diagnosed with neurocysticercosis within the United States yearly.

According to a January 2012 study in PLOS Neglected Tropical Diseases, California bears much of the burden with 304 hospitalized cases in 2009, the most recent year for which statistics exist. Eighty-five percent of patients in California were identified as Latino, and 72 percent were reported in the southern half of the state.

The high percentage of Latino cases is not surprising. Neurocysticercosis is common within third-world countries in Asia, Africa and Latin America. The disease's telltale symptoms of paralysis, extreme headaches

and chronic seizures present themselves in mass form. Individuals contract neurocysticercosis after becoming infected by tapeworm carriers. Immigrants traveling between countries, such as migrant workers, are often unwitting tapeworm hosts, transporting the disease across borders in their guts.

Scientists aren't quite sure how it works, but tapeworm larvae seem to have developed a chemical secretion that keeps the human body's immune system from barging in on their banquet. People can live for decades without any symptoms of neurocysticercosis because the tapeworm larvae break down natural defenses. Unfortunately, tapeworm larvae can't live forever.

"While it's alive, it's a problem, but when it starts to die it's a bigger problem," Despommier says.

When the larvae die, the chemical balance is restored, and the immune system begins to attack, causing headaches, seizures and paralysis. Alvarez says she experienced debilitating headaches for 20 years before her diagnosis, but she probably consumed tapeworm eggs much earlier than that. When Alvarez immigrated to the United States in the late 1980s she complained to American doctors of a pain so absolute it blinded her and made her vomit. They gave her Tylenol.

"That's a very typical story," says Darvin Scott Smith, chief of infectious disease at the Kaiser Hospital.

Many physicians, even those in highly populated areas sizable immigrant populations, are unaware of the disease and how to diagnose it, he adds. Even many of the health organizations that target Latinos had never heard of neurocysticercosis and said their institutions were not funding research or community outreach.

Nobody cares about this disease, and they should, if not from a humanitarian point of view than from a fiscal aspect, says Wilkins, a scientist with the CDC. Drugs such as Ablendazole and certain steroids, which are used to treat tapeworms and brain swelling, are relatively inexpensive - a maximum of a few hundred dollars. Wait until it's a serious problem, though, and the dollar amount rises dramatically.

The CDC reports the average cost of neurocysticercosis at $37,600 per hospitalization.

The most common form of payment is Medicaid, a tax-funded public service. In Los Angeles County, the economic impact is even more pronounced, costing $66,000 on average, the increase likely due to the high cost of health care in the state, says Frank Sorvillo, a University of Los Angeles professor of epidemiology.

Despite a marked decrease in immigration over the past few years, the number of neurocysticercosis cases has remained relatively constant since 2001, when there were 386 recorded hospitalizations in California. This suggests that the parasite has taken hold in the U.S., Sorvillo says.

These numbers are likely underestimated. Only five states — New York, California, Texas, Oregon and Illinois — report the disease, and the data is inconsistent. Oftentimes, departments rely on each other to deal with paperwork, and the numbers are never recorded, Smith says. As a result, not much is known about tapeworm outbreaks in the U.S. — or the parasites themselves. Scientists still consider much of their life cycle a mystery.

Pork tapeworms, or Taenia solium, are complex organisms. They exist in three life stages: egg, larvae and adult, but their growth is not a straight progression from one form to the next. Tapeworm larvae enter the body when humans eat contaminated pork.

The babies, about the size of peas, fight their way into the small intestine and attach, using rows of grappling hook-like teeth to make tiny slices into the soft flesh of the intestinal walls. The parasites cling to the slippery surfaces of their new homes and begin draining nutrients from their host. If all goes well, adults can grow up to 20 feet long.

It sounds unpleasant, but if you're going to contract a tapeworm, dealing with 20 feet of invertebrate is really the way to go. Researchers say that adult Taenia solium is relatively harmless and asymptomatic. The real trouble starts when they begin to reproduce within their human host.

Tapeworm adults are made up of hundreds of segments called proglottids. The parasite grows like a fingernail, the newest addition at the head and old material at the tip. The senior proglottids contain eggs — thousands of them. During the course of a natural lifecycle, the proglottids are discarded through their host's anus. A family member, friend or restaurant cook infected with an adult tapeworm can secrete tens of thousands of tapeworm eggs daily, which can be easily ingested by others.

Being infected with the eggs, however, doesn't result in an adult tapeworm. The eggs just develop into larvae— and grow no further. According to parasitologist Judy Sakanari at the University of California, San Francisco, no one really knows why. Unlike most animals whose lifecycle follows a child-adolescent-adult pattern, these eggs will never mature into adulthood. Their development is stunted at the larvae stage, which allows them to ride the bloodstream. They use their hooks to rip apart tissue and gain access to nutrient-rich hotspots. Some of these miniature reapers ultimately find their way into the brain. That's where the trouble starts — and stops.

While alive, the larvae are not as dangerous as they are when they're dead. The brain calcifies the dead larvae, and, oftentimes, surgery is necessary to remove them. This ramps up costs for the hospital and drains Medicaid

funds. The State of California is not responding to the issue, Wilkins says, because there isn't enough funding to tackle every bug that infiltrates a community. Health officials must pick and choose which diseases require the most resources. So far, neurocysticercosis has not been one of them.

In a 2000 proposal filed by the WHO, doctors called for international monitoring of neurocysticercosis. They argued that surveillance was key to eradication, that statistics were paramount if governments across the globe had any hope of reducing epilepsy and increasing quality of life. So far, the petition has not experienced much success.

In early January 2011, Dr. Smith of Redwood City, Calif. celebrated his birthday in the operating room of Kaiser Hospital, observing Sara Alvarez's brain surgery. Medical professionals trimmed Sara's hair, gingerly peeled away layers of skin and cut through a portion of her skull. Hours later, the chief of infectious disease watched as a neurosurgeon plucked a calcified tapeworm larvae from Sara's head.

