

<http://www.bbc.co.uk/news/health-21635696>

### **Fat gene 'linked with skin cancer'**

*A gene previously shown to be linked to obesity may also increase the risk of a deadly form of skin cancer, say researchers writing in Nature Genetics.*

Analysis of data from 73,000 people, led by the University of Leeds, found a specific section of the "fat gene" was associated with malignant melanoma. It is the first time the gene has been linked with a specific disease independently of weight. The results suggest a wider role for the gene than originally thought.

Malignant melanoma is the fifth most common cancer in the UK with about 12,800 new cases and about 2,200 deaths each year. An international team analysed genetic data from the tumours of 13,000 malignant melanoma patients and 60,000 unaffected individuals. They found that those with particular variations in a stretch of DNA within the "fat gene" or FTO gene, called intron 8, could be at greater risk of developing melanoma.

#### **New targets**

Previous research linking the FTO gene with obesity found that variants in a section called intron 1 are linked with being overweight and overeating. Several other diseases have been linked to the gene but also to having a high body mass index. This is the first time that researchers have found a link between the FTO gene and a disease which is not linked to obesity and BMI. It opens up a new direction in work looking at how the gene functions as until now the focus has been on its effects on weight gain and factors such as regulating appetite. Study author, Dr Mark Iles, a senior research fellow at the Leeds Institute of Molecular Medicine, said: "This is the first time to our knowledge that this major obesity gene, already linked to multiple illnesses, has been linked to melanoma. "This raises the question whether future research will reveal that the gene has a role in even more diseases?" He added: "When scientists have tried to understand how the FTO gene behaves, so far they've only examined its role in metabolism and appetite. "But it's now clear we don't know enough about what this intriguing gene does."

Dr Julie Sharp, Cancer Research UK's senior science information manager, said: "These are fascinating early findings that, if confirmed in further research, could potentially provide new targets for the development of drugs to treat melanoma. "Advances in understanding more about the molecules driving skin cancer have already enabled us to develop important new skin cancer drugs that will make a real difference for patients." She added the best way to prevent melanoma was to avoid damage caused by too much sun exposure and sunbeds. "Getting a painful sunburn just once every two years can triple the risk of melanoma."

<http://phys.org/news/2013-03-human-chromosome-older-previously-thought.html>

### **Human Y chromosome much older than previously thought**

*Analysis indicates lineage diverged from previously known Y chromosomes about 300,000 ago*

Phys.org - University of Arizona geneticists have discovered the oldest known genetic branch of the human Y chromosome – the hereditary factor determining male sex. The new divergent lineage, which was found in an individual who submitted his DNA to Family Tree DNA, a company specializing in DNA analysis to trace family roots, branched from the Y chromosome tree before the first appearance of anatomically modern humans in the fossil record. The results are published in the American Journal of Human Genetics.

"Our analysis indicates this lineage diverged from previously known Y chromosomes about 300,000 ago, a time when anatomically modern humans had not yet evolved," said Michael Hammer, an associate professor in the University of Arizona's department of ecology and evolutionary biology and a research scientist at the UA's Arizona Research Labs. "This pushes back the time the last common Y chromosome ancestor lived by almost 70 percent."

Unlike the other human chromosomes, the majority of the Y chromosome does not exchange genetic material with other chromosomes, which makes it simpler to trace ancestral relationships among contemporary lineages. If two Y chromosomes carry the same mutation, it is because they share a common paternal ancestor at some point in the past. The more mutations that differ between two Y chromosomes the farther back in time the common ancestor lived.

Originally, a DNA sample obtained from an African American living in South Carolina was submitted to the National Geographic Genographic Project. When none of the genetic markers used to assign lineages to known Y chromosome groupings were found, the DNA sample was sent to Family Tree DNA for sequencing.

Fernando Mendez, a postdoctoral researcher in Hammer's lab, led the effort to analyze the DNA sequence, which included more than 240,000 base pairs of the Y chromosome.

Hammer said "the most striking feature of this research is that a consumer genetic testing company identified a lineage that didn't fit anywhere on the existing Y chromosome tree, even though the tree had been constructed based on perhaps a half-million individuals or more. Nobody expected to find anything like this."

About 300,000 years ago falls around the time the Neanderthals are believed to have split from the ancestral human lineage. It was not until more than 100,000 years later that anatomically modern humans appear in the fossil record. They differ from the more archaic forms by a more lightly built skeleton, a smaller face tucked under a high forehead, the absence of a cranial ridge and smaller chins.

Hammer said the newly discovered Y chromosome variation is extremely rare. Through large database searches, his team eventually was able to find a similar chromosome in the Mbo, a population living in a tiny area of western Cameroon in sub-Saharan Africa. "This was surprising because previously the most diverged branches of the Y chromosome were found in traditional hunter-gatherer populations such as Pygmies and the click-speaking KhoeSan, who are considered to be the most diverged human populations living today."

"Instead, the sample matched the Y chromosome DNA of 11 men, who all came from a very small region of western Cameroon," Hammer said. "And the sequences of those individuals are variable, so it's not like they all descended from the same grandfather."

Hammer cautions against popular concepts of "mitochondrial Eve" or "Y chromosome Adam" that suggest all of humankind descended from exactly one pair of humans that lived at a certain point in human evolution.

"There has been too much emphasis on this in the past," he said. "It is a misconception that the genealogy of a single genetic region reflects population divergence. Instead, our results suggest that there are pockets of genetically isolated communities that together preserve a great deal of human diversity."

Still, Hammer said, "It is likely that other divergent lineages will be found, whether in Africa or among African-Americans in the U.S. and that some of these may further increase the age of the Y chromosome tree." He added: "There has been a lot of hype with people trying to trace their Y chromosome to different tribes, but this individual from South Carolina can say he did it."

More information: [www.cell.com/AJHG/abstract/S0002-9297\(13\)00073-6](http://www.cell.com/AJHG/abstract/S0002-9297(13)00073-6)

[http://www.eurekalert.org/pub\\_releases/2013-03/miot-htb030413.php](http://www.eurekalert.org/pub_releases/2013-03/miot-htb030413.php)

## **How the brain loses and regains consciousness**

***Study reveals brain patterns produced by a general anesthesia drug; work could help doctors better monitor patients***

Written by Anne Trafton, MIT News Office

CAMBRIDGE, MA -- Since the mid-1800s, doctors have used drugs to induce general anesthesia in patients undergoing surgery. Despite their widespread use, little is known about how these drugs create such a profound loss of consciousness. In a new study that tracked brain activity in human volunteers over a two-hour period as they lost and regained consciousness, researchers from MIT and Massachusetts General Hospital (MGH) have identified distinctive brain patterns associated with different stages of general anesthesia. The findings shed light on how one commonly used anesthesia drug exerts its effects, and could help doctors better monitor patients during surgery and prevent rare cases of patients waking up during operations.

Anesthesiologists now rely on a monitoring system that takes electroencephalogram (EEG) information and combines it into a single number between zero and 100. However, that index actually obscures the information that would be most useful, according to the authors of the new study, which appears in the Proceedings of the National Academy of Sciences the week of March 4.

"When anesthesiologists are taking care of someone in the operating room, they can use the information in this article to make sure that someone is unconscious, and they can have a specific idea of when the person may be regaining consciousness," says senior author Emery Brown, an MIT professor of brain and cognitive sciences and health sciences and technology and an anesthesiologist at MGH.

Lead author of the paper is Patrick Purdon, an instructor of anesthesia at MGH and Harvard Medical School.

### **Distinctive patterns**

Last fall, Purdon, Brown and colleagues published a study of brain activity in epileptic patients as they went under anesthesia. Using electrodes that had been implanted in the patients' brains as part of their treatment for epilepsy, the researchers were able to identify a signature EEG pattern that emerged during anesthesia.

In the new study, the researchers studied healthy volunteers, measuring their brain activity with an array of 64 electrodes attached to the scalp. Not only did they find patterns that appeared to correspond to what they saw in last year's study, they were also able to discern much more detail, because they gave the dose of propofol over a longer period of time and followed subjects until they came out of anesthesia.

While the subjects received propofol, the researchers monitored their responsiveness to sounds. Every four seconds, the subjects heard either a mechanical tone or a word, such as their name. The researchers measured EEG activity throughout the process, as the subjects pressed a button to indicate whether they heard the sound. As the subjects became less responsive, distinct brain patterns appeared. Early on, when the subjects were just beginning to lose consciousness, the researchers detected an oscillation of brain activity in the low frequency

(0.1 to 1 hertz) and alpha frequency (8 to 12 hertz) bands, in the frontal cortex. They also found a specific relationship between the oscillations in those two frequency bands: Alpha oscillations peaked as the low-frequency waves were at their lowest point.

When the brain reached a slightly deeper level of anesthesia, a marked transition occurred: The alpha oscillations flipped so their highest points occurred when the low frequency waves were also peaking. The researchers believe that these alpha and low-frequency oscillations, which they also detected in last year's study, produce unconsciousness by disrupting normal communication between different brain regions. The oscillations appear to constrain the amount of information that can pass between the frontal cortex and the thalamus, which normally communicate with each other across a very broad frequency band to relay sensory information and control attention.

The oscillations also prevent different parts of the cortex from coordinating with each other. In last year's study, the researchers found that during anesthesia, neurons within small, localized brain regions are active for a few hundred milliseconds, then shut off again for a few hundred milliseconds. This flickering of activity, which creates the slow oscillation pattern, prevents brain regions from communicating normally.

### **Better anesthesia monitoring**

When the researchers began to slowly decrease the dose of propofol, to bring the subjects out of anesthesia, they saw a reversal of the brain activity patterns that appeared when the subjects lost consciousness. A few minutes before regaining consciousness, the alpha oscillations flipped so that they were at their peak when the low-frequency waves were at their lowest point. "That is the signature that would allow someone to determine if a patient is coming out of anesthesia too early, with this drug," Purdon says.

Cases in which patients regain consciousness during surgery are alarming but very rare, with one or two occurrences in 10,000 operations, Brown says. "It's not something that we're fighting with every day, but when it does happen, it creates this visceral fear, understandably, in the public. And anesthesiologists don't have a way of responding because we really don't know when you're unconscious," he says. "This is now a solved problem."

Purdon and Brown are now starting a training program for anesthesiologists and residents at MGH to train them to interpret the information necessary to measure depth of anesthesia. That information is available through the EEG monitors that are now used during most operations, Purdon says. Because propofol is the most widely used anesthesia drug, the new findings should prove valuable for most operations. In follow-up studies, the researchers are now studying the brain activity patterns produced by other anesthesia drugs.

*The research was funded by the National Institutes of Health, including an NIH Director's Pioneer Award, New Innovator Award and K-Award, and the Harvard Clinical and Translational Science Center.*

[http://www.eurekalert.org/pub\\_releases/2013-03/msu-bac030413.php](http://www.eurekalert.org/pub_releases/2013-03/msu-bac030413.php)

### **Brain adds cells in puberty to navigate adult world**

*The brain adds new cells during puberty to help navigate the complex social world of adulthood, two Michigan State University neuroscientists report in the current issue of the Proceedings of the National Academy of Sciences.*

Scientists used to think the brain cells you're born with are all you get. After studies revealed the birth of new brain cells in adults, conventional wisdom held that such growth was limited to two brain regions associated with memory and smell.

But in the past few years, researchers in MSU's neuroscience program have shown that mammalian brains also add cells during puberty in the amygdala and interconnected regions where it was thought no new growth occurred. The amygdala plays an important role in helping the brain make sense of social cues. For hamsters, it picks up signals transmitted by smell through pheromones; in humans, the amygdala evaluates facial expressions and body language.

"These regions are important for social behaviors, particularly mating behavior," said lead author Maggie Mohr, a doctoral student in neuroscience. "So, we thought maybe cells that are added to those parts of the brain during puberty could be important for adult reproductive function."

To test that idea, Mohr and Cheryl Sisk, MSU professor of psychology, injected male hamsters with a chemical marker to show cell birth during puberty. When the hamsters matured into adults, the researchers allowed them to interact and mate with females.

Examining the brains immediately after that rendezvous, the researchers found new cells born during puberty had been added to the amygdala and associated regions. Some of the new cells contained a protein that indicates cell activation, which told Mohr and Sisk those cells had become part of the neural networks involved in social and sexual behavior. "Before this study it was unclear if cells born during puberty even survived into

adulthood," Mohr said. "We've shown that they can mature to become part of the brain circuitry that underlies adult behavior."

Their results also showed that more of the new brain cells survived and became functional in males raised in an enriched environment – a larger cage with a running wheel, nesting materials and other features – than in those with a plain cage.

While people act in more complicated ways than rodents, the researchers said they hope their work ultimately sheds light on human behavior. "We don't know if cells are added to the human amygdala during puberty," Sisk said, "but we know the amygdala plays a similar role in people as in hamsters. We hope to learn whether similar mechanisms are at play as people's brains undergo the metamorphosis that occurs during puberty."

*The National Institutes of Health funded the research.*

[http://www.eurekalert.org/pub\\_releases/2013-03/kki-sei030413.php](http://www.eurekalert.org/pub_releases/2013-03/kki-sei030413.php)

## **Speech emerges in children with autism and severe language delay at greater rate than thought**

*Study by Kennedy Krieger's Center for Autism and Related Disorders reveals key predictors of speech gains*  
Baltimore, MD – New findings published in *Pediatrics* (Epub ahead of print) by the Kennedy Krieger Institute's Center for Autism and Related Disorders reveal that 70 percent of children with autism spectrum disorders (ASD) who have a history of severe language delay, achieved phrase or fluent speech by age eight. This suggests that more children presenting with ASD and severe language delay at age four can be expected to make notable language gains than was previously thought. Abnormalities in communication and language are a defining feature of ASD, yet prior research into the factors predicting the age and quality of speech attainment has been limited.

The study used the largest sample to date to examine the relationship between key deficits associated with ASD and attainment of phrase and/or fluent speech following a severe language delay, characterized by a child not putting words together into meaningful phrases by age four. As a common milestone of speech development, phrase speech is defined as using non-echoed three-word utterances that sometimes involve a verb and are spontaneous meaningful word combinations; whereas fluent speech is defined as the ability to use complex utterances to talk about topics outside of the immediate physical context.

"We found that nonverbal intelligence was the strongest predictor of phrase speech, while social interest and engagement were as robust, if not greater, when predicting the age that children attained phrase speech and fluent speech," said Ericka L. Wodka, Ph.D., a neuropsychologist in Kennedy Krieger's Center for Autism and Related Disorders and lead study author. "Children with typical nonverbal intelligence attained language almost six months ahead of those with scores below the average."

These findings reinforce that core abilities, such as nonverbal intelligence and social engagement, have a greater influence on the development of communication than other behaviors associated with ASD, such as repetitive and abnormal sensory behaviors. "Our findings continue to support the importance of considering both nonverbal intellectual level and social communication in treatment planning, highlighting the differing impact of these factors as related to treatment goals," said Dr. Wodka.

Data for this retrospective study were from the Simon Simplex Collection (SSC), a unique multi-site database project that gathers biological and phenotypic data on children with ASD aged four- to eighteen-years-old without a previous genetic history of ASD. The database establishes a permanent repository of genetic samples from 2,700 families, each of which has one child affected with an ASD and unaffected parents and siblings. From the SSC, a total of 535 children, ages eight years or older, were studied. Using the Autism Diagnostic Interview-Revised (ADI-R), a standard parent-interview that distinguishes children with ASD from non-ASD populations, and the Autism Diagnosis Observation Schedule (ADOS), a clinician-administered observation that assesses social, communicative and stereotyped behaviors, researchers selected children because they either had no phrase speech at their time of enrollment in the SSC or their phrase speech onset occurred after age four. Based on ADI-R results and their language presentation, children in this study were administered one of four evaluation modules – no words or single words (Module 1), phrase speech (Module 2) or fluent speech (Module 3 or 4). Of the 535 participants in the study, 119 children mastered phrase speech and 253 children were speaking fluently by their eighth birthday, while 163 children never attained phrase or fluent speech

"We hope the results of this study empower parents of children with autism and severe language delays to know that, with the appropriate therapy, a child will likely make significant gains in this area over time; however, progress should be expected to be slower for those children with lower intellectual abilities," said Dr. Wodka.

"Additionally, we hope these findings provide clinicians with better defined therapeutic targets for their patients with autism."

Future longitudinal studies, including both simplex and multiplex families, are required to fully capture the prevalence and predictors of language development in children with ASD. Additionally, further research into the impact of social cognition strategies (e.g., perspective taking) on the development of language, as well as the relationship among specific social deficits and fluent speech development, may hold important implications to the design of intervention.

<http://www.bbc.co.uk/news/uk-northern-ireland-21657395>

### **Dentist Philip Lamey prescribed mouthwash to patient with cancer**

*A dentist prescribed mouthwash to a patient who had oral cancer, a misconduct panel has heard.*

The man visited Professor Philip Lamey at his practice in Belfast in May 2006 suffering pain in his jaw. The dentist noted a 'suspicious lesion' but did not do a biopsy that would have detected the disease, it was said. The patient later had to have a neck dissection and surgeons removed 43 lymph nodes, two of which tested positive for cancer.

Expert witness Dr Stephen Layton said: "This is a man in his early fifties who smokes 40 a day and drinks nine units of alcohol a day. "With respect, you couldn't invent a better candidate for oral cancer."

#### **'Vulnerable'**

He accepted the swollen lymph nodes may have been difficult to spot at his first appointment due to infection. But the expert added: "It is very helpful when someone's telling you it is there."

Dr Layton told the General Dental Council the ulcer was on the floor of the patient's mouth with is a 'danger area' for smokers. "This is an area vulnerable to tumours. The professor should have organised a biopsy in May 2006." He also criticised the professor's note-taking as the exact nature of the patient's condition is not known as he did not make a detailed note of the appointment.

The dentist has been brought before his governing body after 135 patients had to be recalled over fears they might have cancer. Four patients died and it is alleged 15 were diagnosed with the disease late because of his failings. The professor denies 46 charges relating to 33 patients he treated at the Royal Victoria Hospital in Belfast. The hearing in central London continues.

<http://www.scientificamerican.com/article.cfm?id=can-tree-mortality-predict-human-mo-2013-03>

### **Can Tree Mortality Predict Human Mortality?**

*A tree saved your life today. You might not know it, but living near trees is drastically improving your health.*

**By Andrew Price Fast Company**

A tree saved your life today. You might not know it, but living near trees is drastically improving your health. We already know that regular contact with the outdoors (you know, plants and natural light and so on) seems to raise students' test scores and help people recover from surgery more quickly. Now new research indicates that the benefits of trees--and the costs of their absence--might be even more important.

Geoffrey Donovan is a researcher at the U.S. Forest Service's Pacific Northwest Research Station. He has long been interested in how trees affect life for city dwellers. And a devastating pest, the emerald ash borer, presented him with an opportunity to find out what happens when a huge number of trees disappear from residential areas on a large scale. "Perhaps we should start thinking of trees as part of our public-health infrastructure."

The emerald ash borer, an invasive beetle from East Asia, was first discovered in North America in 2002. It infects all species of the North American ash, and infected trees almost never survive. By 2012, it had killed an estimated 100 million trees in 15 states. Donovan and his colleagues gathered 18 years of data from all the counties where the ash borer had wreaked its havoc--mostly around the Great Lakes--and looked for correlations between the loss of trees in those areas and human mortality. "I wasn't surprised that we found an effect," he says, "but I was a little surprised by the size of the effect."

According to their mathematical model, the presence of the borer, and the subsequent loss of trees, was associated with 6.8 additional deaths per year from respiratory causes and 16.7 additional deaths per year from cardiovascular causes per 100,000 adults. That's more than 21,000 deaths in total. "Lack of trees was responsible for more than 21,000 deaths in total." To try to make sure this was a case of causation, and not just correlation, they modeled the relationship between the borer and mortality across space and time simultaneously, and controlled for demographic factors like race and income.

So how exactly do trees help our health? Donovan doesn't know precisely. It could be by improving air quality, reducing stress, increasing physical activity, moderating temperature, or a combination of several factors. Regardless, it's becoming increasingly clear that protecting our arboreal friends is more than hippy tree-hugging--it can keep us humans vibrant and salubrious.

As Donovan says, "Perhaps we should start thinking of trees as part of our public-health infrastructure."

<http://www.scientificamerican.com/article.cfm?id=deep-underground-worms-and-zombie-m>

## **Deep Underground, Worms and 'Zombie Microbes' Rule**

*A dark realm far beneath the Earth's surface is a surprisingly rich home for tiny worms and "zombie microbes" that may hold clues to the origins of life, scientists said on Monday.*

By Environment Correspondent Alister Doyle

OSLO (Reuters) - "It's an amazing new world," said Robert Hazen, head of the Deep Carbon Observatory, a decade-long \$500 million project to study the planet's carbon, an element vital to life and found in everything from oil to diamonds.

"It's very possible that there's a deep microbial biosphere that goes down more than 10 km (6 miles), maybe 20," Hazen told Reuters of the first book by the Observatory, published on Monday and written by more than 50 experts in nine countries.

