

Blood type A may predispose to some rotavirus infections

Whether you become infected by some strains of rotavirus may depend on your blood type.

HOUSTON - Some strains of rotavirus find their way into the cells of the gastrointestinal tract by recognizing antigens associated with the type A blood group, a finding that represents a new paradigm in understanding how this gut pathogen infects humans, said Baylor College of Medicine researchers in an online report in the journal *Nature*.

Rotavirus is a major intestinal pathogen that is the leading cause of severe dehydration and diarrhea in infants around the world. An estimated 500,000 people worldwide die from the infection annually.

The structure of a key part of a strain of the virus known as P[14] provides a clue to how the virus infects human cells, said Dr. B. V. Venkataram Prasad, professor of biochemistry and molecular biology at BCM and the report's corresponding author.

In strains of rotavirus that infect animals, the top of a spike on the virus attaches to the cell via a glycan (one of many sugars linked together to form complex branched-chain structures) with a terminal molecule of sialic acid. The same did not appear to be true of virus strains that infect humans, and scientists believed the human rotavirus strains were bound to glycans with an internal sialic acid molecule, but they did not know how this occurs.

"We wondered how this genotype of rotavirus recognized a cellular glycan," said Prasad. "With colleagues at Emory (University School of Medicine), we did a glycan array analysis to see which glycans interacted with the top of the virus spike (called VP8*)."

The only type of glycan that interacted with VP8* was type A histo-blood group antigen, he said.

"That was surprising," he said. "We thought it had to be a glycan with sialic acid."

The histo-blood group antigen A does not have sialic acid.

However, when Dr. Liya Hu, a post-doctoral researcher in Prasad's laboratory, determined the structure of the VP8* domain, she found that the type A glycan bound to the rotavirus spike protein at the same place as the sialic acid would have in an animal rotavirus. Histo-blood group antigens are known to promote binding of norovirus and *Helicobacter pylori* cells to intestinal cells, but this had never been demonstrated in rotavirus.

Hu's structural study, using crystallography, showed subtle changes in the structure of the VP8* domain of the virus that allowed it to use the histo-blood group antigen A as a receptor.

In collaboration with the laboratory of Dr. Mary Estes, professor of molecular virology and microbiology at BCM, Prasad and his colleagues found that laboratory cells modified to express the histo-blood group antigen A were easily infected by this rotavirus strain. Cells that lacked this antigen were not easily infected.

An antibody to the histo-blood group antigen A blocked infection by the virus into human intestinal cells in culture.

"No one expected this," said Prasad. "Is there an emerging theme here with these intestinal pathogens? Do other viruses use these blood group antigens as a door to enter the cell?"

Further studies identified a second rotavirus strain P[9] that uses the histo-blood group antigen as a receptor, he said.

"The question now is do different strains use other histo-blood group antigens in this way?" he said.

Estes said, "These studies are significant because they provide a novel mechanism of transmission for a rotavirus strain that jumps from ungulates (such as horses, zebras, pigs, sheep) into humans."

The authors found humans infected with the P[14] strain had type A blood, but more studies are needed to confirm the connection.

Larger populations of infected individuals need to be studied to determine if there is a clear association of these virus strains using histo-blood group antigens as a receptor," they said.

This finding raises questions about why humans developed different blood groups, Prasad said. It may be an evolutionary change that occurred after the pathogen first invaded human cells.

Others who took part in this work include Sue E. Crawford, Rita Czako and Nicolas W Cortes-Penfield of BCM, David F. Smith of Emory University School of Medicine in Atlanta and Jacques Le Pendu of NSERM, Le Centre national de la recherche scientifique and Le Université de Nantes in France.

Funding for this work came from the National Institutes of Health and the Robert Welch Foundation.

Dr. Estes holds the Cullen Foundation Endowed Chair and director of the Texas Medical Center Digestive Diseases Center. Dr. Prasad holds the Alvin Romansky Chair in Biochemistry.

International team uncovers new genes that shape brain size, intelligence
UCLA-launched partnership identifies genes that boost or lessen risk of brain atrophy, mental illness and Alzheimer's disease

In the world's largest brain study to date, a team of more than 200 scientists from 100 institutions worldwide collaborated to map the human genes that boost or sabotage the brain's resistance to a variety of mental illnesses and Alzheimer's disease. Published April 15 in the advance online edition of Nature Genetics, the study also uncovers new genes that may explain individual differences in brain size and intelligence.