Before she was diagnosed, Alvarez had never heard of neurocysticercosis, and she still isn't sure who gave her the eggs. It could have been a chance encounter, or one of her loved ones might be a carrier. She'll never know for sure. The host may remain undetected and contagious, spreading the disease — thousands of eggs at a time.

Story and images by permission of Sara Alvarez and Dr. Darvin Scott Smith

http://www.sciencedaily.com/releases/2012/09/120906123324.htm

'I Knew It All Along ... Didn't I?' -- Understanding Hindsight Bias

The situation may be different each time, but we hear ourselves say it over and over again: "I knew it all along."

ScienceDaily - The fourth-quarter comeback to win the game. The tumor that appeared on a second scan. The guy in accounting who was secretly embezzling company funds. The situation may be different each time, but we hear ourselves say it over and over again: "I knew it all along."

The problem is that too often we actually didn't know it all along, we only feel as though we did. The phenomenon, which researchers refer to as "hindsight bias," is one of the most widely studied decision traps and has been documented in various domains, including medical diagnoses, accounting and auditing decisions, athletic competition, and political strategy.

In a new article in the September 2012 issue of Perspectives on Psychological Science, a journal of the Association for Psychological Science, psychological scientists Neal Roese of the Kellogg School of Management at Northwestern University and Kathleen Vohs of the Carlson School of Management at the University of Minnesota review the existing research on hindsight bias, exploring the various factors that make us so susceptible to the phenomenon and identifying a few ways we might be able to combat it. This article is the first overview to draw insights together from across different disciplines.

Roese and Vohs propose that there are three levels of hindsight bias that stack on top of each other, from basic memory processes up to higher-level inference and belief. The first level of hindsight bias, memory distortion, involves misremembering an earlier opinion or judgment ("I said it would happen"). The second level, inevitability, centers on our belief that the event was inevitable ("It had to happen"). And the third level, foreseeability, involves the belief that we personally could have foreseen the event ("I knew it would happen").

The researchers argue that certain factors fuel our tendency toward hindsight bias. Research shows that we selectively recall information that confirms what we know to be true and we try to create a narrative that makes sense out of the information we have. When this narrative is easy to generate, we interpret that to mean that the outcome must have been foreseeable. Furthermore, research suggests that we have a need for closure that motivates us to see the world as orderly and predictable and to do whatever we can to promote a positive view of ourselves.

Ultimately, hindsight bias matters because it gets in the way of learning from our experiences.

"If you feel like you knew it all along, it means you won't stop to examine why something really happened," observes Roese. "It's often hard to convince seasoned decision makers that they might fall prey to hindsight bias."

Hindsight bias can also make us overconfident in how certain we are about our own judgments. Research has shown, for example, that overconfident entrepreneurs are more likely to take on risky, ill-informed ventures that fail to produce a significant return on investment.

While our inclination to believe that we "knew it all along" is often harmless, it can have important consequences for the legal system, especially in cases of negligence, product liability, and medical malpractice. Studies have shown, for example, that hindsight bias routinely afflicts judgments about a defendant's past conduct.

And technology may make matters worse. "Paradoxically, the technology that provides us with simplified ways of understanding complex patterns -- from financial modeling of mortgage foreclosures to tracking the flow of communications among terrorist networks -- may actually increase hindsight bias," says Roese.
So what, if anything, can we do about it?
Roese and Vohs suggest that considering the opposite may be an effective way to get around our cognitive fault, at least in some cases. When we are encouraged to consider and explain how outcomes that didn't happen could have happened, we counteract our usual inclination to throw out information that doesn't fit with our narrative. As a result, we may be able to reach a more nuanced perspective of the causal chain of events.

http://www.sciencedaily.com/releases/2012/09/120906141119.htm

Nutritional Supplement Offers Promise in Treatment of Unique Form of Autism

Researcher, have identified a form of autism with epilepsy that may potentially be treatable with a common nutritional supplement.