Microbes had been reported, for instance, in rocks recovered by drilling more than 6 km below the surface in China's Songliao basin, he said. And tiny worms have been found in fractures in rocks 1.3 km deep in a South African mine.

The single-celled microbes found deep underground include bacteria, which need water and nutrients to grow but not necessarily oxygen, and archaea, which can live off compounds such as ammonia or sulphur.

A lack of food in what the 700-page report called the "Stygian realm" - after the River Styx of the underworld in Greek mythology - meant some microbes might be "zombies", or so slow-living as to seem dead.

The book, "Carbon in Earth", said some microbes may live deep below ground and grow and reproduce extremely slowly or perhaps even "live without dividing for millions to tens of millions of years".

Hazen, who works at the Carnegie Institution of Washington, DC, said the scientists working on the project, which began in 2009, were also studying the possibility that life on Earth might have originated underground. "You have everything you need to make life," he said, including energy, water and carbon-rich molecules that could have made the underground zone, rather than the surface of the planet, the cradle of the very first life on Earth.

### **ASTEROIDS**

"We think of the deep subsurface as extreme but it's really quite protected - from asteroids or from volcanic eruptions," he said. "Deep sub-surface rocks are a nice, safe haven."

And the ability of microbes to survive almost indefinitely in rocks on Earth also raises the possibility that rocks from Mars, which had liquid water before Earth, could have seeded our planet with life.

Rocks from Mars sometimes land on Earth after asteroid strikes on Mars blow debris into space. "It's possible that every single cell on Earth is descended from a Martian. That's not crazy science fiction," Hazen said.

The scientists also found viruses deep underground. Unlike those at the surface, those in the energy-poor subsurface typically insert their genes into microbes without seeking to reproduce aggressively, apparently waiting for better conditions.

"This role of virus as parasite, as mutualist, and as a sharer of information through gene transfer may be a fundamental underpinning of life in the deep subsurface," the book suggests. It says underground microbial life should be studied more to ensure that plans for deep burial of nuclear waste, or carbon dioxide from power plants as part of a strategy to combat climate change, would not be destabilized by microbes.

Among other research, the scientists are trying to find out whether there are major sources of oil and gas and methane produced by chemical reactions deep in the Earth, rather than from the well-understood process of ancient surface vegetation being buried and crushed under high pressure.

They will also study diamonds, which form at high pressures deep in the Earth, beyond the depths of current drilling. And they will try to map where the Earth's carbon is stored, from the crust to the core.

"A problem is that a huge part of the system is inaccessible," Rajdeep Dasgupta, an expert at Rice University in Texas, told Reuters. Part of the study will aim to re-create temperatures and conditions of the deep in the laboratory.

<http://phys.org/news/2013-03-detox-poisons-good.html>

### **Plants that can detox waste lands will put poisons to good use**

*Common garden plants are to be used to clean polluted land, with the extracted poisons being used to produce car parts and aid medical research.*

Scientists will use plants such as alyssum, pteridaceae and a type of mustard called sinapi to soak up metals from land previously occupied by factories, mines and landfill sites. Dangerous levels of metals such as arsenic and platinum, which can lurk in the ground and can cause harm to people and animals, will be extracted using a

natural process known as phytoremediation. Once the plants have drawn contaminated material out of the soil, researchers will harvest and then process the plants into materials that can be used more productively. The conversion process will take place in a biorefinery, which will use specifically engineered bacteria to transform the toxic metal ions into more useful metallic nanoparticles.

These tiny particles will be used to make catalytic converters for motor vehicles. They will also be used to help develop cancer treatments.

Cleaning polluted land and rivers could also allow land to be reclaimed and reused, the researchers say. The project involves scientists from the University of Edinburgh, the Universities of Warwick and Birmingham, Newcastle University and Cranfield University.

Dr Louise Horsfall of the University of Edinburgh's School of Biological Sciences, said: "Land is a finite resource. As the world's population grows along with the associated demand for food and shelter, we believe that it is worth decontaminating land to unlock vast areas for better food security and housing. I hope to use synthetic biology to enable bacteria to produce high value nanoparticles and thereby help make land decontamination financially viable." *Provided by University of Edinburgh*

<http://www.rawstory.com/rs/2013/03/05/japanese-man-dies-after-being-turned-away-from-25-hospitals>

### **Japanese man dies after being turned away from 25 hospitals**

*A 75-year-old Japanese man died after 25 hospitals refused to admit him to their emergency rooms 36 times over two hours, citing lack of beds or doctors to treat him, an official said Tuesday.*

**By Agence France-Presse**

The man, who lived alone in a city north of Tokyo, called an ambulance after suffering breathing problems at his home in January. Paramedics rushed to his house but were told in turn by all 25 hospitals in the area that they could not accept the man because they did not have enough doctors or any free beds, a local city official said, adding some institutions were contacted more than once.

The ambulance eventually made a 20 minute drive to a hospital in neighbouring Ibaraki prefecture, but the man was pronounced dead shortly after arrival. The cause of death has not been made public.

One of the paramedics told Jiji Press they had never experienced "a patient being rejected so many times".

The city of Kuki, where the man lived, in Saitama prefecture, has asked hospitals in the region to improve their emergency room capacity, the official said.

Public healthcare in Japan is heavily subsidised and generally of a high global standard.

But commentators warn that with a population that is living longer and with fewer young people entering the workforce, healthcare operators could become increasingly strained over the coming decades.

<http://phys.org/news/2013-03-astronomers-window-europa-ocean.html>

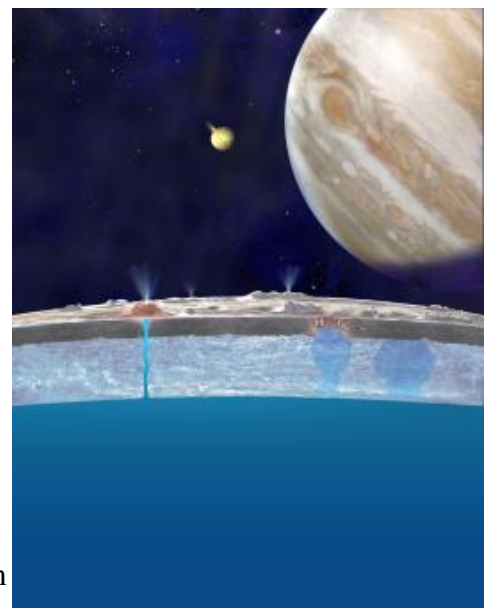
### **Astronomers open window into Europa's ocean**

*Strongest evidence yet that salty water from the vast liquid ocean beneath Europa's frozen exterior actually makes its way to the surface*

Phys.org - With data collected from the mighty W. M. Keck Observatory, California Institute of Technology (Caltech) astronomer Mike Brown—known as the Pluto killer for discovering a Kuiper-belt object that led to the demotion of Pluto from planetary status—and Kevin Hand from the Jet Propulsion Laboratory (JPL) have found the strongest evidence yet that salty water from the vast liquid ocean beneath Europa's frozen exterior actually makes its way to the surface.

The data suggests there is a chemical exchange between the ocean and surface, making the ocean a richer chemical environment, and implies that learning more about the ocean could be as simple as analyzing the moon's surface. The work is described in a paper that has been accepted for publication in the *Astronomical Journal*.

The findings were derived from spectroscopy delivered from the Keck Observatory, which operates the largest and most scientifically productive telescopes on Earth. "We now have the best spectrum of this thing in the world," Brown says. "Nobody knew there was this little dip in the spectrum because no one had the resolution to zoom in on it before."



*Based on new data from the W. M. Keck Observatory about Jupiter's moon Europa, astronomers hypothesize that chloride salts bubble up from the icy moon's global liquid ocean and reach the frozen surface where they are bombarded with sulfur from volcanoes on Jupiter's largest moon, Io. This illustration of Europa (foreground), Jupiter (right) and Io (middle) is an artist's concept. Credit: NASA/JPL-Caltech*

Ten-meter Keck II, fitted with Adaptive Optics (AO) to adjust for the blurring effect of Earth's atmosphere, and its OH-Suppressing Infrared Integral Field Spectrograph (OSIRIS) produced details not capable of collection when NASA's Galileo mission (1989–2003) was sent to study Jupiter and its moons.

"We now have evidence that Europa's ocean is not isolated—that the ocean and the surface talk to each other and exchange chemicals," says Brown, the Richard and Barbara Rosenberg Professor and professor of planetary astronomy at Caltech. "That means that energy might be going into the ocean, which is important in terms of the possibilities for life there. It also means that if you'd like to know what's in the ocean, you can just go to the surface and scrape some off."

"The surface ice is providing us a window into that potentially habitable ocean below," says Hand, deputy chief scientist for solar system exploration at JPL.

Since the days of the Galileo mission, when the spacecraft showed that Europa was covered with an icy shell, scientists have debated the composition of Europa's surface. The infrared spectrometer aboard Galileo was not capable of providing the detail needed to definitively identify some of the materials present on the surface. Now, using current technology on ground-based telescopes, Brown and Hand have definitively identified a spectroscopic feature on Europa's surface that indicates the presence of a magnesium sulfate salt, a mineral called epsomite, that could only originate from the ocean below.

"Magnesium should not be on the surface of Europa unless it's coming from the ocean," Brown says. "So that means ocean water gets onto the surface, and stuff on the surface presumably gets into the ocean water."

Europa's ocean is thought to be 100 kilometers deep and covers the entire globe. The moon remains locked in relation to Jupiter, with the same hemisphere always leading and the other trailing in its orbit. The leading hemisphere has a yellowish appearance, while the trailing hemisphere seems to be splattered and streaked with a red material.

The spectroscopic data from that red side has been a cause of scientific debate for 15 years. It is thought that one of Jupiter's largest moons, Io, spews volcanic sulfur from its atmosphere, and Jupiter's strong magnetic field sends some of that sulfur hurtling toward the trailing hemisphere of Europa, where it sticks. It was also clear from Galileo's data that there is something other than pure water ice on the trailing hemisphere's surface. The debate has focused on what that other something is—i.e., what has caused the spectroscopic data to deviate from the signature of pure water ice.

"From Galileo's spectra, people knew something was there besides water. They argued for years over what it might be—sodium sulfate, hydrogen sulfate, sodium hydrogen carbonate, all these things that look more or less similar in this range of the spectrum," says Brown. "But the really difficult thing was that the spectrometer on the Galileo spacecraft was just too coarse."

Brown and Hand decided that the latest spectrometers on ground-based telescopes could improve the data pertaining to Europa, even from a distance of about 400 million miles. Using the Keck II telescope on Mauna Kea, they first mapped the distribution of pure water ice versus anything else on the moon. The spectra showed that even Europa's leading hemisphere contains significant amounts of nonwater ice. Then, at low latitudes on the trailing hemisphere—the area with the greatest concentration of the nonwater ice material—they found a tiny dip in the spectrum that had never been detected before.

The two researchers racked their brains to come up with materials that might explain the new spectroscopic feature, and then tested everything from sodium chloride to Drano in Hand's lab at JPL, where he tries to simulate the environments found on various icy worlds. "We tried to think outside the box to consider all sorts of other possibilities, but at the end of the day, the magnesium sulfate persisted," Hand says.

Some scientists had long suspected that magnesium sulfate was on the surface of Europa. But, Brown says, "the interesting twist is that it doesn't look like the magnesium sulfate is coming from the ocean." Since the mineral he and Hand found is only on the trailing side, where the moon is being bombarded with sulfur from Io's, they believe that there is a magnesium-bearing mineral everywhere on Europa that produces magnesium sulfate in combination with sulfur. The pervasive magnesium-bearing mineral might also be what makes up the nonwater ice detected on the leading hemisphere's surface.

Brown and Hand believe that this mystery magnesium-bearing mineral is magnesium chloride. But magnesium is not the only unexpected element on the surface of Europa. Fifteen years ago, Brown showed that Europa is surrounded by an atmosphere of atomic sodium and potassium, presumably originating from the surface. The researchers reason that the sodium and potassium chlorides are actually the dominant salts on the surface of Europa, but that they are not detectable because they have no clear spectral features.

The scientists combined this information with the fact that Europa's ocean can only be one of two types—either sulfate-rich or chlorine-rich. Having ruled out the sulfate-rich version since magnesium sulfate was found only



on the trailing side, Brown and Hand hypothesize that the ocean is chlorine-rich and that the sodium and potassium must be present as chlorides.

Therefore, Brown says, they believe the composition of Europa's sea closely resembles the salty ocean of Earth. "If you could go swim down in the ocean of Europa and taste it, it would just taste like normal old salt," he says. Hand emphasizes that, from an astrobiology standpoint, Europa is considered a premier target in the search for life beyond Earth; a NASA-funded study team led by JPL and the Johns Hopkins University Applied Physics Laboratory have been working with the scientific community to identify options to explore Europa further. "If we've learned anything about life on Earth, it's that where there's liquid water, there's generally life," Hand says. "And of course our ocean is a nice salty ocean. Perhaps Europa's salty ocean is also a wonderful place for life." *The Astronomical Journal* paper is titled "Salts and radiation products on the surface of Europa."

[http://www.eurekalert.org/pub\\_releases/2013-03/bumc-slo030413.php](http://www.eurekalert.org/pub_releases/2013-03/bumc-slo030413.php)

### **Safe, long-term opioid therapy is possible**

#### ***Prescription opioid abuse can be minimized by monitoring patients closely using urine drug testing***

(Boston) – In a Clinical Crossroads article featured in the March 6, 2013 issue of the Journal of the American Medical Association (JAMA), Dr. Dan Alford from Boston University School of Medicine (BUSM) and Boston Medical Center (BMC) suggests that prescription opioid abuse can be minimized by monitoring patients closely for harm by using urine drug testing (UDT), pill counts, and reviewing prescription drug monitoring program data when available. Approximately 100 million Americans have chronic pain. The safe and effective use of opioids for the management of chronic pain is complex. Clinicians must balance the goals of relieving pain and suffering while not harming the patient resulting in addiction and overdose.

The JAMA article describes a 71-year old man who had been treated for chronic low back pain since 1981. After getting no pain relief from non-opioids, he achieved pain control with long-term opioids. However a UDT found no opioid in his system on two occasions and his opioid was discontinued. He explained that he occasionally drinks alcohol and does not take his opioid medication when doing so.

"When a patient exhibits behavior for opioid misuse, the clinician should first confirm that the UDT was accurate. If confirmed, the clinician should interview the patient considering the full differential diagnosis for the behavior of concern. Once the etiology has been determined, a change in treatment plan may occur," explained Alford, an associate professor of medicine at BUSM and the Director of the Addiction Medicine Fellowship program at BMC.

Alford stresses that monitoring for benefit includes measuring improvement in pain, function and quality of life. Monitoring for harm includes detecting opioid misuse through UDT, pill counts and use of state prescription drug monitoring programs.

Decisions to continue or discontinue opioids should be based on the risk-to-benefit ratio. "In this case of the patient with no opioid in his UDT if he was benefiting but taking less than prescribed, I would inquire about the status and safe storage of his extra medication. I would decrease his dose and schedule close follow up with random pill counts and UDT. If there was too much risk (misuse such as diversion) despite benefit, I would discontinue his opioid therapy as was done in this case," added Alford.

[http://www.eurekalert.org/pub\\_releases/2013-03/uoc--net030513.php](http://www.eurekalert.org/pub_releases/2013-03/uoc--net030513.php)

### **New evidence that comets could have seeded life on Earth**

#### ***Experiments show that complex molecules can form in icy grains in space***

It's among the most ancient of questions: What are the origins of life on Earth?

A new experiment simulating conditions in deep space reveals that the complex building blocks of life could have been created on icy interplanetary dust and then carried to Earth, jump-starting life.

Chemists from the University of California, Berkeley, and the University of Hawaii, Manoa, showed that conditions in space are capable of creating complex dipeptides – linked pairs of amino acids – that are essential building blocks shared by all living things. The discovery opens the door to the possibility that these molecules were brought to Earth aboard a comet or possibly meteorites, catalyzing the formation of proteins (polypeptides), enzymes and even more complex molecules, such as sugars, that are necessary for life.

"It is fascinating to consider that the most basic biochemical building blocks that led to life on Earth may well have had an extraterrestrial origin," said UC Berkeley chemist Richard Mathies, coauthor of a paper published online last week and scheduled for the March 10 print issue of *The Astrophysical Journal*.

While scientists have discovered basic organic molecules, such as amino acids, in numerous meteorites that have fallen to Earth, they have been unable to find the more complex molecular structures that are prerequisites for our planet's biology. As a result, scientists have always assumed that the really complicated chemistry of life must have originated in Earth's early oceans.

In an ultra-high vacuum chamber chilled to 10 degrees above absolute zero (10 Kelvin), Seol Kim and Ralf Kaiser of the Hawaiian team simulated an icy snowball in space including carbon dioxide, ammonia and various hydrocarbons such as methane, ethane and propane. When zapped with high-energy electrons to simulate the cosmic rays in space, the chemicals reacted to form complex, organic compounds, specifically dipeptides, essential to life.

At UC Berkeley, Mathies and Amanda Stockton then analyzed the organic residues through the Mars Organic Analyzer, an instrument that Mathies designed for ultrasensitive detection and identification of small organic molecules in the solar system. The analysis revealed the presence of complex molecules – nine different amino acids and at least two dipeptides – capable of catalyzing biological evolution on earth.

*The research was supported by the National Science Foundation and the Mathies Royalty Fund at UC Berkeley.*

[http://www.eurekalert.org/pub\\_releases/2013-03/uom-gte030513.php](http://www.eurekalert.org/pub_releases/2013-03/uom-gte030513.php)

## **Green tea extract interferes with the formation of amyloid plaques in Alzheimer's disease**

***Researchers at the University of Michigan have found a new potential benefit of a molecule in green tea: preventing the misfolding of specific proteins in the brain.***

ANN ARBOR—The aggregation of these proteins, called metal-associated amyloids, is associated with Alzheimer's disease and other neurodegenerative conditions. A paper published recently in the Proceedings of the National Academy of Sciences explained how U-M Life Sciences Institute faculty member Mi Hee Lim and an interdisciplinary team of researchers used green tea extract to control the generation of metal-associated amyloid- $\beta$  aggregates associated with Alzheimer's disease in the lab.

The specific molecule in green tea, (—)-epigallocatechin-3-gallate, also known as EGCG, prevented aggregate formation and broke down existing aggregate structures in the proteins that contained metals—specifically copper, iron and zinc.

"A lot of people are very excited about this molecule," said Lim, noting that the EGCG and other flavonoids in natural products have long been established as powerful antioxidants. "We used a multidisciplinary approach. This is the first example of structure-centric, multidisciplinary investigations by three principal investigators with three different areas of expertise." The research team included chemists, biochemists and biophysicists. While many researchers are investigating small molecules and metal-associated amyloids, most are looking from a limited perspective, said Lim, assistant professor of chemistry and research assistant professor at the Life Sciences Institute, where her lab is located and her research is conducted. "But we believe you have to have a lot of approaches working together, because the brain is very complex," she said.

The PNAS paper was a starting point, Lim said, and her team's next step is to "tweak" the molecule and then test its ability to interfere with plaque formation in fruit flies. "We want to modify them for the brain, specifically to interfere with the plaques associated with Alzheimer's," she said.

Lim plans to collaborate with Bing Ye, a neurobiologist in the LSI. Together, the researchers will test the new molecule's power to inhibit potential toxicity of aggregates containing proteins and metals in fruit flies.

*Other authors of the paper, all from U-M, are: Sanghyun Lee and Jung-Suk Choi of the Life Sciences Institute; Alaina DeToma, Suk-Joon Hyung, Akiko Kochi and Brandon Ruotoloa of the Department of Chemistry; and Jeffrey Brender, Ayyalusamy Ramamoorthy and Subramanian Vivekanandan of the Department of Chemistry and Biophysics.*

*The work was supported by the National Institutes of Health, Alzheimer's Association, Alzheimer's Art Quilt Initiative, American Heart Association, and a Graduate Research Fellowship from the National Science Foundation Study:*

<http://www.pnas.org/content/early/2013/02/19/1220326110.abstract>

<http://www.scientificamerican.com/article.cfm?id=can-livestock-grazing-stop-desertification>

## **Can Livestock Grazing Stop Desertification?**

***Overgrazing has been a major cause of the creeping advance of deserts worldwide but new management techniques might make livestock part of the solution***

**By Colin Sullivan and ClimateWire | Tuesday, March 5, 2013 | 3**

Zimbabwe's foremost land degradation expert has come up with a readily available solution for reversing the spread of deserts around the planet and slowing climate change in the process: He wants to let cows and sheep eat their way through the problem. In a provocative appearance on the video blog Ted Talks, biologist Allan Savory said desertification of the world's grasslands may be releasing more carbon into the atmosphere than burning fossil fuels. Savory should know: He has been studying the spread of deserts for more than 50 years. But the former revolutionary turned scientist recently came to a surprising conclusion about how best to bring back grasslands and in the process help address poverty and social breakdown in some of the poorest corners of the planet. He turned to holistic management of livestock like cattle and sheep, overriding his own belief that grazing animals had been part of the problem when it came to green, fertile lands widely becoming barren and dry.