"We searched for two things in this study," said senior author Paul Thompson, professor of neurology at the David Geffen School of Medicine at UCLA and a member of the UCLA Laboratory of Neuro Imaging. "We hunted for genes that increase your risk for a single disease that your children can inherit. We also looked for factors that cause tissue atrophy and reduce brain size, which is a biological marker for hereditary disorders like schizophrenia, bipolar disorder, depression, Alzheimer's disease and dementia."

Three years ago, Thompson's lab partnered with geneticists Nick Martin and Margaret Wright at the Queensland Institute for Medical Research in Brisbane, Australia; and with geneticist Barbara Franke of Radboud University Nijmegen Medical Centre in the Netherlands. The four investigators recruited brain-imaging labs around the world to pool their brain scans and genomic data, and Project ENIGMA (Enhancing Neuro Imaging Genetics through Meta-Analysis) was born.

"Our individual centers couldn't review enough brain scans to obtain definitive results," said Thompson, who is also a professor of psychiatry at the Semel Institute for Neuroscience and Human Behavior at UCLA. "By sharing our data with Project ENIGMA, we created a sample large enough to reveal clear patterns in genetic variation and show how these changes physically alter the brain."

In the past, neuroscientists screened the genomes of people suffering from a specific brain disease and combed their DNA to uncover a common variant. In this study, Project ENIGMA researchers measured the size of the brain and its memory centers in thousands of MRI images from 21,151 healthy people while simultaneously screening their DNA. "Earlier studies have uncovered risk genes for common diseases, yet it's not always understood how these genes affect the brain," explained Thompson. "This led our team to screen brain scans worldwide for genes that directly harm or protect the brain."

In poring over the data, Project ENIGMA researchers explored whether any genetic variations correlated to brain size. In particular, the scientists looked for gene variants that deplete brain tissue beyond normal in a healthy person. The sheer scale of the project allowed the team to unearth new genetic variants in people who have bigger brains as well as differences in regions critical to learning and memory.

When the scientists zeroed in on the DNA of people whose images showed smaller brains, they found a consistent relationship between subtle shifts in the genetic code and diminished memory centers. Furthermore, the same genes affected the brain in the same ways in people across diverse populations from Australia, North America and Europe, suggesting new molecular targets for drug development.

"Millions of people carry variations in their DNA that help boost or lower their brains' susceptibility to a vast range of diseases," said Thompson. "Once we identify the gene, we can target it with a drug to reduce the risk of disease. People also can take preventive steps through exercise, diet and mental stimulation to erase the effects of a bad gene."

In an intriguing twist, Project ENIGMA investigators also discovered genes that explain individual differences in intelligence. They found that a variant in a gene called HMGA2 affected brain size as well as a person's intelligence. DNA is comprised of four bases: A, C, T and G. People whose HMGA2 gene held a letter "C" instead of "T" on that location of the gene possessed larger brains and scored more highly on standardized IQ tests.

"This is a really exciting discovery: that a single letter change leads to a bigger brain," said Thompson. "We found fairly unequivocal proof supporting a genetic link to brain function and intelligence. For the first time, we have watertight evidence of how these genes affect the brain. This supplies us with new leads on how to mediate their impact."

Because disorders like Alzheimer's, autism and schizophrenia disrupt the brain's circuitry, Project ENIGMA will next search for genes that influence how the brain is wired. Thompson and his colleagues will use diffusion imaging, a new type of brain scan that maps the communication pathways between cells in the living brain. *Project ENIGMA received funding from hundreds of federal and private agencies around the world. Thompson's UCLA coauthors included first author Jason Stein, Derrek Hibar, Rudy Senstad, Neda Jahanshad, Arthur Toga, Rita Cantor, Dr. Nelson Freimer, Roel Ophoff, Kristy Hwang, Dr. Liana Apostolova and Dr. Giovanni Coppola.*

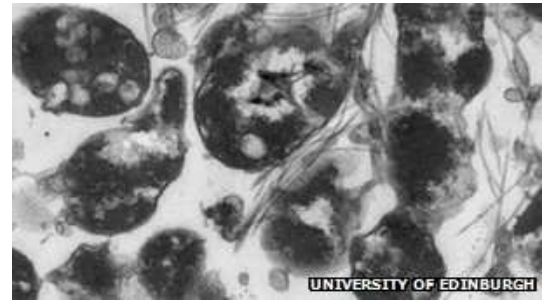
<http://www.bbc.co.uk/news/uk-scotland-17705984>

Asteroid craters could provide clue to life on Mars

The chances of finding life on Mars could be improved by looking in craters made by asteroids, according to a study.