ScienceDaily - An international team of researchers, led by scientists at the University of California, San Diego and Yale University schools of medicine, have identified a form of autism with epilepsy that may potentially be treatable with a common nutritional supplement.
The findings are published in the Sept. 6, 2012 online issue of Science.
Roughly one-quarter of patients with autism also suffer from epilepsy, a brain disorder characterized by repeated seizures or convulsions over time. The causes of the epilepsy are multiple and largely unknown. Using a technique called exome sequencing, the UC San Diego and Yale scientists found that a gene mutation present in some patients with autism speeds up metabolism of certain amino acids. These patients also suffer from epileptic seizures. The discovery may help physicians diagnose this particular form of autism earlier and treat sooner.
The researchers focused on a specific type of amino acid known as branched chain amino acids or BCAAs. BCAAs are not produced naturally in the human body and must be acquired through diet. During periods of starvation, humans have evolved a means to turn off the metabolism of these amino acids. It is this ability to shut down that metabolic activity that researchers have found to be defective in some autism patients.
"It was very surprising to find mutations in a potentially treatable metabolic pathway specific for autism," said senior author Joseph G. Gleeson, MD, professor in the UCSD Department of Neurosciences and Howard Hughes Medical Institute investigator. "What was most exciting was that the potential treatment is obvious and simple: Just give affected patients the naturally occurring amino acids their bodies lack."
Gleeson and colleagues used the emerging technology of exome sequencing to study two closely related families that have children with autism spectrum disorder. These children also had a history of seizures or abnormal electrical brain wave activity, as well as a mutation in the gene that regulates BCAAs. In exome sequencing, researchers analyze all of the elements in the genome involved in making proteins.
In addition, the scientists examined cultured neural stem cells from these patients and found they behaved normally in the presence of BCAAs, suggesting the condition might be treatable with nutritional supplementation. They also studied a line of mice engineered with a mutation in the same gene, which showed the condition was both inducible by lowering the dietary intake of the BCAAs and reversible by raising the dietary intake. Mice treated with BCAA supplementation displayed improved neurobehavioral symptoms, reinforcing the idea that the approach could work in humans as well.
"Studying the animals was key to our discovery," said first author Gaia Novarino, PhD, a staff scientist in Gleeson's lab. "We found that the mice displayed a condition very similar to our patients, and also had spontaneous epileptic seizures, just like our patients. Once we found that we could treat the condition in mice, the pressing question was whether we could effectively treat our patients."
Using a nutritional supplement purchased at a health food store at a specific dose, the scientists reported that they could correct BCAA levels in the study patients with no ill effect. The next step, said Gleeson, is to determine if the supplement helps reduce the symptoms of epilepsy and/or autism in humans.
"We think this work will establish a basis for future screening of all patients with autism and/or epilepsy for this or related genetic mutations, which could be an early predictor of the disease," he said. "What we don't know is how many patients with autism and/or epilepsy have mutations in this gene and could benefit from treatment, but we think it is an extremely rare condition."
Co-authors are Paul El-Fishawy, Child Study Center, Yale University School of Medicine; Hulya Kayserili, Medical Genetics Department, Istanbul University, Turkey; Nagwa A. Meguid, Rehab O. Khalil, Adel F. Hashish and Hebatalla S. Hashem, Department of Research on Children with Special Needs, National Research Centre, Cairo, Egypt; Eric M. Scott, Jana Schroth, Jennifer L. Silhavy, Neurogenetics Laboratory, Howard Hughes Medical Institute, Department of Neurosciences, UC San

Diego; Majdi Kara, Pediatric Department, Tripoli Children's Hospital, Libya; Tawfeq Ben-Omran, Clinical and Metabolic Genetics Division, Department of Pediatrics, Hamad Medical Corporation, Doha, Qatar; A. Gulhan Ercan-Sencicek, Stephan J. Sanders and Matthew W. State, Program on Neurogenetics, Child Study Center, Department of Psychiatry and Department of Genetics, Yale University School of Medicine; Abha R. Gupta, Child Study Center, Department of Pediatrics, Yale University School of Medicine; Dietrich Matern, Biochemical Genetics Laboratory, Department of Laboratory Medicine and Pathology, Mayo Clinic; Stacy Gabriel, Broad Institute of Harvard and Massachusetts Institute of Technology; Larry Sweetman, Institute of Metabolic Disease, Baylor Research Institute; Yasmeen Rahimi and Robert A. Harris, Roudebush VA Medical Center and Department of Biochemistry and Molecular Biology, Indiana University School of Medicine.
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Gaia Novarino, Paul El-Fishawy, Hulya Kayserili, Nagwa A. Meguid, Eric M. Scott, Jana Schroth, Jennifer L. Silhavy, Majdi Kara, Rehab O. Khalil, Tawfeg Ben-Omran, A. Gulhan Ercan-Sencicek, Adel F. Hashish, Stephan J. Sanders, Abha R. Gupta, Hebatalla S. Hashem, Dietrich Matern, Stacey Gabriel, Larry Sweetman, Yasmeen Rahimi, Robert A. Harris, Matthew W. State, and Joseph G. Gleeson. Mutations in BCKD-kinase Lead to a Potentially Treatable Form of Autism with Epilepsy. Science, 2012; DOI: 10.1126/science.1224631

http://www.wired.com/wiredscience/2012/09/tiny-carboniferous-steps/

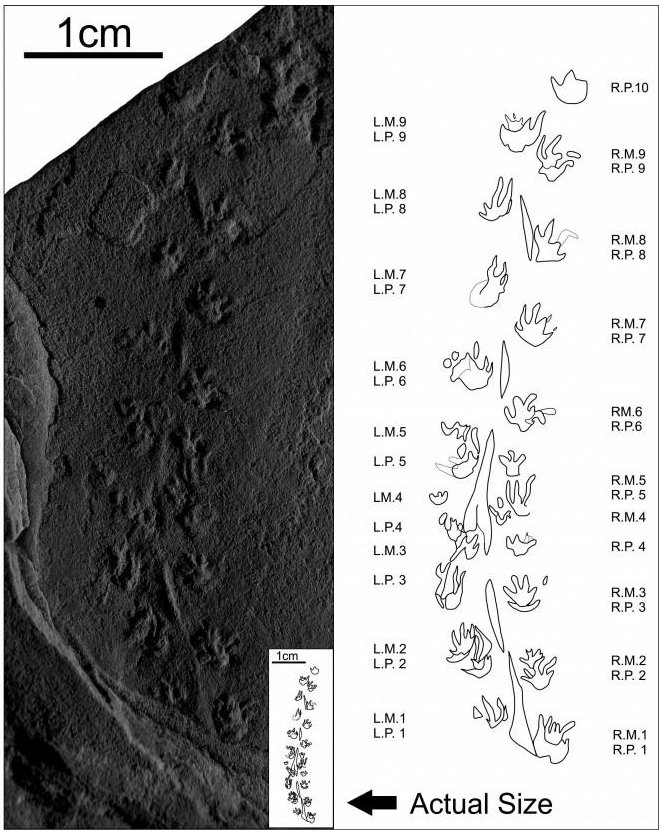
Tiny Carboniferous Steps

The smallest fossil vertebrate footprints yet found.