That notion, Savory said, was dead wrong. He cited an experiment he conducted in the 1950s in the country then known as Rhodesia, when he helped exterminate more than 40,000 grazing elephants to protect land thought to be stressed and dying from their annual trampling rituals.

He called that project "the saddest and greatest blunder" of his life. "We were once just as certain that the world was flat," Savory said on the Ted Talks appearance. "We were wrong then, and we're wrong again."

Savory said the annual rite of movement through a region by large herds actually protects the environment. A wildebeest migration in central Africa, for instance, eats up grasses as it moves along and leaves behind a protective layer of trampled dung, dust and soil.

### **Trying to mimic the roles of wild herds**

That protective layer, it turns out, is vital for healthy soils that trap carbon, break down methane and produce more grasses every year to feed returning grazers. In turn, those herds feed predators like lions, cheetahs and, yes, human beings.

So Savory decided to mimic the great herds of old, which have died out in many regions or persist in far reduced numbers, with managed "strategic" herds of grazing vegetarians. The sheep and cattle picked for the project, if managed properly, would theoretically bring nature back to its normal cycle in semiarid regions where rains for part of the year are followed by long dry spells.

Savory said his experiments have worked, and he showed a number of before-and-after pictures as evidence during his talk. He thinks the same approach can be taken in the two-thirds of the planet that is rapidly desertifying, including parts of the American Southwest.

Fire has long been used as a means to kill woody vegetation in semiarid regions and restore soils, but Savory said that solution has never quite panned out because fire can strip land of its base layers, not to mention release carbon. So he turned to cattle and sheep.

"There was only one option left to climatologists and scientists, and that is to do the unthinkable: Use livestock bunched and moving as a proxy for former moving herds and predators," he said.

Savory's experiments with livestock have reversed degraded dry lands in Zimbabwe, Mexico, the Horn of Africa and Argentina, he said. He added that putting the same idea into motion in just half the world's troubled grasslands would result in bringing the planet back to preindustrial levels of greenhouse gas emissions.

"I can think of nothing that offers more hope for your planet," he said.

[Click here](#) to view the video.

<http://www.scientificamerican.com/article.cfm?id=insect-wings-shred-bacteria-to-pieces>

## **Insect Wings Shred Bacteria to Pieces**

### ***Antibacterial "nanopillars" on cicada wings pull bacterial membranes apart***

By Trevor Quirk and Nature magazine | Tuesday, March 5, 2013 | 4

The veined wing of the clanger cicada kills bacteria solely through its physical structure — one of the first natural surfaces found to do so. An international team of biophysicists has now come up with a detailed model of how this defense works on the nanoscale. The results are published in the latest issue of the Biophysical Journal.

The clanger cicada (*Psaltoda claripennis*) is a locust-like insect whose wings are covered by a vast hexagonal array of 'nanopillars' — blunted spikes on a similar size scale to bacteria (see video, bottom). When a bacterium settles on the wing surface, its cellular membrane sticks to the surface of the nanopillars and stretches into the crevices between them, where it experiences the most strain. If the membrane is soft enough, it ruptures (see video, below).



***Antibacterial: The clanger cicada kills bacteria solely via its physical structure.*** Image: Flickr/LadyDragonflyCC

Lead study author Elena Ivanova of Australia's Swinburne University of Technology in Hawthorne, Victoria, says that she was surprised that the bacterial cells are not actually punctured by the nanopillars. The rupturing effect is more like "the stretching of an elastic sheet of some kind, such as a latex glove. If you take hold of a piece of latex in both hands and slowly stretch it, it will become thinner at the center, [and] will begin to tear," she explains.

To test their model, Ivanova and her team irradiated bacteria with microwaves to generate cells that had different levels of membrane rigidity. Their hypothesis was that the more rigid bacteria would be less likely to rupture between the nanopillars. The results validated the model, but also demonstrated that the cicada's nanopillar defense is limited to bacteria that have sufficiently soft membranes.

Further study of the cicada's wing is needed before its physical-defense properties can be mimicked in man-made materials. Anne-Marie Kietzig, a chemical engineer at McGill University in Montreal, Canada, who was not involved in the study, suggests that materials based on this model could one day be applied to public surfaces that commonly harbor disease, such as bus railings. "This would provide a passive bacteria-killing surface," she says, adding that it "does not require active agents like detergents, which are often environmentally harmful".

<http://www.livescience.com/27624-mummy-head-middle-ages-anatomy.html>

## Grotesque Mummy Head Reveals Advanced Medieval Science

*New analysis of the oldest-known preserved human dissection in Europe reveals doctors in medieval Europe weren't as idle as it may seem*

Stephanie Pappas, LiveScience Senior Writer

In the second century, an ethnically Greek Roman named Galen became doctor to the gladiators. His glimpses into the human body via these warriors' wounds, combined with much more systematic dissections of animals, became the basis of Islamic and European medicine for centuries.

Galen's texts wouldn't be challenged for anatomical supremacy until the Renaissance, when human dissections — often in public — surged in popularity. But doctors in medieval Europe weren't as idle as it may seem, as a new analysis of the oldest-known preserved human dissection in Europe reveals.



*This anatomical specimen dating to the 1200s is the oldest known in Europe. CREDIT: photo courtesy Archives of Medical Science*

The gruesome specimen, now in a private collection, consists of a human head and shoulders with the top of the skull and brain removed. Rodent nibbles and insect larvae trails mar the face. The arteries are filled with a red "metal wax" compound that helped preserve the body.

The preparation of the specimen was surprisingly advanced. Radiocarbon dating puts the age of the body between A.D. 1200 and A.D.1280, an era once considered part of Europe's anti-scientific "Dark Ages." In fact, said study researcher Philippe Charlier, a physician and forensic scientist at University Hospital R. Poincare in France, the new specimen suggests surprising anatomical expertise during this time period.

"It's state-of-the-art," Charlier told LiveScience. "I suppose that the preparator did not do this just one time, but several times, to be so good at this."

### Myths of the middle ages

Historians in the 1800s referred to the Dark Ages as a time of illiteracy and barbarianism, generally pinpointing the time period as between the fall of the Roman Empire and somewhere in the Middle Ages. To some, the Dark Ages didn't end until the 1400s, at the advent of the Renaissance. But modern historians see the Middle Ages quite differently. That's because continued scholarship has found that the medieval period wasn't so ignorant after all. "There was considerable scientific progress in the later Middle Ages, in particular from the 13th century onward," said James Hannam, an historian and author of "The Genesis of Science: How the Christian Middle Ages Launched the Scientific Revolution" (Regnery Publishing, 2011).

For centuries, the advancements of the Middle Ages were forgotten, Hannam told LiveScience. In the 16th and 17th centuries, it became an "intellectual fad," he said, for thinkers to cite ancient Greek and Roman sources rather than scientists of the Middle Ages. In some cases, this involved straight-up fudging. Renaissance mathematician Copernicus, for example, took some of his thinking on the motion of the Earth from Jean Buridan, a French priest who lived between about 1300 and 1358, Hannam said. But Copernicus credited the ancient Roman poet Virgil as his inspiration.

Much of this selective memory stemmed from anti-Catholic feelings by Protestants, who split from the church in the 1500s. As a result, "there was lots of propaganda about how the Catholic Church had been holding back human progress, and it was great that we were all Protestants now," Hannam said.

### Anatomical dark ages?

From this anti-Catholic sentiment arose a great many myths, such as the idea that everyone believed the world to be flat until Christopher Columbus sailed to the Americas. ("They thought nothing of the sort," Hannam said.) Similarly, Renaissance propagandists spread the rumor that the Medieval Christian church banned autopsy and human dissection, holding back medical progress.

In fact, Hannam said, many societies have banned or limited the carving up of human corpses, from the ancient Greeks and Romans to early Europeans (that's why Galen was stuck dissecting animals and peering into

gladiator wounds). But autopsies and dissection were not under a blanket church ban in the Middle Ages. In fact, the church sometimes ordered autopsies, often for the purpose of looking for signs of holiness in the body of a supposedly saintly person.

The first example of one of these "holy autopsies" came in 1308, when nuns conducted a dissection of the body of Chiara of Montefalco, an abbess who would be canonized as a saint in 1881. The nuns reported finding a tiny crucifix in the abbess' heart, as well as three gallstones in her gallbladder, which they saw as symbolic of the Holy Trinity. Other autopsies were entirely secular. In 1286, an Italian physician conducted autopsies in order to pinpoint the origin of an epidemic, according to Charlier and his colleagues.

Some of the belief that the church frowned on autopsies may have come from a misinterpretation of a papal edict from 1299, in which the Pope forbade the boiling of the bones of dead Crusaders. That practice ensured Crusaders' bones could be shipped back home for burial, but the Pope declared the soldiers should be buried where they fell. "That was interpreted in the 19th century as actually being a stricture against human dissection, which would have surprised the Pope," Hannam said.

### **Well-studied head**

While more investigation of the body was going on in the Middle Ages than previously realized, the 1200s remain the "dark ages" in the sense that little is known about human anatomical dissections during this time period, Charlier said. When he and his colleagues began examining the head-and-shoulders specimen, they suspected it would be from the 1400s or 1500s. "We did not think it was so antique," Charlier said.

But radiocarbon dating put the specimen firmly in the 1200s, making it the oldest European anatomical preparation known. Most surprisingly, Charlier said, the veins and arteries are filled with a mixture of beeswax, lime and cinnabar mercury. This would have helped preserve the body as well as give the circulatory system some color, as cinnabar mercury has a red tint.

Thus, the man's body was not simply dissected and tossed away; it was preserved, possibly for continued medical education, Charlier said. The man's identity, however, is forever lost. He could have been a prisoner, an institutionalized person, or perhaps a pauper whose body was never claimed, the researchers write this month in the journal *Archives of Medical Science*.

The specimen, which is in private hands, is set to go on display at the Parisian Museum of the History of Medicine, Charlier said. "This is really interesting from a historical and archaeological point of view," Charlier said, adding, "We really have a lack of skeletons and anthropological pieces."

<http://www.scientificamerican.com/article.cfm?id=child-hiv-cure-duplicate>

### **Yes, a Child Has Been Pronounced Cured of HIV—but Can It Be Duplicated?**

*A baby born exposed to the AIDS-causing virus received aggressive treatment that appears to have cured the child, and promises to spark new avenues for future research as well*

By Marissa Fessenden | Tuesday, March 5, 2013 | 6

A child born to an HIV-infected mother in Mississippi may be cured after a swiftly administered course of drugs. A number of factors make the child's case unique, however, and clinicians caution that we have not discovered a general cure for HIV yet. Still, the medical first may hint at ways to fight the AIDS-causing virus. An HIV cure has been elusive because the virus has ways of hiding within the body. It can secret itself into blood cells and other so-called reservoirs. Faced with powerful drugs that prevent viral replication, called antiretroviral therapy (ART), HIV levels in the blood drop down to nearly undetectable levels. Take the pressure off by halting treatment, however, and the virus comes roaring back.

Recent years have offered some hints about how to disable HIV's assaults. A rare category of individuals, dubbed "elite controllers" can drop the drugs and still show no symptoms. Also, researchers are developing a treatment that will eliminate one of HIV's entryways into immune cells through a gene-editing process. Yet, the best approach already available is to prevent infection, a daunting challenge despite decades of progress.

Preventing infection in the very young is a priority. Every day, approximately 1,000 babies are infected around the world with HIV during gestation, birth or breast-feeding, according to the United Nations Children's Fund. Typically, newborns at risk of contracting HIV may receive one or two antiretroviral drugs prophylactically. If at six to eight weeks of age the baby tests positive for the viral antibodies, the physician will switch to the therapeutic cocktail and doses. The baby from Mississippi received a combination of the drugs zidovudine, lamivudine and nevirapine, just 30 hours after birth.

This aggressive treatment is not typical because antiretroviral drugs are toxic and infection is not always certain. The mother passes HIV antibodies on to her child during gestation. Only after six to eight weeks of life can clinicians tell whether the baby is actually infected with HIV and not simply carrying the mother's antibodies. "These drugs are not like vitamins," says Lynne Mofenson, chief of the Maternal and Pediatric Infectious

Disease Branch at the National Institute of Child Health and Human Development. “You only use them when the child is at high risk.”

Current guidelines in the U.S. recommend that expectant mothers who are infected with HIV receive drugs during pregnancy. Then, babies should be delivered via cesarean section and be formula-fed. Those recommendations can minimize the risk of transmission to less than 1 percent Mofenson says. Accordingly, mother-to-child transmission of the virus is rare in developed nations but much more common where anti-HIV drugs are scarce. Currently, fewer than 200 children in the U.S. are born HIV-positive each year.

The mother in the new case did not receive any prenatal care or ART. She arrived at the clinic in labor and delivered her baby prematurely (at 35 weeks into her pregnancy). When a test came back showing she was HIV-positive, University of Mississippi Medical Center pediatric HIV specialist Hannah Gay determined that the risk to the child was great. Therefore she decided to treat, even though clinicians hadn’t confirmed that the baby was infected.

The newborn did test positive for viral DNA and RNA at two days old. She also tested positive at days seven, 12 and 20, but the viral load dropped off, indicating the drug cocktail was working as expected. The baby was given liquid ART every day: a combination of zidovudine, lamivudine and co-formulated lopinavir–ritonavir. At 29-days old, the child’s HIV RNA levels had fallen so low that they were undetectable in clinical tests. After 18 months, in January 2012, the mother stopped visiting to the clinic for unreported reasons. When the physicians tracked her and the baby down in autumn 2012 they found the viral RNA was still undetectable despite months off the anti-HIV medications. Only ultrasensitive tests revealed extremely low levels of the virus. They reported the case at the Conference on Retroviruses and Opportunistic Infections in Atlanta on March 3.

The case report’s lead-investigator Deborah Persaud, a virologist at Johns Hopkins University, also presented the results of a small study of teens infected with HIV at birth. Five of the teenagers received anti-HIV drugs at two months of age and carried lower viral DNA levels than four teens that had received the drugs later in childhood.

“Taken together, the findings of our two studies show that very early ART in infants prevents the development of long-term viral reservoirs, and in doing so may put newborns on a path to long-term remission and on the road to a functional cure,” Persaud said in a prepared statement.

The case is an important proof of concept, says Anthony Fauci, director of the National Institute of Allergy and Infectious Diseases, who was not involved in the case. The DNA and RNA tests demonstrated that the baby had the virus for at least 10 or 12 days, indicating a probable, if early, infection. “The caveat is that this is still a single case,” he adds. “This is not something that is immediately generalizable, but it does tell you that under some circumstances there is a chance to cure.”

“It is a very interesting case report,” Mofenson says, who was also not involved in the Mississippi case. “It has some unusual aspects to it.” She first learned about the situation last year at a think tank for researchers. The mother presented with extremely low viral loads, which is unusual for an infected adult not taking anti-HIV drugs. The child’s first HIV tests also showed very low levels. These facts indicate that there is something unusual about the virus or the host, Mofenson says.

The new case may be a cure for just one child. Or it could be an anomaly—perhaps the child was never infected or is not actually cured, says Joseph M. McCune, a professor of experimental medicine at the University of California, San Francisco. He says he is most intrigued by the idea that the immaturity of a newborn’s immune system somehow enables it to cope better with the HIV infection. Previous research shows that the inflammatory response mounted by an immune system under threat can actually make the HIV virus grow more readily. An inflammatory response brings more immune cells to the site of injury or infection, increases cell division and boosts the production of molecules called cytokines. The HIV virus has evolved to take advantage of each of these processes—it spreads from cell to cell, so rapid division nearby helps HIV replicate quickly. Cytokines, which are small proteins that cells use to communicate, seem to be another cue the virus uses to know when to replicate, McCune says. But a newborn does not mount an inflammatory response as readily as an adult does. So the virus may take longer to fully infect a baby.

The immune response of the fetus differs from that of the newborn as well because cells from the mother move across the placenta and enter the fetus. “The fetus doesn’t want to make an inflammatory response against mother,” McCune says. “So the fetus has developed an immune system that says ‘do not respond.’” That calming of the immune response may hold over to the first few days of the newborn’s life; the inflammatory response does not fully activate, robbing the new HIV infection of additional fuel. This delay, combined with a short course of aggressive treatment, may give the body enough of a head start to eradicate the virus on its own, he notes.

"It's very exciting," McCune says. "Many revolutions in medicine have occurred because of a single case." Mofenson cautions that the result from the Mississippi case does not mean that clinicians and pediatricians should change any of their practices yet. Even children or older patients with very low viral load levels should continue antiretroviral therapy. There are "multiple questions raised by this case that really need urgent and further research." To that end, her institute has put out a call for research proposals. "Now people are aware of this and can bring other children to our attention," she says. "Hopefully, within a year or two we will have better answers."

[http://www.eurekalert.org/pub\\_releases/2013-03/uorm-sbi030113.php](http://www.eurekalert.org/pub_releases/2013-03/uorm-sbi030113.php)

**Study: Brain injury may be autoimmune phenomenon, like multiple sclerosis**  
*Damage to the blood-brain barrier and the resulting autoimmune response might be the cause of neurological disorders*

Most scientists are starting to agree that repeat, sub-concussive hits to the head are dangerous and linked to neurological disorders later in life. A new collaborative study, though, attempted to find out why – and discovered that damage to the blood-brain barrier and the resulting autoimmune response might be the culprit. Published in journal PLOS ONE by the University of Rochester Medical Center and the Cleveland Clinic, the research suggests a new way of thinking about concussions: That the brain degeneration observed among professional football players (including the much-publicized chronic traumatic encephalopathy) could result from an out-of-control immune response, similar to what multiple sclerosis patients experience. If so, this opens the door to investigating a vaccine or drug therapy to prevent head trauma.

Although he emphasized that the research is preliminary, co-author Jeffrey J. Bazarian, M.D., M.P.H., associate professor of Emergency Medicine at URM, said it's exciting to discover a theory that appears to fit with the reality of what experts observe among athletes. Bazarian worked closely with lead investigator Damir Janigro, Ph.D., professor of Molecular Medicine at the Cleveland Clinic, and 67 college football players from northeast Ohio and Rochester, N.Y., who agreed to participate in the research.

"Although the awareness of sports-related concussions is much higher, we still know very little about the long-term consequences and what happens inside the brain," Bazarian said. "Our theory is plausible as an explanation for how routine head hits that come with playing football can lead to severe neuro-degeneration later in life," said Bazarian, a national expert who has served on an Institute of Medicine committee for brain injury. "If others confirm this, it could present options with drugs that influence the immune response."

The blood-brain barrier is like a semi-permeable gate between the brain and bloodstream. No other organ has such a barrier. When the barrier is working properly, it holds in proteins and molecules that bathe the brain and protect it from foreign substances. With blows to the head, however, the barrier opens slightly and allows some proteins to leak into the bloodstream. Researchers found that S100B, a well-accepted protein biomarker for traumatic brain injury, was present in varying degrees in the blood samples of the 67 football players after every game -- even though none of them suffered a concussion. This demonstrates that even the most routine hits have some impact on the blood-brain barrier and possibly the brain itself, Bazarian said.

For the purposes of this project, however, the team wanted to explore what happens after S100B surges from the brain and enters the bloodstream. Again, they made an important finding – that the body views S100B as an enemy and begins to form antibodies against it as if it were a virus.

Researchers hypothesized that a buildup of antibodies would result in a more vigorous attack on S100B in the bloodstream. But in the process, they learned, some antibodies sneak back through the damaged blood-brain barrier to the brain and begin to harm the healthy brain cells that produced the S100B protein in the first place. This is analogous to a missile searching for a target, Bazarian said, with some unintended targets eventually falling under attack. Researchers also showed that S100B accumulates in dendritic cells, which regulate autoimmune responses. Therefore, as the blood-brain barrier repeatedly opens during the football season it might set the stage for a continuous autoimmune-type attack on the brain, they reasoned.

In multiple sclerosis a similar breakdown occurs, when the body's own immune system damages myelin sheaths around the brain. Other health conditions that harm the blood-brain barrier include sepsis (overwhelming infection), burns, critical illness, or seizures.

The methods used to test the hypothesis involved each player giving blood samples before and after games. Researchers then analyzed the samples for S100B levels and auto-immune antibody levels. They also monitored the number of hits each player sustained by viewing game films and conducting post-game interviews, and gave each player standard cognitive and functional tests, pre-season and post-season. In addition, a subset of 10 players from the University of Rochester received special brain scans with diffusion tensor imaging, a more sensitive MRI that can detect subtle axonal injury.

Results showed that players with the most head hits also had the highest S100B levels and elevated levels of autoimmune antibodies. Players who often remained on the sidelines had significantly lower S100B levels. In addition, the blood samples predicted abnormalities seen in the imaging tests, and correlated with observed cognitive changes.

Although many scientists are actively investigating concussions in the United States right now, it's been difficult to study the link between brain injury, blood-brain barrier damage, and the long-term risk of neurodegeneration because of a lack of simple, non-invasive tools, Bazarian said. But demonstrating that S100B can be used in this way adds a new dimension to the scientific literature. Other investigators have also used the S100B protein to study Alzheimer's patients, the study noted.