Scientists at the University of Edinburgh said organisms had been discovered thriving deep underneath a site in the US where an asteroid crashed 35 million years ago. They believe such craters provide refuge for microbes. The findings suggest that crater sites on other planets may be "hiding life".

To find the microbes, researchers drilled almost 2km below one of the largest asteroid impact craters on Earth, in Chesapeake, US. Samples from below ground showed that microbes are unevenly spread throughout the rock, suggesting that the environment is continuing to settle 35 million years after impact.



The microbes were discovered by scientists at a US site where an asteroid crashed 35 million years ago

Researchers from the University of Edinburgh said heat from the impact of an asteroid collision would kill everything at the surface, but fractures to rocks deep below would allow water and nutrients to flow in and support life. They believe asteroid craters provide shelter to microbes, shielding them from the effects of changing seasons and events such as global warming or ice ages.

Prof Charles Cockell, from the University of Edinburgh, said: "The deeply fractured areas around the impact craters can provide a safe haven in which microbes can flourish for long periods of time. "Our findings suggest that the subsurface of craters on Mars might be a promising place to search for evidence of life."

http://www.eurekalert.org/pub_releases/2012-04/uoc-rro041612.php

Research reveals 1 of the earliest farming sites in Europe

University of Cincinnati research is revealing early farming in a former wetlands region that was largely cut off from Western researchers until recently.

The UC collaboration with the Southern Albania Neolithic Archaeological Project (SANAP) will be presented April 20 at the annual meeting of the Society for American Archaeology (SAA).

Susan Allen, a professor in the UC Department of Anthropology who co-directs SANAP, says she and co-director Ilirjan Gjipali of the Albanian Institute of Archaeology created the project in order to address a gap not only in Albanian archaeology, but in the archaeology in Eastern Europe as a whole, by focusing attention on the initial transition to farming in the region. Allen was awarded a \$191,806 (BCS- 0917960) grant from the National Science Foundation to launch the project in 2010.

"For Albania, there has been a significant gap in documenting the Early Neolithic (EN), the earliest phase of farming in the region," explains Allen. "While several EN sites were excavated in Albania in the '70s and '80s, plant and animal remains – the keys to exploring early farming – were not recovered from the sites, and sites were not dated with the use of radiocarbon techniques," Allen says.

"At that time (under communist leader Enver Hoxha), Albania was closed to outside collaborations and methodologies that were rapidly developing elsewhere in Europe, such as environmental archaeology and radiocarbon dating. The country began forming closer ties with the West following Hoxha's death in 1985 and the fall of communism in 1989, paving the way for international collaborations such as SANAP, which has pushed back the chronology of the Albanian Early Neolithic and helped to reveal how early farmers interacted with the landscape."

The findings show that Vashtëmi, located in southeastern Albania, was occupied around 6,500 cal BC, making it one of the earliest farming sites in Europe. The location of early sites such as Vashtëmi near wetland edges suggests that the earliest farmers in Europe preferentially selected such resource-rich settings to establish pioneer farming villages.

During this earliest phase of farming in Europe, farming was on a small scale and employed plant and animal domesticates from the Near East. At Vashtëmi, the researchers have found cereal-based agriculture including emmer, einkorn and barley; animals such as pigs, cattle and sheep or goats (the two are hard to tell apart for many bones of the skeleton); and deer, wild pig, rabbit, turtle, several species of fish and eels. What seems evident is that the earliest farmers in the region cast a wide net for food resources, rather than relying primarily on crops and domesticated animals, as is widely assumed.

Allen and Gjipali's research team included graduate and undergraduate students from UC's departments of anthropology and classics. SANAP is an international collaboration with researchers representing the U.S., Spain, France, Greece and Albania.

This planet obeys the law—stats on volcanic eruptions show pattern called Benford's Law

This planet obeys the law—stats on volcanic eruptions show pattern called Benford's Law

By Scott K. Johnson

Scientists delight in extracting order from chaos - finding patterns in the complexity of the real world that pull back the curtain and reveal how things work. Sometimes, though, those patterns create more head-scratching than excitement. Such is the case with Benford's law. One might expect a collection of real-world data - say, the half-lives of various isotopes, for example - to pretty much look like random numbers. And one might further expect the first (non-zero) digit of each of those numbers to also be random (i.e. just as many 2s as 9s).