By Brian Switek

Fossil footprints are lovely vestiges of prehistoric life. Even though skeletons are wonderful things, and immense mounts of defleshed dinosaurs were what inspired my love of paleontology in the first place, the variety of fossil tracks paleontologists have collected and cataloged are intricate snapshots of life in motion. Imprints and trackways record movement and behavior – petrified outlines of prehistoric moments that we can't observe directly. And it's this variety of fossil that allowed paleontologists to follow a tiny 315 million year old tetrapod on a little jaunt.

Discovered by amateur paleontologist Gloria Melanson along Nova Scotia's Joggins Cliffs, the tiny trackway contains what Melanson, Matt Stimson, and Spencer Lucas deem to be the smallest fossil vertebrate footprints yet found. The whole trackway, containing about 30 footprints, is just under two inches long, and the imprints themselves range from 0.09 to 0.06 of an inch long each. This was a tiny, tiny tetrapod that strutted along a floodplain during a time when insects were giants and our own ancestors were small, lizard-like creatures. Based on the details of the tracks, it seems that the vertebrate was walking at a normal pace before slightly speeding up toward the end of the slab. But just what sort of animal made the tracks?



The tiny Batrachichnus salamandroides trackway found in Nova Scotia. Image courtesy Justin Spielmann.

Students of trace fossils – technically known as ichnologists – constantly face what paleontologist Martin Lockley has called the "Cinderella Syndrome." Unless an organism literally dies in its tracks, paleontologists face the problem of matching the trace to the trace-maker. Indeed, the situations that preserved body fossils weren't always amenable to setting trace fossils in stone, and vice versa. And even in places like the Joggins Cliffs, where body and trace fossils are both found, many of the body fossils are fragments preserved in different settings than the footprints. In this case, what we know about the skeletons of the Joggins Cliffs animals comes from bones found inside the fossilized trunks of tree-like lycopsids – vascular plants most closely related to today's club mosses and quillworts.

A restoration of the kind of amphibian that may have made the Batrachichnus tracks, courtesy Justin Spielmann.

Stimson and co-authors attribute the tracks to the ichnospecies Batrachichnus salamandroides. This is the name for the particular track type, but not the animal itself. (Just like body fossils, trace fossils are organized according to a binomial system which refers to specific trace fossil forms.) But the paleontologists also narrow down the list of possible creatures that could have made the Batrachichnus salamandroides footprints. Their prime candidate is a juvenile Dendrerpeton– a roughly salamanderish amphibian known from an articulated skeleton and other fossils found in the ancient lycopsid stumps. The crawler's proportions, when shrunk down to scale, seem to match the proportions of the trackmaker.

Then again, Dendrerpeton isn't the only suitable candidate. A different variety of amphibian, called a microsaur, could have made similar tracks. As Stimson and colleagues conclude, "multiple biotaxa of varying sizes could produce Batrachichnus salamandroides tracks." We may never know for sure who left the tracks. The trail of itty bitty footprints can only take us so far into the past.

Matt Stimson, Spencer G. Lucas & Gloria Melanson (2012): The Smallest Known Tetrapod Footprints: Batrachichnus salamandroides from the Carboniferous of Joggins, Nova Scotia, Canada, Ichnos: An International Journal for Plant and Animal Traces, 19:3, 127-140

http://www.eurekalert.org/pub_releases/2012-09/uab-abc090712.php

Ancient, bottom-dwelling critter proves: Newer isn't always better

Tiny, humble rhabdopleurids have lived on the ocean floor for some 500 million years, outlasting more elaborate descendants

This is a Rhabdopleura compacta zooid. A new study identifies rhabdopleurids as an ancestor of more elaborate species that have since died off. **Dr. Atsuko Sato, University of Oxford**

BUFFALO, N.Y. -- Tiny sea creatures called rhabdopleurids reside on the ocean floor, building homes of collagen on the shells of dead clams. Rhabdopleurid colonies are small, and the critters are by no means the dominant animals in their ecosystem. But they have lived this way -- and survived -- for more than 500 million years. And in doing so, they have outlasted more elaborate species that also descended from a common ancestor, according to a new study in the journal Lethaia.

Though rhabdopleurids' age and modern existence are well-documented, the paper breaks new ground by identifying them as a predecessor to ancient zooplankton -- known as pelagic graptolites -- that went extinct about 350 million years ago. The lesson, according to lead author Charles Mitchell: Newer isn't always better.

"We think that change is always going to lead us to a better place, that evolution is always going to lead to something better," said Mitchell, a

University at Buffalo geology professor. "But all this progress in making all these wonderful pelagic graptolites didn't lead them to take over the world. They didn't survive, but these simple dudes, these bottom-dwelling creatures, did."

Mitchell's partners on the research included Michael J. Melchin from St. Francis Xavier University in Nova Scotia, Canada; Chris B. Cameron of the Université de Montréal; and Jörg Maletz from the Frei Universität Berlin. The paper, which appeared online on Aug. 2, used rhabdopleurids' structure and form to determine that they were some of the most primitive graptolites that ever existed.

While their zooplankton relatives evolved rapidly, splitting into many new species and evolving many new traits, rhabdopleurids pretty much stayed the same over the course of history. As the zooplankton developed ways to live closer to the ocean's surface, the rhabdopleurids continued dwelling on the ocean floor. The zooplankton became important players in their new ecosystems. The rhabdopleurids remained inconspicuous. Ultimately, the conservative approach won out: The rhabdopleurids survived and are still around today, living in areas from Bermuda to the Bering Sea. The zooplankton graptolites went extinct.

"High speciation rates generally go hand in hand with high extinction rates, and likewise low with low," Mitchell said. "Conservative lineages may weather the storms of climate change and other events, but do not become big parts of the ecosystem, whereas the major players are impressive but often brought low by mass extinction and other 'slings and arrows of outrageous fortune.'"

The idea that conservative approaches can bear rewards over time is one that holds true not only in biology, but in other fields of study as well, Mitchell said. He pointed to financial markets as one example.