Bazarian hopes that eventually S100B will be a tool for emergency rooms and other clinical settings to screen for concussions. Doctors can accurately measure it with a simple finger prick; many European countries already use S100B to decide which patients need a CT scan when a concussion is suspected.

<http://www.sciencedaily.com/releases/2013/03/130305174043.htm>

### **Is It a Stroke or Benign Dizziness? A Simple Bedside Test Can Tell**

*A bedside electronic device that measures eye movements can successfully determine whether the cause of severe, continuous, disabling dizziness is a stroke or something benign, according to results of a small study led by Johns Hopkins Medicine researchers.*

"Using this device can directly predict who has had a stroke and who has not," says David Newman-Toker, M.D., Ph.D., an associate professor of neurology and otolaryngology at the Johns Hopkins University School of Medicine and leader of the study described in the journal *Stroke*. "We're spending hundreds of millions of dollars a year on expensive stroke work-ups that are unnecessary, and probably missing the chance to save tens of thousands of lives because we aren't properly diagnosing their dizziness or vertigo as stroke symptoms." Newman-Toker says if additional larger studies confirm these results, the device could one day be the equivalent of an electrocardiogram (EKG), a simple noninvasive test routinely used to rule out heart attack in patients with chest pain. And, he adds, universal use of the device could "virtually eliminate deaths from misdiagnosis and save a lot of time and money."

To distinguish stroke from a more benign condition, such as vertigo linked to an inner ear disturbance, specialists typically use three eye movement tests that are essentially a stress test for the balance system. In the hands of specialists, these bedside clinical tests (without the device) have been shown in several large research studies to be extremely accurate -- "nearly perfect, and even better than immediate MRI," says Newman-Toker. One of those tests, known as the horizontal head impulse test, is the best predictor of stroke. To perform it, doctors or technicians ask patients to look at a target on the wall and keep their eyes on the target as doctors move the patients' heads from side to side. But, says Newman-Toker, it requires expertise to determine whether a patient is making the fast corrective eye adjustments that would indicate a benign form of dizziness as opposed to a stroke.

For the new study, researchers instead performed the same test using a small, portable device — a video-oculography machine that detects minute eye movements that are difficult for most physicians to notice. The machine includes a set of goggles, akin to swimming goggles, with a USB-connected webcam and an accelerometer in the frame. The webcam is hooked up to a laptop where a continuous picture of the eye is taken. Software interprets eye position based on movements and views of the pupil, while the accelerometer measures the speed of the movement of the head.

Newman-Toker says the test could be easily employed to prevent misdiagnosis of as many as 100,000 strokes a year, leading to earlier stroke diagnosis and more efficient triage and treatment decisions for patients with disabling dizziness. Overlooked strokes mean delayed or missed treatments that lead to roughly 20,000 to 30,000 preventable deaths or disabilities a year, he says. The technology, he adds, could someday be used in a smartphone application to enable wider access to a quick and accurate diagnosis of strokes whose main symptom is dizziness, as opposed to one-sided weakness or garbled speech.

The diagnosis of stroke in patients with severe dizziness, vomiting, difficulty walking and intolerance to head motion is difficult, Newman-Toker says. He estimates there are 4 million emergency department visits annually in the United States for dizziness or vertigo, at least half a million of which involve patients at high risk for stroke. The most common causes are benign inner ear conditions, but many emergency room doctors, Newman-Toker says, find it nearly impossible to tell the difference between the benign conditions and something more serious, such as a stroke. So they often rely on brain imaging -- usually a CT scan, an expensive and inaccurate technology for this particular diagnosis.



The Hopkins-led study enrolled 12 patients at The Johns Hopkins Hospital and the University of Illinois College of Medicine at Peoria, who later underwent confirmatory MRI. Six were diagnosed with stroke and six with a benign condition using video-oculography. MRI later confirmed all 12 diagnoses.

The device was developed overseas and is used in balance clinics there, but is not yet approved for use in the United States. A company, GN Otometrics, which makes the devices used in Newman-Toker's proof-of-concept study, loaned them to the research team, but did not have any involvement, financial or otherwise, in the study, he says.

Newman-Toker says head CT scans are ordered for roughly 40 percent of patients who come to the emergency room with dizziness, but that CT misses more than 80 percent of acute strokes occurring in the brainstem and cerebellum, making it impossible to rule out stroke in dizzy patients this way. Instead, MRI is the definitive test, though it isn't readily available in many emergency departments or in rural hospitals, and it costs roughly four times the price of a CT scan. MRI also misses 10 to 20 percent of this type of stroke in the first 48 hours after symptoms begin, he says.

*The research was supported by grants from the Swiss National Science Foundation (PBBEP2 136573), the Agency for Healthcare Research and Quality (R18 HS017690) and the National Institutes of Health's National Center for Research Resources (K23 RR024009) and National Eye Institute (R01EY019347).*

*Other Johns Hopkins researchers involved in the study include Ali S. Saber Tehrani, M.D.; Georgios Mantokoudis, M.D.; Ari Blitz, M.D.; Sarah H. Ying, M.D.; Yu-Hsiang Hsieh, Ph.D.; Richard E. Rothman, M.D., Ph.D.; Daniel F. Hanley, M.D.; and David S. Zee, M.D. Collaborators from other institutions include John H. Pula, M.D.; Cynthia I. Guede, R.N., B.S.N.; Kevin A. Kerber, M.D., M.S.; and senior author Jorge C. Kattah, M.D.*

*D. E. Newman-Toker, A. S. S. Tehrani, G. Mantokoudis, J. H. Pula, C. I. Guede, K. A. Kerber, A. Blitz, S. H. Ying, Y.-H. Hsieh, R. E. Rothman, D. F. Hanley, D. S. Zee, J. C. Kattah. Quantitative Video-Oculography to Help Diagnose Stroke in Acute Vertigo and Dizziness: Toward an ECG for the Eyes. Stroke, 2013; DOI: 10.1161/STROKEAHA.111.000033*

<http://www.bbc.co.uk/news/health-21671455>

### **Stomach cancer 'spotted by breath test'**

***A quick and simple breath test can diagnose stomach cancer, study findings reveal.***

**By Michelle Roberts Health editor, BBC News online**

Scientists from Israel and China found the test was 90% accurate at detecting and distinguishing cancers from other stomach complaints in 130 patients.

The British Journal of Cancer says the test could revolutionise and speed up the way this cancer is diagnosed. About 7,000 UK people develop stomach cancer each year and most have an advanced stage of the disease. Two-fifths of patients survive for at least a year, but only a fifth are still alive after five years, despite treatment. Currently doctors diagnose stomach cancer by taking a biopsy of the stomach lining using a probe and a flexible camera passed via mouth and down the gullet.

The new test looks for chemical profiles in exhaled breath that are unique to patients with stomach cancer.

#### **Volatile organic compounds**

Cancer appears to give off a signature smell of volatile organic compounds that can be detected using the right technical medical kit - and perhaps even dogs. The science behind the test itself is not new - many researchers have been working on the possibility of breath tests for a number of cancers, including lung.

But the work by Prof Hossam Haick, of the Technion-Israel Institute of Technology, suggests it is a good way to spot stomach cancer. In the study, 37 of the patients had stomach cancer, 32 had stomach ulcers and 61 had other stomach complaints. As well as accurately distinguishing between these conditions 90% of the time, the breath test could tell the difference between early and late-stage stomach cancers.

The team are now running a bigger study in more patients to validate their test.

Kate Law, director of clinical research at Cancer Research UK, said: "The results of this latest study are promising - although large scale trials will now be needed to confirm these findings.

"Only one in five people are able to have surgery as part of their treatment as most stomach cancers are diagnosed at stages that are too advanced for surgery. Any test that could help diagnose stomach cancers earlier would make a difference to patients' long-term survival."

[http://www.eurekalert.org/pub\\_releases/2013-03/plos-dm030113.php](http://www.eurekalert.org/pub_releases/2013-03/plos-dm030113.php)

### **'Prevent death' message more effective than 'save life' in blood donation campaigns**

***'Prevent loss' message better than 'provide benefits' to increase volunteerism***

Subtle changes in messaging can have a profound impact on the effectiveness of charitable messages such as calls for blood donations, according to research published March 6 in the open access journal PLOS ONE by Eileen Chou from the University of Virginia and co-author Keith Murnighan at Northwestern University.

Though chronic shortages in U.S blood banks could be alleviated by a small increase in the number of blood donors, people are not always motivated enough to help. In the current study, researchers collaborated with the

Red Cross to assess the effects of changing the urgency and messaging of a call for blood donations. The scientists found that on a college campus, describing blood donations as a way to "prevent a death" rather than "save a life" significantly increased the rate of donations.

In a second study, the researchers assessed the effects of these slight changes in framing a charitable message on people's emotional motivation for a monetary donation. Here, they found that framing an appeal as "helping people to avoid a loss" rather than "helping people to gain benefits" led to increased intentions to volunteer and more helping behavior. Volunteers presented with such "prevention of loss" messages were also more likely to expect larger donations to their cause. "These findings demonstrated a simple, reliable, and effective method for charities to significantly increase important helping behaviors," Chou said.

*Citation: Chou EY, Murnighan JK (2013) Life or Death Decisions: Framing the Call for Help. PLOS ONE 8(3): e57351. doi:10.1371/journal.pone.0057351 Financial Disclosure: The authors have no funding or support to report.*

[http://www.eurekalert.org/pub\\_releases/2013-03/plos-sfr030113.php](http://www.eurekalert.org/pub_releases/2013-03/plos-sfr030113.php)

### **Siberian fossil revealed to be one of the oldest known domestic dogs**

***DNA analysis finds 33,000-year old dog ancestor was more related to modern dogs than wolves***

Analysis of DNA extracted from a fossil tooth recovered in southern Siberia confirms that the tooth belonged to one of the oldest known ancestors of the modern dog, and is described in research published March 6 in the open access journal PLOS ONE by Anna Druzhkova from the Institute of Molecular and Cellular Biology, Russian Federation, and colleagues from other institutions.

Human domestication of dogs predates the beginning of agriculture about 10,000 years ago, but when modern dogs emerged as a species distinct from wolves is still unclear. Although some previous studies have suggested that this separation of domestic dogs and wolves occurred over 100,000 years ago, the oldest known fossils of modern dogs are only about 36,000 years old.

The new research published today evaluates the relationship of a 33,000 year old Siberian fossil to modern dogs and wolves based on DNA sequence. The researchers found that this fossil, named the 'Altai dog' after the mountains where it was recovered, is more closely related to modern dogs and prehistoric canids found on the American continents than it is to wolves. They add, ""These results suggest a more ancient history of the dog outside the Middle East or East Asia, previously thought to be the centers where dogs originated."

*Citation: Druzhkova AS, Thalmann O, Trifonov VA, Leonard JA, Vorobieva NV, et al. (2013) Ancient DNA Analysis Affirms the Canid from Altai as a Primitive Dog. PLoS ONE 8(3): e57754. doi:10.1371/journal.pone.0057754*

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*Competing Interest Statement: The authors have declared that no competing interests exist.*

[http://www.eurekalert.org/pub\\_releases/2013-03/yo-foa030413.php](http://www.eurekalert.org/pub_releases/2013-03/yo-foa030413.php)

### **Flip of a single molecular switch makes an old brain young**

***The flip of a single molecular switch helps create the mature neuronal connections that allow the brain to bridge the gap between adolescent impressionability and adult stability.***

Now Yale School of Medicine researchers have reversed the process, recreating a youthful brain that facilitated both learning and healing in the adult mouse.

Scientists have long known that the young and old brains are very different. Adolescent brains are more malleable or plastic, which allows them to learn languages more quickly than adults and speeds recovery from brain injuries. The comparative rigidity of the adult brain results in part from the function of a single gene that slows the rapid change in synaptic connections between neurons.

By monitoring the synapses in living mice over weeks and months, Yale researchers have identified the key genetic switch for brain maturation a study released March 6 in the journal *Neuron*. The Nogo Receptor 1 gene is required to suppress high levels of plasticity in the adolescent brain and create the relatively quiescent levels of plasticity in adulthood. In mice without this gene, juvenile levels of brain plasticity persist throughout adulthood. When researchers blocked the function of this gene in old mice, they reset the old brain to adolescent levels of plasticity.

"These are the molecules the brain needs for the transition from adolescence to adulthood," said Stephen Strittmatter, Vincent Coates Professor of Neurology, Professor of Neurobiology and senior author of the paper. "It suggests we can turn back the clock in the adult brain and recover from trauma the way kids recover."

Rehabilitation after brain injuries like strokes requires that patients re-learn tasks such as moving a hand. Researchers found that adult mice lacking Nogo Receptor recovered from injury as quickly as adolescent mice and mastered new, complex motor tasks more quickly than adults with the receptor.

"This raises the potential that manipulating Nogo Receptor in humans might accelerate and magnify rehabilitation after brain injuries like strokes," said Feras Akbik, Yale doctoral student who is first author of the study.

Researchers also showed that Nogo Receptor slows loss of memories. Mice without Nogo receptor lost stressful memories more quickly, suggesting that manipulating the receptor could help treat post-traumatic stress disorder. "We know a lot about the early development of the brain," Strittmatter said, "But we know amazingly little about what happens in the brain during late adolescence."

*Other Yale authors are: Sarah M. Bhagat, Pujan R. Patel and William B.J. Cafferty*

*The study was funded by the National Institutes of Health. Strittmatter is scientific founder of Axerion Therapeutics, which is investigating applications of Nogo research to repair spinal cord damage.*

<http://phys.org/news/2013-03-shipwreck-legendary-sunstone.html>

### **Shipwreck find could be legendary 'sunstone'**

***An oblong crystal found in the wreck of a 16th-century English warship is a sunstone, a near-mythical navigational aid said to have been used by Viking mariners, researchers said on Wednesday.***

An oblong crystal found in the wreck of a 16th-century English warship is a sunstone, a near-mythical navigational aid said to have been used by Viking mariners, researchers said on Wednesday.

The stone is made of Iceland spar, a transparent, naturally-occurring calcite crystal that polarises light and can get a bearing on the Sun, they said.

It was found in the remains of a ship that had been dispatched to France in 1592 by Queen Elizabeth I as a precaution against a second Spanish Armada but foundered off the island of Alderney, in the Channel.

British and French scientists have long argued that the find is a sunstone—a device that fractures the light, enabling seafarers to locate the Sun even when it is behind clouds or has dipped below the horizon.

Sunstones, according to a theory first aired 45 years ago, helped the great Norse mariners to navigate their way to Iceland and even perhaps as far as North America during the Viking heyday of 900-1200 AD, way before the magnetic compass was introduced in Europe in the 13th century.

But there is only a sketchy reference in ancient Norse literature to a "solarsteinn," which means the idea has remained frustratingly without solid proof.

In a study published in the British journal Proceedings of the Royal Society A, investigators carried out a chemical analysis on a tiny sample, using a device called a spectrometer, which confirmed that the stone was a calcite.

The stone is about the size of a small bar of soap whose edges have been trimmed at an angle. In technical terms, its shape is rhombohedral.

It is milky white in appearance, and not transparent, but the new experiments show that this is surface discolouration, caused by centuries of immersion in sea water and abrasion by sand, the study said.

Using a transparent crystal similar to the original, the scientists were able to follow the track of the setting Sun in poor light, with an accuracy of one degree. In a second experiment, they were able to locate the Sun for 40 minutes after sunset.

Other factors provide evidence that this is a sunstone, according to the investigation, led by Guy Ropars of the University of Rennes, in France's western region of Brittany.

The crystal was found in the wreckage alongside a pair of navigation dividers. And tests that placed a magnetic compass next to one of the iron cannons excavated from the ship found that the needle swung wildly, by as much as 100 degrees.

Put together, these suggest the sunstone may have been kept as a backup to a magnetic compass.

"Although easy to use, the magnetic compass was not always reliable in the 16th century, as most of the magnetic phenomena were not understood," says the study.

"As the magnetic compass on a ship can be perturbed for various reasons, the optical compass giving an absolute reference may be used when the Sun is hidden."

The authors also note previous research that some species of migrating birds appear to have used polarised light from the sky as a navigational aid or to recalibrate their magnetic compass around sunrise and sunset.

How does the sunstone work?

If you put a dot on top of the crystal and look at it from below, two dots appear, because the light is "depolarised" and fractured along different axes.

You then rotate the crystal until the two points have exactly the same intensity or darkness.

"At that angle, the upward-facing surface of the crystal indicates the direction of the Sun," Ropars told AFP in an interview in 2011, when preliminary research about the Alderney stone was published.

<http://phys.org/news/2013-03-herbal-medicine-evolutionary-lens.html>

## **Herbal medicine through an evolutionary lens**

*A phylogenetic study has shown that related plants are used traditionally in three disparate regions to treat similar medical conditions.*

There is often scepticism surrounding traditional herbal treatments, partly due to scarcity in large-scale evidence of efficacy of traditional medicine. A team of researchers from Kew, the University of Reading, Imperial College and RBG Edinburgh, in collaboration with colleagues from Nepal and New Zealand, have conducted a phylogenetic study that provides support for herbal remedies.

The researchers constructed a genus-level family tree representing 20,000 plant species found in three disparate regions (Nepal, New Zealand, and the Cape of South Africa), in order to compare medicinal plants used in these geographic areas. They found that plants traditionally used to treat similar health conditions came from the same plant clusters across the three floras. These shared phylogenetic patterns in traditional herbal medicine were interpreted as independent discovery of efficacy in these plant groups. This was supported by the finding that many plants used to produce drugs come from these clusters highlighted by traditional knowledge, suggesting that plant bioactivity underlies traditional medicine worldwide.

*More information: Haris Saslis-Lagoudakis, C., et al. (2012). Phylogenies reveal predictive power of traditional medicine in bioprospecting. Proc. Natl. Acad. Sci. 109: 15835-1584.*

<http://phys.org/news/2013-03-emergency-immobiliser-accident-victims.html>

## **Emergency immobiliser for accident victims**

*Smart textile material can adopt different shapes, turns rigid when vacuum is applied to it and achieves hardness equivalent to that of a conventional plastic*

The Centre for Applied Research Tecnia Research & Innovation has through its FIK initiative designed Varstiff, a smart textile material that can adopt different shapes; when vacuum is applied to it, it turns rigid once again and achieves hardness equivalent to that of a conventional plastic.

The material reverts to its flexible state once the vacuum is released. The first product Varstiff will be used for will be an emergency immobiliser for accident victims.

Thanks to the encouragement and collaboration of Janus Development, this product has been selected by the Botín Foundation as one of the three award-winning projects in the first edition of its programme "Mind the Gap".

To support its launch onto the market a new technology-based enterprise will be set up to operate in the healthcare sector initially, but with plans to expand its activity to other sectors like the automotive or leisure sectors.

The new material designed by Tecnia can be adapted to any part of the body and in any situation; it can be fitted in its soft, malleable state so that afterwards when vacuum is applied to it, it becomes as stiff as plaster of Paris. This makes it possible in the event of an accident to immediately immobilise parts of the victim's body that are difficult to access without moving it, like the neck, back or thorax.

The funding of 350,000 euros over two years provided by the Botín Foundation through its "Mind the Gap" programme will enable the development of the first two products to be completed: an immobiliser for emergencies, and a position fixator to improve the life quality of people who have to use wheel chairs.

The new enterprise is expected to be up and running by the end of 2013 and is expected to launch its first product onto the market early in 2014. Its headquarters will be located in the Basque Country and it is estimated that it will achieve an accumulated turnover of 2 million euros during the first four years it is operating.

### **Other solutions**

This revolutionary material also offers solutions in other spheres of healthcare, like orthopaedics, where it has advantages over ordinary solutions that use elastic straps that are closed using Velcro, or inflatable cushions. These solutions apply pressure and therefore exert force on the skin; and apart from lacking the necessary stiffness, they reduce comfort.

The automotive sector, leisure and sports are other fields in which this material can have new uses to ensure the safety and comfort of its users. In the automotive sector this material will contribute greater comfort and personalisation of different items, like seats that can be adjusted to each person, systems for absorbing energy in doors, or flexible luggage racks. In the sphere of sports, it could lead to flexible items for camping like chairs, tables, mats, etc.

Likewise, this material could have a pioneering role in the development of high-performance protection textiles, like for example, clothing for extreme sports or for security personnel.

<http://www.wired.com/wiredscience/2013/03/cre-cdc/>

**‘We Have a Limited Window of Opportunity’: CDC Warns of Resistance ‘Nightmare’**  
*It’s not often that you get to hear a top federal health official deliberately deploy a headline-grabbing word such as “nightmare,” or warn: “We have a very serious problem, and we need to sound an alarm.”*

By **Marvyn McKenna**

Dr. Thomas Frieden, director of the US Centers for Disease Control and Prevention, said both Tuesday, during a press conference announcing new CDC statistics on the advance of the highly drug-resistant bacteria known as CRE. His language — plus the fact that he conducted the entire press conference himself, instead of just making a brief opening statement — seem to me a clear signal that the CDC is taking this resistance problem seriously, and hoping we do too.

And we should. Here’s what the CDC [announced Tuesday](#):

- **Healthcare institutions in 42 states have now identified at least one case of CRE.**
- **The occurrence of this resistance in the overall family of bacteria has risen at least four-fold over 10 years.**
- **In the CDC’s surveillance networks, 4.6 percent of hospitals and 17.8 percent of long-term care facilities diagnosed this bug in the first half of 2012.**

Those are dire reports.

Here’s some back-story: CRE stands for “carbapenem-resistant Enterobacteriaceae.” Enterobacteriaceae are a family of more than 70 bacteria which share the characteristic of being gut-dwelling (“entero”); they include *Klebsiella*, *Salmonella*, *Shigella* and *E. coli*. Carbapenems are a “last-resort” family of antibiotics — imipenem, meropenem, doripenem and ertapenem — which are used against these bacteria when they have become resistant to other drugs. (Carbapenem resistance is conferred by a number of different genes and so sometimes goes by a number of other acronyms, including KPC, VIM, OXA and the “Indian superbug” NDM-1.) CRE tends to attack in ICUs and other critical care, and also in rehab units and nursing homes. That is for several reasons. First, because patients in those settings are uniquely vulnerable to infection, not just because of their illness but because the protective barrier of their skin has been breached by ports and catheters, and also because they are visited and touched by a lot of people. Second, because they are likely to be receiving heavy-duty antibiotics which put the bacteria in their bodies under evolutionary pressure. Third, because those drugs plus others cause diarrhea, which spreads gut-dwelling bacteria into the air and area. And fourth, because those bacteria are particularly good at surviving on the kind of surfaces — plastic, glass and metal — that you find in health care.

Carbapenem resistance first appeared in the US in 1996, in a single sample containing KPC that the CDC found in a hospital in North Carolina. By the early 2000s, it was causing significant outbreaks in hospitals in New York City; from there, it spread with New Yorkers to “snowbird” vacation locations, and then to Israel, and then started moving around the globe. (You can read that history in a piece I did for [Scientific American in April 2012](#); and my past posts on all this [are here](#).)

The map of CRE in the US now looks like this, as released by the CDC yesterday. Among the outbreaks represented on that map are the “NIH superbug” outbreak last year (described in these [two posts](#) and [by Carl Zimmer on the magazine side of Wired](#)), and also an outbreak of [NDM-1 in a Rhode Island hospital](#) in 2011. But — and the CDC acknowledges this — the map is probably an understatement, for several reasons. CRE is not what public health calls a “reportable disease”; according to the CDC, only six states require that physicians or hospitals tell the rest of the world they have diagnosed it. (Three others are “considering” making it reportable.) Plus, surveillance for CRE is patchy; yesterday’s CDC report comprised data from three different surveillance systems. And also, there are carbapenem-resistant bacteria causing outbreaks in the US which are not counted as CREs because the bacteria are not Enterobacteriaceae. For one example, take a look at the breathtaking trend line in this [map of carbapenem-resistant Acinetobacter](#), put together by the ResistanceMap project at the Center for Disease Dynamics, Economics and Policy.

The tone of the CDC press conference yesterday was unusually somber and blunt. [Frieden said](#):

Carbapenemase-producing CRE in the United States



States with confirmed CRE cases caused by the KPC enzyme

Alabama	Nevada
Arizona	New Hampshire
Arkansas	New Jersey
California (including CRE caused by the NDM enzyme, VIM or IMP enzyme)	New Mexico
Colorado (including CRE caused by the NDM enzyme)	New York
Connecticut	North Carolina
Delaware	North Dakota
Florida	Ohio
Georgia	Oregon
Illinois (including CRE caused by the NDM enzyme)	Pennsylvania
Indiana	Puerto Rico
Iowa	Rhode Island (including CRE caused by the NDM enzyme)
Kentucky	South Carolina
Louisiana	South Dakota
Maryland (including CRE caused by the NDM enzyme)	Tennessee
Massachusetts (including CRE caused by the NDM enzyme)	Texas
Michigan	Utah
Minnesota (including CRE caused by the NDM enzyme)	Virginia (including CRE caused by the NDM enzyme)
Mississippi	Washington (including CRE caused by the NDM enzyme, VIM enzyme)
Missouri	West Virginia
Montana	Wisconsin
	Wyoming

CRE... pose a triple threat. First, they're resistant to all or nearly all antibiotics. Even some of our last-resort drugs. Second, they have high mortality rates. They kill up to half of people who get serious infections with them. And third, they can spread their resistance to other bacteria. So one form of bacteria, for example, carbapenem-resistant *Klebsiella*, can spread the genes that destroy our last antibiotics to other bacteria, such as *E. coli*, and make *E. coli* resistant to those antibiotics also... We only have a limited window of opportunity. The underlying risk here is that effectively untreatable CRE will spread out from hospitals and into the wider world, where it will become vastly more common and much harder to detect. That is not an unreasonable fear, given that the Enterobacteriaceae include incredibly common *E. coli*, which has already been found to be causing [bladder infections bearing a slightly less dire form of multi-drug resistance](#), known as ESBL.

So what's to be done? In their press push yesterday, the CDC reviewed six steps that they [first published last year in a CRE Toolkit](#) and want health care facilities to take:

- *enforce infection-control precautions (that's hand-washing, gowning and gloving, and so on)*
- *group patients with CRE together in one part of a unit or facility*
- *reserve certain rooms, pieces of equipment and staff members for CRE patients*
- *require hospitals, nursing homes and so on to tell each other when they are transferring a patient with CRE*
- *interrogating patients about recent medical care elsewhere, including in other countries*
- *and using antibiotics conservatively, so that bacteria don't get much of a chance to develop resistance to the last-resort drugs.*

But an important point is that none of this is required, and none of this is funded. When the Netherlands wanted to beat back the emergence of MRSA, that country passed laws requiring every hospital to test patients before letting them in the door. (That story is told in [this book](#).) When Israel wanted to counter KPC, which was ripping through its hospitals after arriving from the US, it created a national task force and imposed mandatory national measures for detecting and confining the infection. (That program is described in [this 2011 paper](#).) And hospitals are on their own in figuring out how to organize and pay for CRE control. There are no reimbursements, under Medicare, for infection-control as a hospital task; and as infection-prevention physician Eli Perencevich [demonstrated two years ago](#), the National Institutes of Health is not funding resistance-countering research.

(It is well worth reading [Perencevich's response, posted yesterday](#), to CDC's CRE announcement. Money quote: "This is not a national response. This is a national tragedy.")

So what's the takeaway? Public reaction to news of antibiotic resistance seems to follow a predictable pattern: Instant alarm, followed almost immediately by apathy. Despite writing about this for years, I still haven't figured out whether people think it will never happen to them, or whether they assume there will always be another drug to save them — both assumptions that are incorrect. But I've also written about the CDC for years, and I can't remember many times when they have made statements as strongly worded as yesterday's. It will be interesting to see whether the news sinks in this time.

For more:

- *The CDC's report from yesterday: "[Vital Signs: Carbapenem-Resistant Enterobacteriaceae](#)," MMWR, March 5, 2013. 62 (Early Release);1-6*
- *You can track the emergence of carbapenem-resistant organisms across the US over time using [CDDEP's Resistance Map](#).*
- *And if you want to know more about what it took to end the NIH CRE outbreak, you can listen to [Eli Perencevich and I discuss it](#) on NPR's Talk of the Nation.*

<http://www.sciencedaily.com/releases/2013/03/130306134358.htm>

## **Excess Dietary Salt May Drive the Development of Autoimmune Diseases**

*Increased dietary salt intake can induce a group of aggressive immune cells that are involved in triggering and sustaining autoimmune diseases.*

This conclusion is the result of a study conducted by Dr. Markus Kleinewietfeld, Prof. David Hafler (both Yale University, New Haven and the Broad Institute of the Massachusetts Institute of Technology, MIT, and Harvard University, USA), PD Dr. Ralf Linker (Dept. of Neurology, University Hospital Erlangen), Professor Jens Titze (Vanderbilt University and Friedrich-Alexander-Universität Erlangen-Nürnberg, FAU, University of Erlangen-Nuremberg) and Professor Dominik N. Müller (Experimental and Clinical Research Center, ECRC, a joint cooperation between the Max-Delbrück Center for Molecular Medicine, MDC, Berlin, and the Charité – Universitätsmedizin Berlin and FAU). In autoimmune diseases, the immune system attacks healthy tissue instead of fighting pathogens.

In recent decades scientists have observed a steady rise in the incidence of autoimmune diseases in the Western world. Since this increase cannot be explained solely by genetic factors, researchers hypothesize that the sharp increase in these diseases is linked to environmental factors. Among the suspected culprits are changes in lifestyle and dietary habits in developed countries, where highly processed food and fast food are often on the

daily menu. These foods tend to have substantially higher salt content than home-cooked meals. This study is the first to indicate that excess salt intake may be one of the environmental factors driving the increased incidence of autoimmune diseases.

A few years ago Jens Titze showed that excess dietary salt (sodium chloride) accumulates in tissue and can affect macrophages (a type of scavenger cells) of the immune system. Independent of this study, Markus Kleinewietfeld and David Hafler observed changes in CD4 positive T helper cells (Th) in humans, which were associated with specific dietary habits. The question arose whether salt might drive these changes and thus can also have an impact on other immune cells. Helper T cells are alerted of imminent danger by the cytokines of other cells of the immune system. They activate and "help" other effector cells to fight dangerous pathogens and to clear infections. A specific subset of T helper cells produces the cytokine interleukin 17 and is therefore called Th17 for short. Evidence is mounting that Th17 cells, apart from fighting infections, play a pivotal role in the pathogenesis of autoimmune diseases.

### **Salt dramatically boosts the induction of aggressive Th17 immune cells**

In cell culture experiments the researchers showed that increased sodium chloride can lead to a dramatic induction of Th17 cells in a specific cytokine milieu. "In the presence of elevated salt concentrations this increase can be ten times higher than under usual conditions," Markus Kleinewietfeld and Dominik Müller explained. Under the new high salt conditions, the cells undergo further changes in their cytokine profile, resulting in particularly aggressive Th17 cells.

In mice, increased dietary salt intake resulted in a more severe form of experimental autoimmune encephalomyelitis, a model for multiple sclerosis. Multiple sclerosis is an autoimmune disease of the central nervous system in which the body's own immune system destroys the insulating myelin sheath around the axons of neurons and thus prevents the transduction of signals, which can lead to a variety of neurological deficits and permanent disability. Recently, researchers postulated that autoreactive Th17 cells play a pivotal role in the pathogenesis of multiple sclerosis.

Interestingly, according to the researchers, the number of pro-inflammatory Th17 cells in the nervous system of the mice increased dramatically under a high salt diet. The researchers showed that the high salt diet accelerated the development of helper T cells into pathogenic Th17 cells. The researchers also conducted a closer examination of these effects in cell culture experiments and showed that the increased induction of aggressive Th17 cells is regulated by salt on the molecular level. "These findings are an important contribution to the understanding of multiple sclerosis and may offer new targets for a better treatment of the disease, for which at present there is no known cure," said Ralf Linker, who as head of the Neuroimmunology Section and Attending Physician at the Department of Neurology, University Hospital Erlangen, seeks to utilize new laboratory findings for the benefit of patients.

Besides multiple sclerosis, Dominik Müller and his colleagues want to study psoriasis, another autoimmune disease with strong Th17 components. The skin, as Jens Titze recently discovered, also plays a key role in salt storage and affects the immune system. "It would be interesting to find out if patients with psoriasis can alleviate their symptoms by reducing their salt intake," the researchers said. "However, the development of autoimmune diseases is a very complex process which depends on many genetic and environmental factors," the immunologist Markus Kleinewietfeld said. "Therefore, only further studies under less extreme conditions can show the extent to which increased salt intake actually contributes to the development of autoimmune diseases."

*Markus Kleinewietfeld, Arndt Manzel, Jens Titze, Heda Kvakana, Nir Yosef, Ralf A. Linker, Dominik N. Müller, David A. Hafler. Sodium chloride drives autoimmune disease by the induction of pathogenic TH17 cells. Nature, 2013; DOI: 10.1038/nature11868*

[http://www.eurekalert.org/pub\\_releases/2013-03/uocf-kcc030613.php](http://www.eurekalert.org/pub_releases/2013-03/uocf-kcc030613.php)

### **Killing cancer cells with acid reflux**

***A University of Central Florida chemist has come up with a unique way to kill certain cancer cells – give them acid reflux.***

Chemistry professor Kevin Belfield used a special salt to make cancer cells more acidic – similar to the way greasy foods cause acid reflux in some people. He used a light-activated, acid-generating molecule to make the cells more acidic when exposed to specific wavelengths of light, which in turn kills the bad cells. The surrounding healthy cells stay intact.

The technique is a simple way around a problem that has frustrated researchers for years. For photodynamic therapy (the special laser-light treatment) to work, cancer cells loaded with photosensitizers need oxygen to trigger the fatal reaction. But by their very nature, most cancer cells lack oxygen. Nonetheless, scientists were intent on making the photodynamic system work because it offers a way to target cancer cells deep within

human tissue without causing a lot of collateral damage. Instead of focusing on oxygen, Belfield flipped the problem around and found another way to poison the bad cells, while protecting the healthy ones.

"It's the first time we've found a way around the oxygen problem," Belfield said. "This work is truly ground breaking. It should eventually provide a therapeutic means to treat certain types of cancers with minimal side effects. It should also be a very useful tool for cell biologists and biomedical researchers. It could even find a place in treating other diseases such as neurodegenerative diseases." His work was recently published in the Journal of the American Chemical Society. (<http://pubs.acs.org/doi/full/10.1021/ja3122312>)

Belfield and his team at UCF used human colorectal carcinoma cells for the study, which was funded by the National Science Foundation and the National Institutes for Health. More research is needed to determine that there are no serious side effects in humans and whether the technique will work on a variety of cancers, but Belfield is optimistic. "Predicting commercialization is difficult at best," he said. "But we are well situated to forge ahead".

So how did Belfield come up with such an "outside the box" approach? His other non-medical related research was the inspiration. Belfield has developed a three-dimensional, optical data-storage system, which involves the use of acid generators. About six years ago he wondered if his approach could have applications in medical therapy. "It took about five years to get someone in my research group interested to take on the unorthodox project," Belfield said. "But it seems to have paid off."

Other contributors to the research are Xiling Yue, Ciceron O. Yanez and Sheng Yao, researchers and students at UCF students focusing on chemistry or photonics.

Belfield is one of the pioneers in two-photon absorbing materials, two-photon photochemistry, and two-photon photophysics. His research spans a number of disciplines including organic, polymer, and physical chemistry, as well as optics, optical microscopy, and bioimaging. His research has potential applications in everything from the way people store data on DVDs to fighting cancer.

[http://www.eurekalert.org/pub\\_releases/2013-03/uog-nhw030713.php](http://www.eurekalert.org/pub_releases/2013-03/uog-nhw030713.php)

### **New hypothesis: Why bacteria are becoming increasingly more resistant to antibiotics**

*According to his theory, bacteria that are non-resistant to antibiotics acquire said resistance accidentally because they take up the DNA of others that are resistant, due to the stress to which they are subjected.*

A University of Granada researcher has formulated a new hypothesis concerning an enigma that the scientific community has still not been able to solve and which could revolutionise the pharmaceutical industry: Why are bacteria becoming increasingly more resistant to antibiotics? His work has revealed that the use of antibiotics can even cause non-resistant bacteria to become resistant because they take up the DNA of others that are already resistant.

Mohammed Bakkali, a scientist in the Genetics Department at the Faculty of Science of the UGR, maintains that our abuse of antibiotics "forces" the bacteria to take up the DNA of other bacteria that are resistant to said antibiotics, since the presence of antibiotics exposes them to a great stress. According to the researcher, "In this way, the non-resistant bacteria become resistant completely by accident on ingesting this DNA and can even become much more virulent, partly due to the stress we subject them to when we make an abusive use of antibiotics".

For decades, scientists from all over the world have been researching into when, how and why bacteria take up DNA from other antibiotic-resistant bacteria, thus becoming also resistant. The answers as to when there is DNA uptake (in unfavourable or stressful circumstances) and as to how the bacteria take it up are clear, but, up until now, "nobody has pinpointed the reason why bacteria ingest this genetic material", as Bakkali points out in an article published in the latest edition of the journal "Archives of Microbiology".

Under normal conditions, a bacterium could have a lot to lose if it 'decides' to take up DNA, since it does not have a 'DNA reader' enabling it to take up only those molecules that are of use to it and the most likely is that this DNA will be dangerous, or even lethal.

### **They do not want that DNA, because they break it up**

In his article, Mohammed Bakkali argues that, in reality, bacteria do not look for DNA to take up (they appear not to 'want' this DNA, since they are constantly degrading it; in other words, breaking it up) and that this uptake is a chance event and the sub-product of a type of bacterial motility that is part of its response to the stress that the bacteria may be subjected to.

Therefore, our current indiscriminate use of antibiotics "not only selects the resistant bacteria, but also means that the bacteria take up more DNA, due to their increased motility in response to the stress that the antibiotic subjects them to". The result is that the stress caused by the antibiotic itself induces the uptake of genetic material that can bring about resistance to the antibiotic by bacteria that, otherwise, would not have taken up that



DNA nor become resistant to the antibiotic. Furthermore, this effect is strengthened by its lack of specificity, since it occurs both in the target pathogen and in other bacteria.

The UGR researcher states that, when a bacterium takes up DNA from another antibiotic-resistant one (and which could have died due to another environmental factor), the bacterium that takes it up becomes resistant to that antibiotic. "Thus, the bacteria can go on adding to their arsenal of resistance to antibiotics and end up being resistant to a wide range of them, such as is the case of the multi-resistant strain of a staphylococcus, called *Staphylococcus aureus*, which creates havoc in many operating theatres.

*Reference: Could DNA Uptake Be a Side Effect of Bacterial Adhesion and Twitching Motility? M.Bakkali. Archives of Microbiology (Springer). DOI10.1007/s00203-013-0870-1*

[http://www.eurekalert.org/pub\\_releases/2013-03/uoc--itp030713.php](http://www.eurekalert.org/pub_releases/2013-03/uoc--itp030713.php)

### **Is this peptide a key to happiness?**

#### ***UCLA findings suggests possible new treatment for depression, other disorders***

What makes us happy? Family? Money? Love? How about a peptide?

The neurochemical changes underlying human emotions and social behavior are largely unknown. Now though, for the first time in humans, scientists at UCLA have measured the release of a specific peptide, a neurotransmitter called hypocretin, that greatly increased when subjects were happy but decreased when they were sad.

The finding suggests that boosting hypocretin could elevate both mood and alertness in humans, thus laying the foundation for possible future treatments of psychiatric disorders like depression by targeting measurable abnormalities in brain chemistry.

In addition, the study measured for the first time the release of another peptide, this one called melanin concentrating hormone, or MCH. Researchers found that its release was minimal in waking but greatly increased during sleep, suggesting a key role for this peptide in making humans sleepy.

The study is published in the March 5 online edition of the journal *Nature Communications*.

"The current findings explain the sleepiness of narcolepsy, as well as the depression that frequently accompanies this disorder," said senior author Jerome Siegel, a professor of psychiatry and director of the Center for Sleep Research at UCLA's Semel Institute for Neuroscience and Human Behavior. "The findings also suggest that hypocretin deficiency may underlie depression from other causes."

In 2000, Siegel's team published findings showing that people suffering from narcolepsy, a neurological disorder characterized by uncontrollable periods of deep sleep, had 95 percent fewer hypocretin nerve cells in their brains than those without the illness. The study was the first to show a possible biological cause of the disorder.

Since depression is strongly associated with narcolepsy, Siegel's lab began to explore hypocretin and its possible link to depression.

Depression is the leading cause of psychiatric disability in the U.S, Siegel noted. More than 6 percent of the population is affected each year, with lifetime prevalence exceeding 15 percent. Yet the use of antidepressants, such as selective serotonin reuptake inhibitors (SSRIs), has not been based on evidence of a deficiency, or excess, of any neurotransmitter. Several recent studies have questioned whether SSRIs, as well as other depression-fighting drugs, are any more effective than placebos.

In the current study, the researchers obtained their data on both hypocretin and MCH directly from the brains of eight patients who were being treated at Ronald Reagan UCLA Medical Center for intractable epilepsy. The patients had been implanted with intracranial depth electrodes by Dr. Itzhak Fried, a UCLA professor of neurosurgery and psychiatry and a co-author of the study, to identify seizure foci for potential surgical treatment. The location of electrodes was based solely on clinical criteria. The researchers, with the patients' consent, used these same electrodes to "piggyback" their research. A membrane similar to that used for kidney dialysis and a very sensitive radioimmunoassay procedure were used to measure the release of hypocretin and MCH.

The patients were recorded while they watched television; engaged in social interactions such as talking to physicians, nursing staff or family; ate; underwent various clinical manipulations; and experienced sleep-wake transitions. Notes of activities were made throughout the study every 15 minutes in synchrony with a 15-minute microdialysis sample collection by a researcher in the patients' rooms.

The subjects rated their moods and attitudes on a questionnaire, which was administered every hour during waking.