"You can pick 'safe' investments like bonds and blue chip stocks, and so expose your money to low risk of decline in values, but the yield is low, as well: Values do not grow much," Mitchell said. "On the other hand

one can pick high-yield tech stocks like Facebook and Apple, but the risk of declines in value, especially in bad economic times, is also high."
Though humble, rhabdopleurids and the colonies they build are beautiful to behold under a microscope. The creatures themselves are about a millimeter long and Y-shaped, with a pair of tentacled arms extending from a narrow body to filter food from the water. The colonies they fashion are whimsical-looking structures, consisting of a network of copper-colored tubes that resemble tiny elephant trunks, each one bearing numerous ridges.
The knowledge that rhabdopleurids are ancient graptolites will enable researchers to gain insight into poorly understood aspects of graptolite biology. Studying rhabdopleurids could reveal new clues about how early graptolites looked and reproduced, and even what they ate.
Support for the research that appeared in Lethaia came from the Natural Sciences and Engineering Research Council of Canada Discovery Grant program, the U.S. National Science Foundation and the St. Francis Xavier University James Chair Visiting Professorship. The paper is titled "Phylogenetic analysis reveals that Rhabdopleura is an extant graptolite."

http://www.eurekalert.org/pub_releases/2012-09/haoq-twf090712.php

Treatment with fungi makes a modern violin sound like a Stradiavarius

A good violin depends not only on the expertise of the violin maker, but also on the quality of the wood that is used.

The Swiss wood researcher Professor Francis W. M. R. Schwarze (Empa, Swiss Federal Laboratories for Materials Science and Technology, St. Gallen, Switzerland) has succeeded in modifying the wood for a violin through treatment with special fungi. This treatment alters the acoustic properties of the instrument, making it sound indistinguishably similar to a Stradivarius. In his dinner talk at the 1st ECRC "Franz-Volhard" Symposium of the Max Delbrück Center for Molecular Medicine (MDC) and Charité - Universitätsmedizin on September 7, 2012 in Berlin-Buch, Schwarze reported on his research and gave a preview of what his wood treatment method could mean, particularly for young violinists.
Low density, high speed of sound and a high modulus of elasticity – these qualities are essential for ideal violin tone wood. In the late 17th and early 18th century the famous violin maker Antonio Stradivari used a special wood that had grown in the cold period between 1645 and 1715. In the long winters and the cool summers, the wood grew especially slowly and evenly, creating low density and a high modulus of elasticity. Until now, modern violin makers could only dream of wood with such tonal qualities.
Professor Schwarze's developments could soon make similarly good wood available for violin making. He discovered two species of fungi (Physisporinus vitreus and Xylaria longipes), which decay Norway spruce and sycamore – the two important kinds of wood used for violin making – to such an extent that their tonal quality is improved. "Normally fungi reduce the density of the wood, but at the same time they unfortunately reduce the speed with which the sound waves travel through the wood," the researcher explained. "The unique feature of these fungi is that they gradually degrade the cell walls, thus inducing a thinning of the walls. But even in the late stages of the wood decomposition, a stiff scaffold structure remains via which the sound waves can still travel directly." Even the modulus of elasticity is not compromised; the wood remains just as resistant to strain as before the fungal treatment – an important criterion for violin making. Before the wood is further processed to a violin, it is treated with ethylene oxide gas. "No fungus can survive that," Professor Schwarze said. That ensures that fungal growth in the wood of the violin is completely stopped.
Together with the violin makers Martin Schleske and Michael Rhonheimer, Professor Schwarze developed violins made of mycowood (wood treated with wood decay fungi). In 2009 the violins were played in a blind, behind-the-curtain test versus a genuine Stradivarius from 1711. All the violins were played by the British violinist Matthew Trusler. The result was surprising for all participants: Both the jury of experts and the majority of the audience thought that the mycowood violin that Schwarze had treated with fungi for nine months was the actual Strad. "Of course, such a test is always subjective," Professor Schwarze said. "There is no clear-cut, scientific method for measuring tonal quality."
Currently Professor Schwarze is working on an interdisciplinary project to develop a quality-controlled treatment for violin wood, with successful, reliable and reproducible results. Until 2014, within the scope of the project that is funded by the Swiss Walter Fischli Foundation, 30 additional violins shall be made from fungally-treated wood. Schwarze explained what opportunities this project can lead to: "The successful implementation of biotechnological methods for treating soundboard wood could in the future give young musicians the opportunity to play on a violin with the sound quality of an expensive – and for most musicians unaffordable – Stradivarius."

http://phys.org/news/2012-09-overfishing-tuna-stocks-brink-experts.html

Overfishing pushes tuna stocks to the brink: experts

Global tuna stocks are fast reaching the limits of fishing sustainability, decimated by an absence of comprehensive, science-based catch limits, conservation experts warned Saturday.

Global tuna stocks are fast reaching the limits of fishing sustainability, decimated by an absence of comprehensive, science-based catch limits, conservation experts warned Saturday.

Five of the world's eight tuna species are already classified as threatened or nearly threatened with extinction, according to the Red List of Threatened Species compiled by the International Union for Conservation of Nature (IUCN).

At the IUCN's World Conservation Congress currently underway in South Korea's southern Jeju Island, experts said partial quotas currently in place were inadequate and uninformed. "The problem is, there is lack of science-based catch limits to ensure effective management and conservation," said Amanda Nickson, Director of Global Tuna Conservation at the Pew Environment Group.

The five Regional Fisheries Management Organisations (RFMOs) that manage the global tuna fishing industry do have some measures in place, including restricting the catch of certain species to the amount caught in a previously defined year.

They also operate "input controls" that, among other things, limit the number of fishing vessels, but Nickson argued these were ineffective as they simply provided an incentive to develop more effective fishing methods.