The researchers found that hypocretin levels were not linked to arousal in general but were maximized during positive emotions, anger, social interactions and awakening. In contrast, MCH levels were maximal during sleep onset and minimal during social interactions.

"These results suggest a previously unappreciated emotional specificity in the activation of arousal and sleep in humans," Siegel said. "The findings suggest that abnormalities in the pattern of activation of these systems may contribute to a number of psychiatric disorders."

Siegel noted that hypocretin antagonists are now being developed by several drug companies for use as sleeping pills. The current work suggests that these drugs will alter mood as well sleep tendency.

The Siegel lab has also previously reported that hypocretin is required for the "pursuit of pleasure" in rodents but plays no role in avoidance behavior.

"These results, in conjunction with the current findings, suggest that hypocretin administration will elevate both mood and alertness in humans," Siegel said.

*Other authors on the study were Ashley M. Blouin, Charles L. Wilson, Richard J. Staba, Eric J. Behnke, Hoa A. Lam, Nigel T. Maidment, Karl A. Karlsson and Jennifer L. Lapierre. Funding was provided by National Institutes of Health grants MH064109, NS14610, NS33310 and NS02808 and by the Medical Research Service of the Department of Veterans Affairs.*

[http://www.eurekalert.org/pub\\_releases/2013-03/uog-anm030713.php](http://www.eurekalert.org/pub_releases/2013-03/uog-anm030713.php)

## **A new material using doped carbon allows fuels to be produced while reducing CO2 emissions**

*Researchers from the University of Granada (UGR) have developed a new material using doped carbon that allows low-cost energy to be produced and also reduces the amount of CO2 released into the atmosphere.*

The recently-patented material is a gel that enables the CO2 to be turned back into hydrocarbons via electro-catalytic transformation, with great savings both in time and money

At present, power stations run using renewable energies (wind, solar or wave) produce energy peaks that are wasted, since they do not coincide with the energy needs. Storing this energy in batteries for its later use would be a very costly process that requires huge amounts of very expensive pure metals, such as nickel or copper, which is why this process is currently hardly ever used.

The doped carbon gel developed by the UGR acts as a highly-dispersed (it is made up of 90% carbon and a small quantity of heavy metals) and effective electro-catalyst, which means it enables CO2 to be turned into hydrocarbons at a low cost. This new material, developed entirely at the UGR, following more than 10 years of research into carbon gels, has recently been patented by the Institution's Office for the Transfer of Research Results (OTRI).

As the project's principal researcher, Agustin F. Perez Cadenas, explains, the doped carbon gel "is not a magical solution to prevent CO2 emissions into the atmosphere and stop the contamination caused by the greenhouse effect, but it does enable them to be reduced considerably, as well as reducing energy costs". At the moment, this system is in its laboratory phase and has still not been applied in actual power stations, though the tests carried out at the UGR have led to some "highly promising" results.

The research team currently working in this line of investigation is formed by the UGR lecturers Agustin F. Perez Cadenas, Carlos Moreno Castilla, Francisco Carrasco Marin, Francisco J. Maldonado Hodar and Sergio Morales Torres, along with Maria Perez Cadenas from the UNED. Initially, there was also another collaborator, Freek Kapteijn, from the TUDelft (Netherlands).

[http://www.eurekalert.org/pub\\_releases/2013-03/acoc-loa030613.php](http://www.eurekalert.org/pub_releases/2013-03/acoc-loa030613.php)

## **Lack of aspirin before angioplasty linked with higher mortality**

*Failure to follow basic aspirin protocol raises questions about adherence to other guidelines*

SAN FRANCISCO - Despite recommendations from leading medical groups, a surprising number of patients are not given aspirin before artery-clearing coronary angioplasty and stenting, and those patients have a significantly higher in-hospital death rate, according to research from a Michigan network being presented at the American College of Cardiology's 62nd Annual Scientific Session.

Aspirin use before angioplasty is a Class I recommendation of the American College of Cardiology and American Heart Association, the highest level of evidence for ACC/AHA guidelines. Aspirin has well-documented anti-platelet activity in reducing the risk of cardiac events.

Researchers evaluated registry data for 65,175 patients who had angioplasty and stenting, a percutaneous coronary intervention or PCI, at one of 42 hospitals enrolled in the Blue Cross Blue Shield of Michigan Cardiovascular Consortium PCI Quality Improvement Initiative from January 2010 through December 2011. Of these, 4,640 patients, or 7.1 percent, did not receive aspirin as recommended within 24 hours before PCI. Roughly 90 percent of the non-aspirin patients had no documented barriers to aspirin. Records did show that aspirin was withheld from some patients who had a history of gastrointestinal bleeding, even though such a history usually does not preclude pre-PCI aspirin.

"Our study is not designed to confirm a direct causal effect of aspirin use on PCI outcomes, but [rather] to examine any association with worse outcomes," said Mohamad Kenaan, MD, a cardiovascular medicine fellow with University of Michigan Health Systems, Ann Arbor, Mich., and the study's lead investigator. "Moreover, it highlights an unexpectedly significant number of patients undergoing PCI without receiving aspirin, despite the lack of a documented contraindication in the majority of cases—even in institutions that are active participants in an ongoing quality improvement initiative."

The in-hospital mortality rate of 3.9 percent for the non-aspirin group was considerably higher than the aspirin group's 1.2 percent, and the disadvantage remained after adjustment for confounding bias: death, 3.9 vs. 2.8 percent and stroke, 0.5 vs. 0.1 percent.

Those findings held across subgroups, including gender, age, type of coronary artery disease presentation and diabetes. The exception was cardiogenic shock—a state that presents challenges for use of oral medicines like aspirin. There was no difference between the groups in bleeding, need for transfusions or kidney damage caused by imaging contrast agent.

Additional data analysis will address other subgroups, hospital length of stay and bleeding. The registry does not contain post-discharge data for longer-term follow-up.

"The strong association our study demonstrated between aspirin non-use before PCI and worse outcomes, including in-hospital death, across all types of ischemic heart disease should be used as a platform for more studies to confirm our findings and motivate quality efforts focused on optimizing aspirin use before PCI," Dr. Kenaan said. "Our findings also may indicate lack of adherence to other guidelines, thus leading to worse outcomes."

*This research was funded by the Blue Cross Blue Shield of Michigan Cardiovascular Consortium Percutaneous Coronary Intervention Quality Improvement Initiative.*

<http://www.bbc.co.uk/news/technology-21697704>

### **Frozen Android phones give up data secrets**

*Freezing an Android phone can help reveal its confidential contents, German security researchers have found.*

The team froze phones for an hour as a way to get around the encryption system that protects the data on a phone by scrambling it.

Google introduced the data scrambling system with the version of Android known as Ice Cream Sandwich. The attack allowed the researchers to get at contact lists, browsing histories and photos.

#### **Cold start**

Android's data scrambling system was good for end users but a "nightmare" for law enforcement and forensics workers, the team at Erlangen's Friedrich-Alexander University (FAU) wrote in a blogpost about their work. To get around this, researchers Tilo Muller, Michael Spreitzenbarth and Felix Freiling from FAU put Android phones in a freezer for an hour until the device had cooled to below -10C.

The trio discovered that quickly connecting and disconnecting the battery of a frozen phone forced the handset into a vulnerable mode. This loophole let them start it up with some custom-built software rather than its onboard Android operating system.

The researchers dubbed their custom code Frost - Forensic Recovery of Scrambled Telephones.

The Frost software helped them copy data on a phone that could then be analysed on a separate computer.

A chilled phone also helped their hacking project. Data fades from memory much more slowly when chips are cold which allowed them to grab the encryption keys and speed up unscrambling the contents of a phone.

PhD student Tilo Muller told the BBC that the attack generally gave them access to data that had been put in memory as users browsed websites, sent messages or shared pictures.

The researchers tested their attack against a Samsung Galaxy Nexus handset as it was one of the first to use Android's disk encryption system.

However, they said, other phones were just as likely to be vulnerable to the attack. The team are planning further tests on other Android handsets.

While the "cold boot" attack had been tried on desktop PCs and laptops, Mr Muller said the trio were the first to try it on phones. "We thought it would work because smartphones are really small PCs," he said. "but we were quite excited that the trick with the freezer worked so well."

The German research group is now working on defences against the attack that ensures encryption keys are never put in vulnerable memory chips.

Instead they are only used in the memory directly attached to a phone's processor.

<http://phys.org/news/2013-03-revealed-earth-electrical-heartbeat-clouds.html>

## **Revealed: The Earth's 'electrical heartbeat' seen in clouds**

*The height of clouds changes by up to 200m during a day under the influence of a global 'electrical heartbeat' in the atmosphere, scientists at the University of Reading have discovered.*

Phys.org - The findings, made by analysing 10 years' data of cloud heights from the north and south poles, open up a whole new perspective on our understanding of how clouds form and influence our weather and climate. Scientists have been aware of the daily global ebb and flow of electric current through the atmosphere for 100 years, when it was shown to vary consistently throughout the day wherever on the planet it was measured. This regular variation, effectively a global electrical heartbeat, is known as the Carnegie curve, after the ship whose cruises provided the defining experiments in the 1920s.

The electric current is caused by electrified storms across the world. Its daily peak occurs at 7pm GMT each day when the major sources of thunderstorms are the American and African landmasses. The current is usually weakest at 3am GMT, night-time across most of the world's continents, when there are fewest thunderstorms occurring globally.

Previously no connection had been made between this current and the formation of clouds. But, by analysing cloud base measurements made during polar darkness when there are few other influences on cloud formation, University of Reading meteorologists Professor Giles Harrison and Dr Maarten Ambaum found evidence for the first time that cloud heights are closely linked to the Carnegie curve.

Professor Harrison said: "What we found was remarkable. The variations from both north and south poles are almost identical, suggesting a strong link with the Carnegie curve, when other factors are taken out of the equation. This may arise from charging of small droplets in the cloud's base, encouraging them to stick together. "This implies that factors inside or outside the climate system which change the global electric current, such as ocean temperatures or cosmic rays, may influence the properties of layer clouds. However our results say nothing about any long-term effects, as they were found for rapidly-occurring changes from hour to hour." Layer clouds are particularly relevant to global temperatures. At night they act like a warm blanket, preventing heat from being lost from the earth into space, and during the day help cool the surface by reflecting away the sun's energy.

"The realisation the electrical heartbeat of the planet plays a role in the formation of layer clouds indicates that existing models for clouds and climate are still missing potentially important components," said Dr Ambaum. "Understanding these missing elements is crucial to improve the accuracy of our weather forecasts and predicting changes to our climate. The climate system keeps on surprising us with its immense complexity and richness." The findings are published in the journal *Environmental Research Letters*.

*More information: Harrison, G. and Ambaum, M. 2013 Electrical signature in polar night cloud base variations, Environ. Res. Lett. 8 015027 [iopscience.iop.org/1748-9326/8/1/015027/article](http://iopscience.iop.org/1748-9326/8/1/015027/article)*

<http://www.scientificamerican.com/article.cfm?id=metal-oxide-chips-show-promise-as-transistors>

## **Metal Oxide Chips Show Promise as Transistors**

*Materials that flip from insulator to conductor could make more energy-efficient transistors, although the metals are not yet close to competing with silicon*

By Eugenie Samuel Reich and Nature magazine | Thursday, March 7, 2013 | 16

The switches in most electronic circuits are made of silicon, one of the commonest elements. But their successors might contain materials that, for now, are lab-grown oddities: strongly correlated metal oxides. The allure of these materials lies in the outer shells of electrons surrounding their metal atoms. The shells are incomplete, leaving the electrons free to participate in coordinated quantum-mechanical behavior. In some materials, electrons pair up to produce super-conductivity, or coordinate their spins to produce magnetism. Other materials can switch from being an insulator to a conductor.

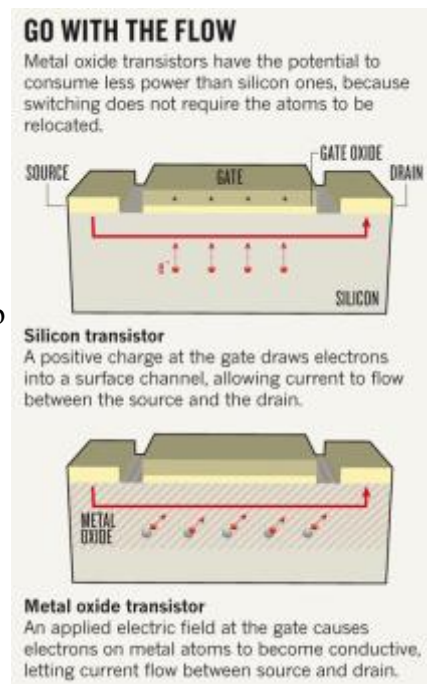
Unlike transitions to superconductivity, which happen as temperatures approach absolute zero, the insulating-to-conducting transition typically happens as temperature increases, and sometimes occurs near room temperature. That has raised hopes that metal oxides could be used instead of silicon to make transistors. A spate of results is now making that look feasible. "People are interested in seeing if oxides can make it to applications," says Manuel Bibes, a physicist at the Joint Physics Unit in Palaiseau, France, which is run by the French National Research Center and electronics company Thales.

Metal oxide transistors have the potential to consume less power than silicon switches, because the phase transition frees electrons from their localized state near each atom, without moving them through the bulk material. By contrast, silicon switches work by pulling electrons through the material to a channel where they conduct current.

In the past 5–10 years, researchers have succeeded in growing high-quality thin films of the metal oxides — overcoming one of the major barriers to applications. In July 2012, for example, a group in Japan reported that it had deposited a thin film of vanadium dioxide that underwent a phase transition in response to an applied electric field — proof that the material could be used as an electronic switch. And last month, a group led by Shriram Ramanathan, a materials scientist at Harvard University in Cambridge, Massachusetts, addressed a fabrication challenge by growing a thin film of samarium nickelate on top of a substrate made of silicon and silicon dioxide.

The nickelate was deposited at a relatively low temperature that did not disturb the underlying silicon layers, raising the possibility of manufacturing metal oxides on top of silicon wafers to form three-dimensional chips, says Andrew Millis, a solid-state theorist at Columbia University in New York. Not only would that allow computing power to be packed much more densely, says Millis, but it would also permit metal oxide switches to be built on top of existing circuit architectures.

Other groups are trying to understand the nature of the phase transition. In January, Ivan Schuller, a solid-state physicist at the University of California, San Diego, and his colleagues showed that in vanadium oxide, the transition is in large part caused by micrometer-scale heating by the applied electric field.



*Go with the Flow Image: Courtesy of Nature Magazine*

Some point to Schuller's work as evidence that metal oxides will never make fast switches, because heating effects are usually quite slow. But Ramanathan says that his own measurements on vanadium oxide demonstrate that the phase transition is quite fast — less than a few nanoseconds — and that it should not hinder applications.

Some physicists are finding further examples of potentially useful materials. Bernhard Keimer at the Max Planck Institute for Solid State Research in Stuttgart, Germany, alternates thin layers of metal oxides to form composites that often turn out to have serendipitous properties. His group layered conducting lanthanum nickelate and insulating lanthanum aluminate and found that the composite underwent a transition between the two properties.

The highest phase-transition temperature for the composite was 150 kelvin above absolute zero — too low for practical applications. But the group is now trying to replicate the phenomenon in other materials that might have higher transition temperatures.

Sandip Tiwari, an applied physicist at Cornell University in Ithaca, New York, acknowledges that metal oxides are not yet close to competing with silicon. But given recent progress, he feels that researchers need to start trying to implement them in devices. That way, he says, all the properties needed for a good transistor will be developed in tandem. "If you just look at whatever property is your favorite, you won't get them all."

<http://www.sciencedaily.com/releases/2013/03/130307145103.htm>

### Hubble Finds 'Birth Certificate' of Oldest Known Star

*A team of astronomers using NASA's Hubble Space Telescope has taken an important step closer to finding the birth certificate of a star that's been around for a very long time.*

"We have found that this is the oldest known star with a well-determined age," said Howard Bond of Pennsylvania State University in University Park, Pa., and the Space Telescope Science Institute in Baltimore, Md. The star could be as old as 14.5 billion years (plus or minus 0.8 billion years), which at first glance would make it older than the universe's calculated age of about 13.8 billion years, an obvious dilemma.

But earlier estimates from observations dating back to 2000 placed the star as old as 16 billion years. And this age range presented a potential dilemma for cosmologists. "Maybe the cosmology is wrong, stellar physics is wrong, or the star's distance is wrong," Bond said. "So we set out to refine the distance."

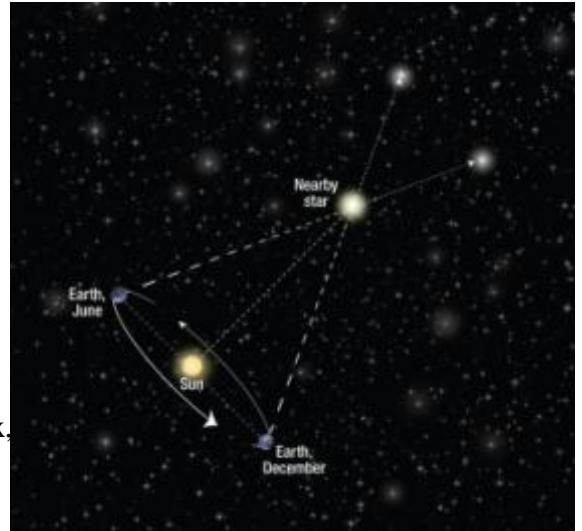
The new Hubble age estimates reduce the range of measurement uncertainty, so that the star's age overlaps with the universe's age -- as independently determined by the rate of expansion of space, an analysis of the microwave background from the big bang, and measurements of radioactive decay.

This "Methuselah star," cataloged as HD 140283, has been known about for more than a century because of its fast motion across the sky. The high rate of motion is evidence that the star is simply a visitor to our stellar neighborhood. Its orbit carries it down through the plane of our galaxy from the ancient halo of stars that encircle the Milky Way, and will eventually slingshot back to the galactic halo.

This conclusion was bolstered by the 1950s astronomers who were able to measure a deficiency of heavier elements in the star as compared to other stars in our galactic neighborhood. The halo stars are among the first inhabitants of our galaxy and collectively represent an older population from the stars, like our Sun, that formed later in the disk. This means that the star formed at a very early time before the universe was largely "polluted" with heavier elements forged inside stars through nucleosynthesis. (The Methuselah star has an anemic 1/250th as much of the heavy element content of our Sun and other stars in our solar neighborhood.)

The star, which is at the very first stages of expanding into a red giant, can be seen with binoculars as a 7th-magnitude object in the constellation Libra.

Hubble's observational prowess was used to refine the distance to the star, which comes out to be 190.1 light-years. Bond and his team performed this measurement by using trigonometric parallax, where an apparent shift in the position of a star is caused by a change in the observer's position. The results are published in the March 1 issue of the *Astrophysical Journal Letters*.



*This is a Digitized Sky Survey image of the oldest star with a well-determined age in our galaxy. The aging star, cataloged as HD 140283, lies 190.1 light-years away. Hubble Space Telescope was used to narrow the measurement uncertainty on the star's distance, and this helped refine the calculation of a more precise age of 14.5 billion years (plus or minus 800 million years). The star is rapidly passing through our local stellar neighborhood. The star's orbit carries it through the plane of our galaxy from the galactic halo that has a population of ancient stars. The Anglo-Australian Observatory (AAO) UK Schmidt telescope photographed the star in blue light. (Credit: Digitized Sky Survey (DSS), STScI/AURA, Palomar/Caltech, and UKSTU/AAO)*

The parallax of nearby stars can be measured by observing them from opposite points in Earth's orbit around the Sun. The star's true distance from Earth can then be precisely calculated through straightforward triangulation. Once the true distance is known, an exact value for the star's intrinsic brightness can be calculated. Knowing a star's intrinsic brightness is a fundamental prerequisite to estimating its age.

Before the Hubble observation, the European Space Agency's Hipparcos satellite made a precise measurement of the star's parallax, but with an age measurement uncertainty of 2 billion years. One of Hubble's three Fine Guidance Sensors measured the position of the Methuselah star. It turns out that the star's parallax came out to be virtually identical to the Hipparcos measurements. But Hubble's precision is five times better than that of Hipparcos. Bond's team managed to shrink the uncertainty so that the age estimate was five times more precise. With a better handle on the star's brightness Bond's team refined the star's age by applying contemporary theories about the star's burn rate, chemical abundances, and internal structure. New ideas are that leftover helium diffuses deeper into the core and so the star has less hydrogen to burn via nuclear fusion. This means it uses fuel faster and that correspondingly lowers the age.

Also, the star has a higher than predicted oxygen-to-iron ratio, and this too lowers the age. Bond thinks that further oxygen measurement could reduce the star's age even more, because the star would have formed at a slightly later time when the universe was richer in oxygen abundance. Lowering the upper age limit would make the star unequivocally younger than the universe.

"Put all of those ingredients together and you get an age of 14.5 billion years, with a residual uncertainty that makes the star's age compatible with the age of the universe," said Bond. "This is the best star in the sky to do precision age calculations by virtue of its closeness and brightness."

This Methuselah star has seen many changes over its long life. It was likely born in a primeval dwarf galaxy. The dwarf galaxy eventually was gravitationally shredded and sucked in by the emerging Milky Way over 12 billion years ago.

The star retains its elongated orbit from that cannibalism event. Therefore, it's just passing through the solar neighborhood at a rocket-like speed of 800,000 miles per hour. It takes just 1,500 years to traverse a piece of sky with the angular width of the full Moon. The star's proper motion angular rate is so fast (0.13 milliarcseconds an hour) that Hubble could actually photograph its movement in a few hours.

*Howard E. Bond, Edmund P. Nelan, Don A. VandenBerg, Gail H. Schaefer, Dianne Harmer. HD 140283: A Star In The Solar Neighborhood That Formed Shortly After The Big Bang. The Astrophysical Journal, 2013; 765 (1): L12 DOI: 10.1088/2041-8205/765/1/L12*

<http://www.sciencedaily.com/releases/2013/03/130307160325.htm>

## Nanoparticles Loaded With Bee Venom Kill HIV

*Nanoparticles carrying a toxin found in bee venom can destroy human immunodeficiency virus (HIV) while leaving surrounding cells unharmed, researchers at Washington University School of Medicine in St. Louis have shown.*

The finding is an important step toward developing a vaginal gel that may prevent the spread of HIV, the virus that causes AIDS.

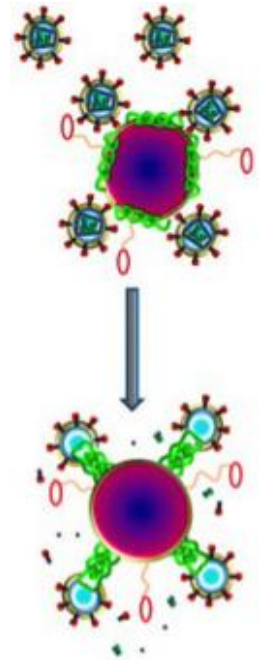
"Our hope is that in places where HIV is running rampant, people could use this gel as a preventive measure to stop the initial infection," says Joshua L. Hood, MD, PhD, a research instructor in medicine.

The study appears in the current issue of *Antiviral Therapy*.

Bee venom contains a potent toxin called melittin that can poke holes in the protective envelope that surrounds HIV, and other viruses. Large amounts of free melittin can cause a lot of damage. Indeed, in addition to anti-viral therapy, the paper's senior author, Samuel A. Wickline, MD, the J. Russell Hornsby Professor of Biomedical Sciences, has shown melittin-loaded nanoparticles to be effective in killing tumor cells.

The new study shows that melittin loaded onto these nanoparticles does not harm normal cells. That's because Hood added protective bumpers to the nanoparticle surface. When the nanoparticles come into contact with normal cells, which are much larger in size, the particles simply bounce off. HIV, on the other hand, is even smaller than the nanoparticle, so HIV fits between the bumpers and makes contact with the surface of the nanoparticle, where the bee toxin awaits.

*Nanoparticles (purple) carrying melittin (green) fuse with HIV (small circles with spiked outer ring), destroying the virus's protective envelope. Molecular bumpers (small red ovals) prevent the nanoparticles from harming the body's normal cells, which are much larger in size. (Credit: Joshua L. Hood, MD, PhD)*



"Melittin on the nanoparticles fuses with the viral envelope," Hood says. "The melittin forms little pore-like attack complexes and ruptures the envelope, stripping it off the virus."

According to Hood, an advantage of this approach is that the nanoparticle attacks an essential part of the virus' structure. In contrast, most anti-HIV drugs inhibit the virus's ability to replicate.

But this anti-replication strategy does nothing to stop initial infection, and some strains of the virus have found ways around these drugs and reproduce anyway.

"We are attacking an inherent physical property of HIV," Hood says. "Theoretically, there isn't any way for the virus to adapt to that. The virus has to have a protective coat, a double-layered membrane that covers the virus." Beyond prevention in the form of a vaginal gel, Hood also sees potential for using nanoparticles with melittin as therapy for existing HIV infections, especially those that are drug-resistant.

The nanoparticles could be injected intravenously and, in theory, would be able to clear HIV from the blood stream.

"The basic particle that we are using in these experiments was developed many years ago as an artificial blood product," Hood says. "It didn't work very well for delivering oxygen, but it circulates safely in the body and gives us a nice platform that we can adapt to fight different kinds of infections."

Since melittin attacks double-layered membranes indiscriminately, this concept is not limited to HIV. Many viruses, including hepatitis B and C, rely on the same kind of protective envelope and would be vulnerable to melittin-loaded nanoparticles.

While this particular paper does not address contraception, Hood says the gel easily could be adapted to target sperm as well as HIV. But in some cases people may only want the HIV protection.

"We also are looking at this for couples where only one of the partners has HIV, and they want to have a baby," Hood says. "These particles by themselves are actually very safe for sperm, for the same reason they are safe for vaginal cells."

While this work was done in cells in a laboratory environment, Hood and his colleagues say the nanoparticles are easy to manufacture in large enough quantities to supply them for future clinical trials.

*This work was supported by the Bill & Melinda Gates Foundation Grand Challenges Explorations grant number OPP1024642 'Fusogenic nanoparticles for combined anti-HIV/contraception.'*

*Joshua L Hood, Andrew P Jallouk, Nancy Campbell, Lee Ratner, Samuel A Wickline. Cytolytic nanoparticles attenuate HIV-1 infectivity. Antiviral Therapy, 2012; 18 (1): 95 DOI: 10.3851/IMP2346*

<http://www.scientificamerican.com/article.cfm?id=what-antarctica-looked-like>

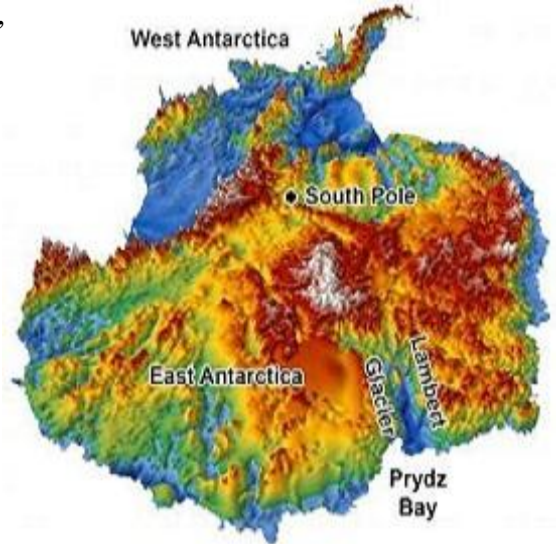
## What Antarctica Looked Like before the Ice

*Antarctica was flat, warm and crisscrossed with rivers before glaciers buzz-sawed its steep valley*

By Becky Oskin and OurAmazing Planet | Thursday, March 7, 2013 | 4

Like Alaska's mighty Yukon, a broad river once flowed across Antarctica, following a gentle valley shaped by tectonic forces at a time before the continent became encased in ice. Understanding what happened when rivers of ice later filled the valley could solve certain climate and geologic puzzles about the southernmost continent. The valley is Lambert Graben in East Antarctica, now home to the world's largest glacier. Trapped beneath the ice, the graben (which is German for ditch or trench) is a stunning, deep gorge. But before Antarctica's deep freeze 34 million years ago, the valley was relatively flat and filled by a lazy river, leaving a riddle for geologists to decode: How did Lambert Graben get so steep, and when was it carved?

The key to Lambert Graben's history was found in layers of sediments just offshore, in Prydz Bay. In a new study, Stuart Thomson, a geologist at the University of Arizona (UA) in Tucson, looked into the past by decoding sands deposited by the river, and the messy piles left behind by the glacier. The river sands are topped with a thick layer of coarser sediment that signals the onset of glacial erosion in the valley, the researchers found. The erosion rate more than doubled when the glaciers moved in, Thomson said. "The only way that could happen is from glaciers," he said. "They started grinding and forming deep valleys."



*This 3-D reconstruction of the topography hidden under Antarctica's two-mile-thick coating of ice was made using data from radar surveys. Image: Stuart N. Thomson/UA department of geosciences*

Understanding when glaciers first wove their way across Antarctica will help scientists better model the ice sheet's response to Earth's climate shifts, the researchers said.

"There's a big effort to model how glaciers flow in Antarctica, and these models need a landscape over which glaciers can flow," Thomson told OurAmazingPlanet. "Once these models can predict past changes, they can more accurately predict what will happen with future climate changes."

The sediments also hold clues to the tectonic evolution of East Antarctica, and a mountain range buried beneath the vast, thick ice sheet. The findings are detailed in the March 2013 issue of the journal *Nature Geoscience*.

### History of the ice

Lambert Graben formed during the breakup of Gondwana, an ancient supercontinent, a process that happened in stages. Antarctica, India and Africa tore apart in the Late Cretaceous (about 80 million years ago). The split created long, linear valleys oriented perpendicular to the continental coastlines. At the time, Earth's climate was warmer than it is today, and as Antarctica moved southward, settling into its home over the South Pole, the continent teemed with plants and animals.

Scientists can partially reconstruct this past environment with fossils and through radar that peers beneath the ice to map the shapes of the rock below. A 3D map of Antarctica today shows chasms carved by glaciers, rugged mountains and other remnants of its warmer existence. But the surveys tell nothing about how the landscape looked before the ice carved out all those features. "People have speculated when the big fjords formed under the ice," Thomson said. "But no one knows for sure until you sample the rocks or the sediments." Thomson and his colleagues analyzed sediments drilled from the ocean floor just offshore of Lambert Glacier, as well as from onshore moraines, the rock piles pushed up by glaciers. Tests on minerals in the sands and muds helped them figure out when and how fast the surface eroded.

Here's what the sediments say: From about 250 million to 34 million years ago, the region around Lambert Glacier was relatively flat, and drained by slow-moving rivers, Thomson said. About 34 million years ago, which coincides with a cooling of Earth's climate, big glaciers appeared, shaping the spectacular valley now hidden under thick ice. "It seemed like it occurred very early on, 34 [million] to 24 million years ago," Thomson said. Erosion slowed dramatically as the ice sheet stabilized about 15 million years ago, he said. Some 5,250 to 8,200 feet (1.6 to 2.5 kilometers) of rock have since disappeared, ground down by glaciers and carried away by the ice, according to the study. "Glaciers can carve deep valleys quickly — and did so on Antarctica before it got so cold that the most of it got covered by 1 or 2 miles [1.6 to 3.2 km] of thick, stationary ice," Peter Reiners, a UA geologist and study co-author, said in a statement.



### Clues to buried mountain range

Lambert Graben extends about 375 miles (600 km) inland, ending at one of Antarctica's most enigmatic features — an entombed mountain range called the Gamburtsev Mountains. Buried under the ice, the mountains rose during Gondwana's rifting. Geologic evidence suggests two pulses of uplift from rifting events about 250 million years ago and 100 million years ago pushed up the jagged peaks.

But Thomson and his colleagues did not find evidence in the sediments for a second uplift phase 100 million years ago. The river sands contain minerals from the Gamburtsev Mountains, and the tiny grains suggest the mountains got their height with one tectonic push.

"This underscores both the mountain range's remarkable age and the extraordinary degree of subglacial landscape preservation," writes Darrel Swift in an accompanying article in *Nature Geoscience*. Swift, a geologist at the University of Sheffield in the United Kingdom, was not involved in the study.

<http://www.sciencedaily.com/releases/2013/03/130307110301.htm>

### Test-Taking May Improve Learning in People of All Ages

*Older adults who haven't been in school for a while are as capable of learning from tests as younger adults and college students, according to new research published by the American Psychological Association.*

No matter their age or if they work or go to college full time, people appear to learn more when tested on material, rather than simply rereading or restudying information, according to research published online in the APA journal *Psychology and Aging*.

"The use of testing as a way to learn new information has been thoroughly examined in young students. This research builds on that and supports the notion that educators, or even employers, can use tests to increase learning in adults of all ages," said the study's lead author, Ashley Meyer, PhD, a cognitive psychologist with the Houston Veterans Affairs Health Services Research and Development Center of Excellence.

In this experiment, adults of various ages improved their retention of new information just as much as college students if they were tested on the material and received feedback on their scores, rather than just restudying the materials, according to the article. The improvement was significant and comparable to the college students' improvement, even though the college students performed better on the initial test.

"Both groups benefited from the initial testing more than the additional studying. Taking the test and then being told how many answers they got wrong or correct was enough for these adults to improve their memory of the material as shown in a final, more difficult, test," said Meyer, who conducted the research with co-author Jessica Logan, PhD, at Rice University.

Participants who took the final test on the same day as the study period did significantly better than participants who took it two days later, according to the article. However, older adults whose memories presumably are not as good as that of young college students still showed improved memory for previously tested material compared to restudied material, even after the two-day delay.

The sample consisted of 60 college students, age 18 to 25, 60 younger adults, age 18 to 25, and 60 older adults, age 55 to 65, either attending school or living in the Houston area. The students were recruited online from Rice University and received partial course credit while the other participants were recruited online and via community flyers and received a small payment. All participants took an intelligence test before starting the experiment.

Participants had 15 minutes to study and read materials on the following four topics: tsunamis, armadillos, the human heart and black holes. After completing some math problems, which served as a distraction from what they had read, the participants completed a multiple-choice test on two of the topics. They received feedback on their performance from researchers (e.g., "You got 14 out of 20 questions correct.").

In addition to taking the multiple-choice test, the participants restudied the other two topics that had not been included in the test.

After completing another set of math problems, some participants took the final test right away while others took it two days later. This final test covered all four topics and was more difficult since it required participants to write answers rather than select from multiple choices.

Since all participants had some college education, the authors suggest that future research should look at adults with less formal education to see if this testing benefit is seen across educational backgrounds and age groups.

"Working adults often need to gain new skills or knowledge as they advance through their careers," said Meyer.

"Our research suggests that testing may be one way to help them improve and move up."

Ashley N. D. Meyer, Jessica M. Logan. *Taking the Testing Effect Beyond the College Freshman: Benefits for Lifelong Learning.* *Psychology and Aging*, 2013; DOI: 10.1037/a0030890

<http://www.sciencedaily.com/releases/2013/03/130307124754.htm>

## Star-Shaped Glial Cells Act as the Brain's 'Motherboard'

*The transistors and wires that power our electronic devices need to be mounted on a base material known as a "motherboard."*

Our human brain is not so different -- neurons, the cells that transmit electrical and chemical signals, are connected to one another through synapses, similar to transistors and wires, and they need a base material too. But the cells serving that function in the brain may have other functions as well. PhD student Maurizio De Pittà of Tel Aviv University's Schools of Physics and Astronomy and Electrical Engineering says that astrocytes, the star-shaped glial cells that are predominant in the brain, not only control the flow of information between neurons but also connect different neuronal circuits in various regions of the brain.

Using models designed to mimic brain signalling, De Pittà's research, led by his TAU supervisor Prof. Eshel Ben-Jacob, determined that astrocytes are actually "smart" in addition to practical. They integrate all the different messages being transferred through the neurons and multiplexing them to the brain's circuitry. Published in the journal *Frontiers in Computational Neuroscience* and sponsored by the Italy-Israel Joint Neuroscience Lab, this research introduces a new framework for making sense of brain communications -- aiding our understanding of the diseases and disorders that impact the brain.

### Transcending boundaries

"Many pathologies are related to malfunctions in brain connectivity," explains Prof. Ben-Jacob, citing epilepsy as one example. "Diagnosis and the development of therapies rely on understanding the network of the brain and the source of undesirable activity."

Connectivity in the brain has traditionally been defined as point-to-point connections between neurons, facilitated by synapses. Astrocytes serve a protective function by encasing neurons and forming borders between different areas of the brain. These cells also transfer information more slowly, says Prof. Ben-Jacob -- one-tenth of a second compared to one-thousandth of a second in neurons -- producing signals that carry larger amounts of information over longer distances. Astrocytes can transfer information regionally or spread it to different areas throughout the brain -- connecting neurons in a different manner than conventional synapses. De Pittà and his fellow researchers developed computational models to look at the different aspects of brain signalling, such as neural network electrical activity and signal transfer by synapses. In the course of their research, they discovered that astrocytes actually take an active role in the way these signals are distributed, confirming theories put forth by leading experimental scientists.

Astrocytes form additional networks to those of the neurons and synapses, operating simultaneously to coordinate information from different regions of the brain -- much like an electrical motherboard functions in a computer, or a conductor ensuring that the entire orchestra is working in harmony, explains De Pittà.

These findings should encourage neuroscientists to think beyond neuron-based networks and adopt a more holistic view of the brain, he suggests, noting that the two communication systems are actually interconnected, and the breakdown of one can certainly impact the other. And what may seem like damage in one small area could actually be carried to larger regions.

### A break in communication

According to Prof. Ben-Jacob, a full understanding of the way the brain sends messages is significant beyond satisfying pure scientific curiosity. Many diseases and disorders are caused by an irregularity in the brain's communication system or by damage to the glial cells, so more precise information on how the network functions can help scientists identify the cause or location of a breakdown and develop treatments to overcome the damage.

In the case of epilepsy, for example, the networks frequently become overexcited. Alzheimer's disease and other memory disorders are characterized by a loss of cell-to-cell connection. Further understanding brain connectivity can greatly aid research into these and other brain-based pathologies.

*Maurizio De Pittà, Vladislav Volman, Hugues Berry, Vladimir Parpura, Andrea Volterra, Eshel Ben-Jacob. Computational quest for understanding the role of astrocyte signaling in synaptic transmission and plasticity. Frontiers in Computational Neuroscience, 2012; 6 DOI: 10.3389/fncom.2012.00098*

[http://www.eurekalert.org/pub\\_releases/2013-03/uons-adb030813.php](http://www.eurekalert.org/pub_releases/2013-03/uons-adb030813.php)

## Anti-aging drug breakthrough

*Drugs that combat ageing may be available within five years, following landmark work led by an Australian researcher.*

The work, published in the March 8 issue of *Science*, finally proves that a single anti-ageing enzyme in the body can be targeted, with the potential to prevent age-related diseases and extend lifespans. The paper shows all of the 117 drugs tested work on the single enzyme through a common mechanism. This means that a whole

new class of anti-ageing drugs is now viable, which could ultimately prevent cancer, Alzheimer's disease and type 2 diabetes. "Ultimately, these drugs would treat one disease, but unlike drugs of today, they would prevent 20 others," says the lead author of the paper, Professor David Sinclair, from UNSW Medicine, who is based at Harvard University. "In effect, they would slow ageing."

The target enzyme, SIRT1, is switched on naturally by calorie restriction and exercise, but it can also be enhanced through activators. The most common naturally-occurring activator is resveratrol, which is found in small quantities in red wine, but synthetic activators with much stronger activity are already being developed. Although research surrounding resveratrol has been going for a decade, until now the basic science had been contested. Despite this, there have already been promising results in some trials with implications for cancer, cardiovascular disease and cardiac failure, type 2 diabetes, Alzheimer's and Parkinson's diseases, fatty liver disease, cataracts, osteoporosis, muscle wasting, sleep disorders and inflammatory diseases such as psoriasis, arthritis and colitis (inflammatory bowel disease). "In the history of pharmaceuticals, there has never been a drug that tweaks an enzyme to make it run faster," says Professor Sinclair, a geneticist with the Department of Pharmacology at UNSW.

The technology was sold to pharmaceutical giant GlaxoSmithKline in 2008[i]. Four thousand synthetic activators, which are 100 times as potent as a single glass of red wine, have been developed – the best three are in human trials. "Our drugs can mimic the benefits of diet and exercise, but there is no impact on weight," says Professor Sinclair, who suggests the first therapeutic to be marketed will be for diabetes.

There have been limited trials in people with type 2 diabetes and the skin inflammatory disease, psoriasis. There were benefits to the metabolism in the first group and a reduction in skin redness in the second.

The drugs can be administered orally, or topically. So far, there have been no drugs developed targeting ageing skin, but one major skin care range has developed a crème with resveratrol in it.

While any drug would be strictly prescribed for certain conditions, Professor Sinclair suggests that one day, they could be taken orally as a preventative. This would be in much the same way as statin drugs are commonly prescribed to prevent, instead of simply treating, cardiovascular disease. In animal models, overweight mice given synthetic resveratrol were able to run twice as far as slim mice and they lived 15 per cent longer.

"Now we are looking at whether there are benefits for those who are already healthy. Things there are also looking promising," says Professor Sinclair, who also heads the Lowy Cancer Research Centre's Laboratory for Ageing Research at UNSW. "We're finding that ageing isn't the irreversible affliction that we thought it was," he says. "Some of us could live to 150, but we won't get there without more research."

*[i] Professor Sinclair formed a started up company Sirtris to develop the anti-ageing technology. This was subsequently sold to GlaxoSmithKline (GSK). Professor Sinclair is now a scientific advisor to GSK. Several other authors on the paper work for GSK or an affiliated company.*

[http://www.eurekalert.org/pub\\_releases/2013-03/au-vag030813.php](http://www.eurekalert.org/pub_releases/2013-03/au-vag030813.php)

### **Virus and genes involved in causation of schizophrenia**

***For the first time, an international team of researchers has found that a combination of a particular virus in the mother and a specific gene variant in the child increases the risk of the child developing schizophrenia***

Viruses and genes interact in a way that may increase the risk of developing schizophrenia significantly. This happens already in the developing foetus.