While acknowledging that scientific data on tuna stocks was "imperfect", Nickson said the UN Fish Stocks Agreement specifically provided for the setting of catch limits if the evidence in favour was compelling enough.

"There is sufficient science available to set precautionary limits," Nickson said. "If we wait five, 10 years for the science to be perfect, in the case of some species we may not have anything left to manage," she added.

The Atlantic bluefin species, which can live to 40 years old and grow to more than four metres (13 feet) long, is in the gravest danger of disappearing with stocks estimated in some areas to have halved over four decades.

It is so highly prized by sushi-loving Japanese that a 269-kilogram (592-pound) fish went for a record 56.49 million yen ($737,000 at the time) in January auctions.

"The message is that some tuna species are in bad shape," said Bruce Collette, chair of the IUCN Tuna and Billfish Specialist Group. "Long living and high value tunas are threatened by over exploitation and under regulation by the regional agencies," Collette warned.

The global tuna industry is an economic juggernaut, with fishing in the Pacific Ocean alone—accounting for 65 percent of the global commercial catch—worth around $5.5 billion a year.

Toshio Katsukawa, a fisheries expert from Mie University in Japan, said only urgent international cooperation could safeguard the future of the Pacific bluefin tuna. "Immediate action is necessary" because the risk of commercial extinction is immediate, Katsukawa said.

http://www.bbc.co.uk/nature/19484530

Lemon sharks 'learn' skills by watching each other

Lemon sharks have the ability to learn from each other's behaviour, US scientists have found.

By Matt Bardo Reporter, BBC Nature

The team compared the performance of inexperienced juvenile sharks working with both trained and untrained partners. The results showed that sharks working with trained partners could complete tasks more quickly and successfully. The study is thought to be the first to demonstrate social learning in any cartilaginous fish.

"I think it's a really cool finding," said lead author Dr Tristan Guttridge from the University of Miami, Florida whose paper was published in the Journal of Animal Cognition. The results are a significant breakthrough, according to Dr Guttridge, director of the Bimini Biological Field Station Foundation in the Bahamas. "It's a pretty exciting finding that these little lemon sharks are able to pick up social cues from each other," he said.

The evidence came from a task-based experiment with juvenile sharks conducted in an underwater pen.

The pen contained an "indicator zone" which functioned as the start area. In the other corner was a "target zone" in which there was a black and white marker that could be covered or exposed by the scientists.

When the sharks swam into the indicator zone, the target was exposed. By swimming into the target zone and bumping the black and white target they earned a piece of barracuda, which was lowered into the pool.

One group of sharks, the "trained demonstrators" was trained in this task until they could complete it roughly six times every minute. Another group, the "sham demonstrators", was left untrained. Members of each group were then paired up with "naive", untrained sharks and the pairs were introduced to the pool, observed and filmed.

"You can see the shark that's been with the demonstrator, how interested he is in the particular zones, moving between them," said Dr Guttridge of the video footage. "It's really quite obvious that they're picking up social cues from the other individual and the excited behaviour of the demonstrator is getting the other guy interested as well." The study then isolated those sharks that had observed the demonstrators to see how they performed on their own. The juveniles that had been paired with "demonstrator sharks" completed a greater number of trials more quickly than those with untrained partners.

Feeding frenzy

Dr Guttridge originally thought of the experiment while watching the behaviour of lemon sharks near his research station. "If you see one performing these kind of tight circles and these excited foraging behaviours, often very quickly another one will start doing the same thing." "[They are] attracted to the kind of behaviour of the other individual and so the lemon shark was the perfect model species for this."

Social learning has already been widely demonstrasted among other species and animal groups including corvids, chimps and bats. "In all these other animals it has been shown to be of real importance to different behavioural processes," said Dr Guttridge, suggesting the same could be true for sharks.

"Sharks do migrate long distances and maybe there's a social context to this as well," he told BBC Nature, comparing his subjects with whales and dolphins that learn their migration routes through culture.

The biologist now hopes to better understand the processes that lemon sharks use to learn from each other. "There are many different social learning processes and in this paper we haven't demonstrated one over another," he said. "We would need to do a much more controlled experiment to eke out the actual social learning processes that are going on."

http://www.eurekalert.org/pub_releases/2012-09/qmuo-rff090512.php

Researchers find first evidence for a genetic cause for Barrett's oesophagus

BE is a condition that predisposes people to develop one of the world's commonest cancers

Genetic variations that are linked with the onset of Barrett's oesophagus (BE), a pre-cancerous condition of the lower end of the gullet, have been identified for the first time. The discovery of variations in regions on two chromosomes makes it possible to develop screening tests for people at high risk of developing the disease.

Although it's been thought for some time that there may be genetic causes for BE as well as environmental ones, such as drinking alcohol and eating fatty food, so far researchers have not found any genetic variations that are associated with the condition.

Now, a multi-national team of researchers led by Professor Janusz Jankowski of the Blizard Institute of Cell and Molecular Science at Queen Mary, University of London (UK), has identified genetic variations on chromosome 6p21 and on chromosome 16q24. Their research is published online today (Sunday) in Nature Genetics [1].

BE [2] is a condition in which abnormal changes occur in cells lining the lower end of the gullet. It is usually caused by acid reflux and the incidence has been increasing over the past few years, with 10-20 per cent of patients with acid reflux developing BE. It can progress to become Barrett's dysplasia, when the cells become pre-cancerous, and can then develop into oesophageal adenocarcinoma (cancer). Five-year survival following a diagnosis of adenocarcinoma is less than 15 per cent, and so it is important to detect and treat the conditions while they are still in their early stages.