An international team of scientists led by Aarhus University, Denmark, has made this discovery. As the first in the world, they scanned the entire genome of hundreds of sick and healthy people to see if there is an interaction between genes and a very common virus - cytomegalovirus - and to see whether the interaction influences the risk of developing schizophrenia. And it does.

Women that have been infected by the virus - and around 70 % has - will have a statistically significant increased risk of giving birth to a child who later develops schizophrenia if the child also has the aforementioned gene variant. This variant is found in 15 percent. The risk is five times higher than usual, the researchers report in *Molecular Psychiatry*.

#### **No cause for alarm**

People infected with cytomegalovirus most often do not know it, as the infection by the virus, which belongs to the herpes virus family, is usually very mild. But the researchers stress that there is no cause for alarm - even if both risk factors are present in mother and child, there may be a variety of other factors that prevents disease development in the child.

But as schizophrenia affects 1 per cent of the global population, this new knowledge is very important.

"In the longer term, the development of an effective vaccine against cytomegalovirus may help to prevent many cases of schizophrenia," says Professor of Medical Genetics at Aarhus University, Anders Børghlum.

"And our discovery emphasizes that mental disorders such as schizophrenia may arise in the context of an interaction between genes and biological environmental factors very early in life."

Read the article [Genome-wide study of association and interaction with maternal cytomegalovirus infection suggests new schizophrenia loci](#)

**FACTS:** The study, which includes genetic investigations of almost 10,000 people, comes from the work of a Danish interdisciplinary research project iPSYCH led by Aarhus University, and Research Centre iSEQ at Aarhus University. It was carried out in collaboration with Statens Serum Institut, Copenhagen University, and researchers from the U.S., Germany and Holland.

[http://www.eurekalert.org/pub\\_releases/2013-03/kcl-btr030813.php](http://www.eurekalert.org/pub_releases/2013-03/kcl-btr030813.php)

### **Biological tooth replacement -- a step closer**

*Scientists have developed a new method of replacing missing teeth with a bioengineered material generated from a person's own gum cells*

Scientists have developed a new method of replacing missing teeth with a bioengineered material generated from a person's own gum cells. Current implant-based methods of whole tooth replacement fail to reproduce a natural root structure and as a consequence of the friction from eating and other jaw movement, loss of jaw bone can occur around the implant. The research is led by Professor Paul Sharpe, an expert in craniofacial development and stem cell biology at King's College London and published in the Journal of Dental Research. Research towards achieving the aim of producing bioengineered teeth – bioteeth – has largely focussed on the generation of immature teeth (teeth primordia) that mimic those in the embryo that can be transplanted as small cell 'pellets' into the adult jaw to develop into functional teeth.

Remarkably, despite the very different environments, embryonic teeth primordia can develop normally in the adult mouth and thus if suitable cells can be identified that can be combined in such a way to produce an immature tooth, there is a realistic prospect bioteeth can become a clinical reality. Subsequent studies have largely focussed on the use of embryonic cells and although it is clear that embryonic tooth primordia cells can readily form immature teeth following dissociation into single cell populations and subsequent recombination, such cell sources are impractical to use in a general therapy.

Professor Sharpe says: 'What is required is the identification of adult sources of human epithelial and mesenchymal cells that can be obtained in sufficient numbers to make biotooth formation a viable alternative to dental implants.'

In this new work, the researchers isolated adult human gum tissue from patients at the Dental Institute at King's College London, grew more of it in the lab, and then combined it with the cells of mice that form teeth. By transplanting this combination of cells into mice the researchers were able to grow hybrid human/mouse teeth containing dentine and enamel, as well as viable roots.

Professor Sharpe concludes: 'Epithelial cells derived from adult human gum tissue are capable of responding to tooth inducing signals from embryonic tooth mesenchyme in an appropriate way to contribute to tooth crown and root formation and give rise to relevant differentiated cell types, following in vitro culture.'

'These easily accessible epithelial cells are thus a realistic source for consideration in human biotooth formation. The next major challenge is to identify a way to culture adult human mesenchymal cells to be tooth-inducing, as at the moment we can only make embryonic mesenchymal cells do this.'

The research was funded by the UK National Institute for Health Research (NIHR) Biomedical Research Centre at Guy's and St Thomas' NHS Foundation Trust and King's College London, UK.

Adult Human Gingival Epithelial Cells as a Source for Whole-tooth Bioengineering *Journal of Dental Research* 2013

<http://www.sciencedaily.com/releases/2013/03/130308093933.htm>

### **Chewing Gum Helps You Concentrate for Longer, Study Suggests**

*Chewing gum can help you stay focused for longer on tasks that require continuous monitoring.*

This is the finding of new research by Kate Morgan and colleagues from Cardiff University due to be published in the British Journal of Psychology today, 8 March. Previous research has shown that chewing gum can improve concentration in visual memory tasks. This study focussed on the potential benefits of chewing gum during an audio memory task.

Kate Morgan, author of the study explained: "It's been well established by previous research that chewing gum can benefit some areas of cognition. In our study we focussed on an audio task that involved short-term memory recall to see if chewing gum would improve concentration; especially in the latter stages of the task." The study involved 38 participants being split in to two groups. Both groups completed a 30 minute audio task that involved listening to a list of numbers from 1-9 being read out in a random manner. Participants were scored on how accurately and quickly they were able to detect a sequence of odd-even-odd numbers, such as 7-2-1. Participants also completed questionnaires on their mood both before and after the task.

The results showed that participants who chewed gum had quicker reaction times and more accurate results than the participants who didn't chew gum. This was especially the case towards the end of the task.

Kate explained: "Interestingly participants who didn't chew gum performed slightly better at the beginning of the task but were overtaken by the end. This suggests that chewing gum helps us focus on tasks that require continuous monitoring over a longer amount of time."

*Kate Morgan, Andrew J. Johnson and Christopher Miles. Chewing gum moderates the vigilance decrement. British Journal of Psychology, 8 MAR 2013 DOI: 10.1111/bjop.12025*

<http://bit.ly/13PjGdK>

## Heavy drinkers get extra brain fuel from alcohol

### *Breakdown product boosts brain energy*

By Meghan Rosen

Alcohol may give heavy drinkers more than just a buzz. It can also fuel their brains, a new study suggests. Long-term booze use boosts brain levels of acetate, an energy-rich by-product of alcohol metabolism, researchers report online March 8 in the *Journal of Clinical Investigation*. In the study, people who downed at least eight drinks per week also sucked more energy from acetate than their light-drinking counterparts.

The extra energy may give heavy drinkers more incentive to imbibe, says study coauthor Graeme Mason of Yale University. And the caloric perk might help explain why alcohol withdrawal is so hard.

"I think it's a very good hypothesis," says biochemical geneticist Ting-Kai Li of Duke University. Scientists had suspected that heavy drinkers absorb and burn more acetate, but, he adds, "Graeme Mason showed that this is actually happening."

Acetate is best known as a chemical in vinegar. But when people drink a glass of wine or drain a can of beer, their liver breaks down the alcohol and pumps out acetate as leftovers. The bloodstream then delivers acetate throughout the body, including to the brain.

Human brains typically run on sugar. But with enough acetate in the blood, Mason thought, brains might crank up their ability to burn it too. To find out if his suspicion was correct, Mason and his colleagues peered into the brains of seven heavy drinkers and seven light drinkers, who quaffed fewer than two drinks per week.

The team injected sober volunteers with a form of acetate that was tagged with a traceable atom. Then the volunteers lay on their backs for two hours in a magnetic resonance imaging, or MRI, machine while Mason's group tracked the tagged acetate.

The scientists shot brief bursts of radio waves into the participants' brains that delivered tiny bits of energy to the tagged atoms and jostled out a return signal. The return signal varied slightly in frequency if the brain had burned acetate, so Mason's team could measure not only how much acetate was there but also how much fuel the participants were using. Heavy drinkers transported more acetate to their brains and burned the chemical about twice as fast as light drinkers, Mason's group found. Like a car that can switch to ethanol when it runs out of gasoline, heavy drinkers' brains could tap energy from an alternate fuel source.

When Mason first saw the results, he says, "I jumped out of my chair and threw my fist in the air." He had suspected that people with high blood acetate levels would be better at wringing energy from the chemical, but he says, "the effect was way bigger than I thought."

For many years, scientists thought that the brain could use only sugar as a source of energy, says Nora Volkow, director of the National Institute on Drug Abuse in Bethesda, Md. But that's changing, she says: Mason and colleagues' study contributes to the shifting view of the brain because "they showed a huge effect."

Next, Mason wants to figure out whether taking acetate would ease alcohol addicts' withdrawal symptoms. But he cautions, "I don't want people to start chugging vinegar." Because the liver is very good at turning alcohol into acetate, he says, people would have to ingest quarts of vinegar to get as much acetate as they do from drinking alcohol.

*L. Jiang et al. Increased brain uptake and oxidation of acetate in heavy drinkers. Journal of Clinical Investigation. doi: 10.1172/JCI65153.*

<http://phys.org/news/2013-03-3d-printer-bio-ink-human-video.html>

## Researchers developing 3D printer, 'bio-ink' to create human organs (w/ video)

*Experts agree that rising Chinese labor costs and improving U.S. technology will gradually cause significant manufacturing activity to return to the United States.*

When it does, a new interdisciplinary manufacturing venture called the Advanced Manufacturing Technology (AMTech) group at the University of Iowa College of Engineering's Center for Computer Aided Design (CCAD) will likely help lead the charge.

AMTech was formed to design, create, and test—both virtually and physically—a wide variety of electromechanical and biomedical components, systems and processes. Currently, the group is working on

projects ranging from printed circuit boards for automobiles and aircraft to replacement parts for damaged and failing human organs and tissue, says Tim Marler, AMTech co-director.

"Electromechanical systems are one of two current branches of the AMTechgroup," he says. "We want to simulate, analyze and test printed circuit boards and assemblies, because they are used in a wide range of products from missiles to power plants to cell phones.

"The second branch of the group involves biomanufacturing and is led by my colleague and AMTech co-director Ibrahim Ozbolat, assistant professor of mechanical and industrial engineering," says Marler. "The long-term goal of this branch is to create functioning human organs some five or 10 years from now. This is not far-fetched."

Using its facilities for engineering living tissue systems, the Biomanufacturing Laboratory at CCAD is working to develop and refine various 3D printing processes required for organ and tissue fabrication, Ozbolat says.

"One of the most promising research activities is bioprinting a glucose-sensitive pancreatic organ that can be grown in a lab and transplanted anywhere inside the body to regulate the glucose level of blood," says Ozbolat. He adds that the 3D printing, as well as virtual electronic manufacturing, being conducted at AMTech are done nowhere else in Iowa.

In fact, the multi-arm bio printer being used in the lab is unique. Ozbolat and Howard Chen, a UI doctoral student in industrial engineering, designed it and Chen built it. It turns out that managing multiple arms without having them collide with one another is difficult enough that other printers used in other parts of the world avoid the problem by using simpler designs calling for single-arm printing. As Chen continues to refine his and Ozbolat's design, the UI printer currently gives the UI researchers a distinct advantage.

While bioprinters at other institutions use one arm with multiple heads to print multiple materials one after the other, the UI device with multiple arms can print several materials concurrently. This capability offers a time-saving advantage when attempting to print a human organ because one arm can be used to create blood vessels while the other arm is creating tissue-specific cells in between the blood vessels.

The biomanufacturing group, which consists of researchers from various disciplines including industrial, mechanical, electrical, polymer and biomedical engineers as well as medical researchers, is working on this and other projects, and collaborates with Dr. Nicholas Zavazava, professor of internal medicine, in the UI Roy J. and Lucille A. Carver College of Medicine. The group also works with researchers from the college's Ignacio V. Ponsetti Biochemistry and Cell Biology Laboratory.

In addition to receiving support from the National Institutes of Health for the artificial pancreas research, AMTech is looking forward to continued support from the Electric Power Research Institute (EPRI) as well as seed funding from the UI for fostering commercialization of a new software product.

"When you look at the U.S. manufacturing environment and relevant technology, this is a perfect time to launch AMTech," says Marler, who also serves as associate research scientist at CCAD and senior research scientist at CCAD's renowned Virtual Soldier Research program.

AMTech co-directors Marler and Ozbolat are advised by Herm Reininga, interim director of the National Advanced Driving Simulator and member of the leadership council of the national Next Generation Manufacturing Technology Initiative. The AMTech group also includes one research staff member, one postdoctoral student, seven graduate students, and four undergraduate students.

Located within CCAD, AMTech conducts cutting-edge research and development aimed at advancing and exploring next generation manufacturing technologies.

<http://www.sciencedaily.com/releases/2013/03/130308183706.htm>

### **Scientists Identify Buphenyl as a Possible Drug for Alzheimer's Disease**

***Buphenyl, an FDA-approved medication for hyperammonemia, may protect memory and prevent the progression of Alzheimer's disease.***

Hyperammonemia is a life-threatening condition that can affect patients at any age. It is caused by abnormal, high levels of ammonia in the blood. Studies in mice with Alzheimer's disease (AD) have shown that sodium phenylbutyrate, known as Buphenyl, successfully increases factors for neuronal growth and protects learning and memory, according to neurological researchers at the Rush University Medical Center. Results from the National Institutes of Health funded study, recently were published in the Journal of Biological Chemistry.

"Understanding how the disease works is important to developing effective drugs that protect the brain and stop the progression of Alzheimer's disease," said Kalipada Pahan, PhD, the Floyd A. Davis professor of neurology at Rush and lead investigator of this study.

A family of proteins known as neurotrophic factors help in survival and function of neurons. Past research indicates that these proteins are drastically decreased in the brain of patients with Alzheimer's disease (AD).

"Neurotrophic factor proteins could be increased in the brain by direct injection or gene delivery," said Pahan. "However, using an oral medication to increase the level of these protein may be the best clinical option and a cost effective way to increase the level of these proteins directly in the brain."

"Our study found that after oral feeding, Buphenyl enters into the brain, increases these beneficial proteins in the brain, protects neurons, and improves memory and learning in mice with AD-like pathology," said Pahan. In the brain of a patient with AD, two abnormal structures called plaques and tangles are prime suspects in damaging and killing nerve cells. While neurons die, other brain cells like astroglia do not die.

The study findings indicate that Buphenyl increases neurotrophic factors from astroglia. Buphenyl stimulates memory-related protein CREB (cyclic AMP response element-binding protein) using another protein known as Protein Kinase C (PKC) and increases neurotrophic factors in the brain.

"Now we need to translate this finding to the clinic and test Buphenyl in Alzheimer's disease patients," said Pahan. "If these results are replicated in Alzheimer's disease patients, it would open up a promising avenue of treatment of this devastating neurodegenerative disease."

Other researchers involved in this study are Grant Corbett, neuroscience graduate student at Rush and Avik Roy, research assistant professor at Rush.

Alzheimer's disease is an irreversible, progressive brain disease that slowly destroys memory and thinking skills, and eventually even the ability to carry out the simplest tasks. In most people with Alzheimer's, symptoms first appear after age 60. Alzheimer's disease is the most common cause of dementia among older people.

Alzheimer's disease affects as many as 5.3 million Americans.

G. Corbett, A. Roy, K. Pahan. *Sodium phenylbutyrate enhances astrocytic neurotrophin synthesis via PKC-mediated activation of CREB: Implications for neurodegenerative disorders. Journal of Biological Chemistry, 2013; DOI:*

*10.1074/jbc.M112.426536*

[http://www.eurekalert.org/pub\\_releases/2013-03/mhif-bnr030813.php](http://www.eurekalert.org/pub_releases/2013-03/mhif-bnr030813.php)

## **Beware: Newly recognized heart cardiomyopathy is not always benign**

*Largely present in women, 'broken heart syndrome' is often triggered by stress*

Even though a newly recognized cardiomyopathy, which mainly impacts women, is typically treatable, Tako-tsubo cardiomyopathy can also be deadly when compounded by other co-morbidities, such as heart failure, according to a study being presented March 9 at the American College of Cardiology (ACC) Scientific Sessions. This condition, formally known as Tako-tsubo cardiomyopathy (TTC) and informally known as stress cardiomyopathy or broken heart syndrome, has abrupt onset of symptoms and is characterized by a distinctive left ventricular (LV) contraction profile. Ninety percent of the time, this condition affects women, who are usually middle aged and older, and the condition usually is triggered by a stressful event.

"Although TTC is typically reversible and considered to have favorable clinical outcomes, we have identified an important subset of patients, particularly those with severe heart failure and hypotension, who can have a substantial mortality risk," says the study's lead author Scott W. Sharkey, MD, a research cardiologist at the Minneapolis Heart Institute Foundation and a physician at the Minneapolis Heart Institute® at Abbott Northwestern Hospital in Minneapolis. "It's also important that physicians are aware this is not a rare a condition, as it is present in nearly 10 percent of women who present to the hospital with suspected heart attacks."

MHIF researchers reviewed 250 TTC patients who presented to the Minneapolis Heart Institute at Abbott Northwestern Hospital between 2001 and 2012. Then, they segregated those TTC patients presenting with particularly severe heart failure and very low pressure, or hypotension (systolic blood pressure < 100 mm Hg), who required supportive treatment. They found that severe hypotensive heart failure occurred in 45 patients. In this subset, 9 female patients died in-hospital despite aggressive treatment intervention, representing the only TTC-related hospital deaths in the 250 patient cohort.

Therefore, Sharkey and his colleagues concluded that TTC is not necessarily a benign condition. Severe hypotensive heart failure of severity necessitating vasopressor and/or intra-aortic balloon pump occurs in nearly 20 percent of patients. Also, all TTC-related hospital deaths occurred in the hypotensive heart failure subgroup with an overall mortality of 3.5 percent. Importantly, triggering physical stressors related to severe co-morbid non-cardiac conditions (8) or advanced age (1) were present in all 9 non-survivors, Sharkey notes.

"Unfortunately, there are not any guidelines or criteria to instruct diagnosis and treatment of these patients at this time," says Sharkey. "Therefore, this study could be a starting point for this process, as it provides a more complete profile of the clinical spectrum of TTC and provides useful guidance for the effective management of these acutely ill patients."

To raise additional awareness and improve care of these patients, he adds that guidelines would be helpful at this time, in order to standardize diagnosis and treatment across varied healthcare settings.

[http://www.eurekalert.org/pub\\_releases/2013-03/aafc-tob030513.php](http://www.eurekalert.org/pub_releases/2013-03/aafc-tob030513.php)

## **Trio of biomarkers may help identify kidney cancer in early stages**

*New immunoassay testing for the presence of three biomarkers appears to be a valid screening method for the early detection of malignant kidney cancer*

PHILADELPHIA — A new immunoassay that tests for the presence of three biomarkers appears to be a valid screening method for the early detection of malignant kidney cancer, according to data published in *Cancer Epidemiology, Biomarkers & Prevention*, a journal of the American Association for Cancer Research.

"Renal cell carcinoma, a malignant tumor arising from the kidney, is one of the most difficult forms of cancer to detect and treat properly because it remains silent until disseminating to other organs," said Nam Hoon Cho, M.D., of the Department of Pathology at Yonsei University Health System in Seoul, Korea. "Furthermore, because imaging, which is high-cost, is seldom performed without any specific reasons, developing a blood-tumor biomarker is a great chance to detect the silent killer."

The new immunoassay developed by Cho and colleagues from Genome Inc. measured the levels of three potential biomarkers for kidney cancer: nicotinamide N-methyltransferase (NNMT), L-plastin (LCP1) and nonmetastatic cells 1 protein (NM23A).

Using this assay, the researchers measured concentrations of NNMT, LCP1 and NM23A in 189 plasma samples from 102 healthy controls and patients with benign tumors and 87 patients with kidney cancer. Plasma levels indicated that all three biomarkers were highly elevated in patients with kidney cancer. For example, the median level of NNMT concentration in healthy controls was 68 pg/mL compared with 420 pg/mL for patients with kidney cancer.

Next, the researchers tested the ability of the immunoassay to distinguish plasma samples from healthy controls and patients with kidney cancer using the same 189 plasma samples already tested. The results indicated that the three-marker assay was highly accurate. When it correctly identified 90 percent of the samples from healthy controls, it also correctly identified 94.4 percent of the samples from patients with kidney cancer.

To validate the accuracy of the test, the researchers blind tested an additional 100 plasma samples from 73 healthy controls and 27 patients with kidney cancer. In this analysis, 67 of the samples from the 73 healthy controls and all of the samples from patients with kidney cancer were classified correctly.

"If this biomarker is truly valid and accurate to detect renal cell carcinoma, a number of patients with renal cell carcinoma could potentially be saved through early diagnosis," Cho said.

Cho and colleagues hope that this biomarker will soon be commercially available. They are currently working toward approval by the U.S. Food and Drug Administration.