Professor Jankowski and colleagues, from over 100 centres in the UK and 20 more around the world, conducted a genome-wide analysis in which they analysed 660,000 genetic variations in 1,800 patients with BE and tested the top 200,000 genetic variations in another set of 1,105 patients, comparing them with large groups of people (over 5,000 in total), acting as controls, who did not have BE. During this process they identified variations in the sequence of single nucleotides – the molecules, A, T, C or G that make up DNA – in two chromosomes. They then tested these two single nucleotide polymorphisms (SNPs) in a further 4,500 patients.

"After these stages, the two SNPs on chromosomes 6p21 and 16q24 showed compelling evidence that they were associated with the development of Barrett's oesophagus," said Professor Jankowski. "This is the first time a genetic link has been shown. Our findings provide a basis for genetically screening 30 per cent of the Western population who get acid reflux to see which 10 to 20 per cent of them – three per cent of the population overall – will go on to develop BE. These genetic variations will also form the basis for developing new targets for therapy."Given that BE is known to be a precursor to oesophageal cancer, it is quite possible that these genetic variations could also be risk factors for developing the cancer and they may give us clues as to the biological mechanisms involved."

The researchers found that one of the genetic variations was close to a gene, FOXF1, which is known to be involved in the development of the gastrointestinal tract (the oesophagus, stomach and intestines), and the other

to the major histocompatability complex (MHC) region where there are genes involved with the immune system, inflammation and the sense of smell.
"Our results provide direct evidence for a genetic cause for Barrett's oesophagus," said Professor Jankowski. "Although it's not completely clear yet what roles are played by the underlying genes, the location of one of the SNPs near to the gene FOXF1 suggests there may be structural factors in the stomach and oesophagus that predispose a person to develop the condition. This is consistent with evidence that a structural deficit, namely hiatus hernia, is known to be strongly associated with BE. We also found evidence to show that SNPs that are known to be associated with increased body weight were also showing a likely association with BE; this suggests that genetic effects may partly underpin the epidemiological observation that obesity is a risk factor for BE."
He continued: "In man, the MHC region occurs on chromosome 6 and consists of 150 genes, of which at least 50 per cent have functions in immunity, auto-immune responses or surveillance. Finding a gene here suggests that the immune system is not under control and is in over-drive in patients with BE. This suggests that it could be controlled by anti-inflammatory agents."
The researchers plan to test another 10,000 patients in order to replicate these results and to see if they can find any other genes that can predict who will go on to develop BE.
"We now know that BE can be inherited like Crohn's or coeliac disease. We have shown that it is likely that the body's control of inflammation and subtle changes to repair mechanisms dictate predisposition to the disease. Our findings make it possible to screen people to predict who will progress to develop BE, and enable us to design new drugs to treat the condition. Given that reflux oesophagitis is the commonest medical condition in the Western adult population, affecting one in three people, these finding have a huge potential impact," concluded Professor Jankowski.
BE occurs in about two per cent of the population, with the highest risk among men over 50 years old in developed countries. The risk of developing oesophageal adenocarcinoma among people with BE has been estimated to be approximately 0.4-1 per cent a year. Oesophageal cancer is the eighth most common cancer worldwide with nearly 482,000 new cases diagnosed and about 406,500 deaths each year [3].
[1] "Common variants at the MHC locus and at chromosome 16q24.1 predispose to Barrett's esophagus", by Janusz A.Z. Jankowski et al. Nature Genetics. doi:10.1038/10.1038/ng.2408
[2] Barrett's oesophagus is named after Norman Barrett, a British surgeon who first described the condition in 1950.
[3] Jankowski JA, Provenzale D, Moayyedi P. "Esophageal adenocarcinoma arising from Barrett's metaplasia has regional variations in the West (systematic review)".

http://www.scientificamerican.com/article.cfm?id=open-mind-longer-life

Creativity Predicts a Longer Life

The trait of openness improves health through creativity

By Tori Rodriguez | Sunday, September 9, 2012 | 4

Researchers have long been studying the connection between health and the five major personality traits: agreeableness, extraversion, neuroticism, openness and conscientiousness. A large body of research links neuroticism with poorer health and conscientiousness with superior health. Now openness, which measures cognitive flexibility and the willingness to entertain novel ideas, has emerged as a lifelong protective factor. The linchpin seems to be the creativity associated with the personality trait—creative thinking reduces stress and keeps the brain healthy.
A study published in the June issue of the Journal of Aging and Health found that higher openness predicted longer life, and other studies this year have linked that trait with lower metabolic risk, higher self-rated health and more appropriate stress response.
The June study sought to determine whether specific aspects of openness better predicted survival rates than overall openness, using data on more than 1,000 older men collected between 1990 and 2008. The researchers found that only creativity—not intelligence or overall openness—decreased mortality risk. One possible reason creativity is protective of health is because it draws on a variety of neural networks within the brain, says study author Nicholas Turiano, now at the University of Rochester Medical Center. “Individuals high in creativity maintain the integrity of their neural networks even into old age,” Turiano says—a notion supported by a January study from Yale University that correlated openness with the robustness of study subjects' white matter, which supports connections between neurons in different parts of the brain.
Because the brain is the command center for all bodily functions, exercising it helps all systems to continue running smoothly. “Keeping the brain healthy may be one of the most important aspects of aging successfully—a fact shown by creative persons living longer in our study,” Turiano says.

He also cites creative people's ability to handle stress—they tend not to get as easily flustered when faced with an emotional or physical hurdle. Stress is known to harm overall health, including cardiovascular, immune and cognitive systems. “Creative people may see stressors more as challenges that they can work to overcome rather than as stressful obstacles they can't overcome,” Turiano says. Although studies thus far have looked at those who are naturally open-minded, the results suggest that practicing creative-thinking techniques could improve anyone's health by lowering stress and exercising the brain.

http://bit.ly/RC7262

Muscles that do nothing can keep you warm and thin

Muscles that burn energy without contracting have yielded new clues about how the body retains a constant temperature – and they may provide new targets for combating obesity.

18:00 09 September 2012 by Andy Coghlan

Traditionally, the body's main thermostat was thought to be brown fat. It raids the body's white fat stores in cold conditions to burn energy and keep the body warm. Muscles also play a role in keeping the body warm by contracting and triggering the shiver response – but this is only a short-term fix because prolonged shivering damages muscles. Now it seems that muscles have another way to turn up the heat.

"Our findings demonstrate for the first time that muscle, which accounts for 40 per cent of body weight in humans, can generate heat independent of shivering," says Muthu Periasamy of Ohio State University in Columbus.

Surviving the chill

Through experiments on mice that had their usual thermostat – brown fat – surgically removed, Periasamy and his colleagues proved that a protein called sarcolipin helps muscle cells keep the body warm by burning energy, almost like an idling motor car, even if the muscles do not contract.

All of the mice had their brown fat removed, but some of them had been genetically engineered to lack sarcolipin too. These rodents could not survive when held at 4 °C, and died of hypothermia within 10 hours. By contrast, mice that could make sarcolipin were able to survive the chilly temperatures and maintained their core body temperature – despite having no brown fat.

Periasamy also showed that an inability to make sarcolipin made mice 33 per cent heavier than normal when fed a high-fat diet. This suggests that idling muscles might also help combat obesity by burning off excess energy. The search is now on for drugs that perform the same role, triggering idling muscles to burn off excess fat.

"The most interesting finding is that mice unable to make sarcolipin are more susceptible to obesity," says Andy Whittle of the University of Cambridge, who is testing spicy dietary treatments to ramp up the fat-burning activity of brown fat. "The research demonstrates that muscle is an important component even in mice, which have comparatively more brown fat than humans. In humans, burning fat in muscle is likely to be even more important for proper energy balance."

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http://www.sciencenews.org/view/generic/id/344133/title/Mars_clays_may_have_volcanic_source

Mars clays may have volcanic source

Deposits didn’t need flowing water to form, new research suggests

By Erin Wayman

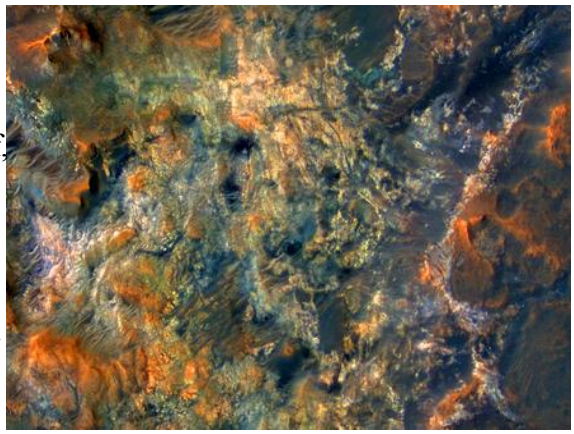
Ancient clay deposits on Mars may not indicate that the Red Planet was originally a warm, wet place, as scientists have thought. Instead of needing liquid water to form, many of Mars’ 4-billion-year-old clays could have originated from cooling lava, researchers report online September 9 in Nature Geoscience.

Clays are widely scattered across Mars’ oldest terrain, dating to the Noachian period 4.1 billion to 3.7 billion years ago. When the Mars Express and Mars Reconnaissance Orbiter discovered these minerals from orbit several years ago, geologists assumed the clays were a result of large bodies of water weathering and altering Mars’ basalt surface. But last year, some researchers suggested that underground hydrothermal activity provided the water that is necessary to form the clays (SN: 12/3/11, p. 5).

Now there’s another suggestion: Crystallizing lava may have contained tiny pockets where water could react with other chemicals to make small amounts of iron- and magnesium-rich clay. No additional water flowing on the surface or belowground would be needed. So early Mars could have been a largely cold, dry world.

“We’re not saying all clays on Mars formed by this process,” says coauthor Bethany Ehlmann, a planetary geologist at Caltech. However, “if most clays formed by a magmatic process, it says maybe water wasn’t so available on early Mars.”

Yet with limited data from the Red Planet, it's too soon to know how pervasive this process might have been. "There is a lot more work to be done before this should be accepted as the prevailing paradigm for clays on Mars," says Laszlo Kestay, director of the U.S. Geological Survey's Astrogeology Science Center in Flagstaff, Ariz.



The researchers investigated the cooling-lava scenario because some Martian clays don't appear to fit with previous explanations, Ehlmann says. Unable to test the idea directly, the team looked for clues on Earth. Some Martian meteorites contain clay minerals with hydrogen isotope compositions characteristic of water coming from Mars' mantle and carried in lava - not from the atmosphere or surface - suggesting water-rich lava has produced some Martian clay.

A warm, wet climate may not have been necessary to form Mars' ancient clays (bright areas in this enhanced-color image). Instead, the minerals might have been the product of cooling lava, researchers suggest. **JPL/NASA, Univ. of Arizona**

The researchers also looked at clay deposits from French Polynesia's Mururoa Atoll in the Pacific Ocean that formed from cooling lava. This clay reflects the same wavelengths of infrared light as Martian deposits, suggesting that both have similar mineralogical properties and thus probably formed in the same way.

The team says cooling lava can account for the most geographically abundant Noachian clay minerals. But that doesn't mean water didn't flow on the surface during brief episodes, as evidenced by the planet's ancient river valleys, says coauthor Alain Meunier of the University of Poitiers in France.

Ehlmann says scientists need to find a spot on Mars where Nochian-aged clay is found so that all three proposed clay-forming mechanisms can be tested. Unfortunately, NASA's Curiosity is not a good test location because the clays there are slightly younger and are clearly part of a sedimentary rather than volcanic deposit.