Oddly, one would (in many cases) be wrong. It turns out that 1s are more likely than 2s, which are more likely than 3s, and so on. Not only that, the probabilities match a logarithmic distribution, just like the spacing on a logarithmic scale. The number 1 will be the first digit about 30 percent of the time, 2 will occur nearly 18 percent of the time, all the way on down to 9 showing up only about 5 percent of the time.

Law-abiding citizens everywhere will be happy to know our planet also obeys Benford's Law, with the duration and size of volcanic eruptions showing the same sort of pattern.

This strange phenomenon was first expressed in 1881 by an astronomer named Simon Newcomb. While using printed tables of logarithms, he noticed that the pages containing numbers that start with 1 were much more worn than the others. After thinking it out, he proposed that the occurrence of digits in the log tables in fact followed a logarithmic distribution themselves.

In 1938, the physicist Frank Benford rediscovered this idea, explored it more fully, and formalized the equation that describes it. He analyzed a number of data sets and showed that the relationship existed in the real world. It's obviously not universal—it won't be true of numbers in a telephone book, for example, which share assigned area codes and prefixes. Still, Benford's law has held good for a truly bewildering variety of data sets, including the surface area of rivers, the specific heat of chemical compounds, mathematical constants in physics, baseball stats, street addresses, populations of US counties, and a number of mathematical tables and series. (Try a few more for yourself.)

Perhaps most famously, Benford's law has been used to detect financial fraud. Folks who cook the books assume that random numbers will look inconspicuous, not realizing that's exactly what can make them look conspicuous. Dodgy rounding will also cause a data set to stick out like a sore thumb and get you caught red-handed. (You can hear about an example in this episode of WNYC's Radiolab.) It's often been suggested that Benford's law should be applied to the results of suspicious elections, but the relationship can be unreliable unless numbers span multiple orders of magnitude.

The burning question that can get some people downright irritated with the whole business of Benford's law is "why the hell should this be true?" No explanation is completely satisfying (unless you've got the fortitude for some mathematical heavy lifting), but a couple come close to de-spookifying the idea in at least some circumstances.

Think of a number starting with a 1. What would it take for it to start with a 2? Well, you'd have to double it. Now consider a number starting with a 9. An increase of only 10% will have it back to starting with a 1 again. And, of course, the process repeats—this number will have to be double again before it will start with a 2. For this reason, financial growth (such as investments) will follow Benford's law quite faithfully.

Several years ago, researchers in the Earth sciences began taking an interest in seeing whether our planet's behavior followed Benford's law. A paper published in 2010 applied the analysis to things like the length of time between geomagnetic reversals (when the Earth's magnetic "north" pole flips to the opposite geographical pole), the depth of earthquakes, greenhouse gas emissions by country, and even the numbers of infectious disease cases reported to the World Health Organization by each nation. All of them showed a decent fit to Benford's law. (As did some things out of this world, like the rotation frequencies of pulsars and masses of exoplanets.)

In a recent paper published in *Geology*, a pair of Spanish researchers extend this to three more data sets: the area and ages of volcanic calderas and the duration of volcanic eruptions between 1900 and 2009. This is more than just a bit of fun with numbers, as we're past the point where Benford's law needs confirmation. The goal is to use it as a sort of simple truth-check on databases of geologic data. If these things don't follow Benford's law, then it could be a sign that a data set is unrepresentative of reality or contains some sort of pervasive error or bias.

Benford's law fit the eruption duration data very well. The fit for the caldera areas was pretty good, too, though a few digits differed just enough that the authors suspect some excessive rounding may have taken place. The caldera eruption ages, however, showed a marked deviation from Benford's distribution. There were too many numbers starting with 2 and 3. When they looked closely, they saw this was due to a large number of North American calderas between 23 and 42 million years old.

As it turns out, this is a well-known anomaly. It's not clear whether there was really an unusual cluster of calderas at that time or this is simply a case of one area being studied more intensely. Regardless, removing those calderas from the analysis returned the data set to harmony with Benford's law. In essence, Benford's law provided another way to show that those calderas are anomalous.

Because researchers often want to know whether the data they're analyzing is a representative sample of the world at large, any technique that could help them do so is likely to get a serious look. The authors conclude, "Since the use of Benford's law may serve as a simple and quick quality test of data, and provide new ways to detect anomalous signals in data sets, it could be used as a validity check on future databases related to volcanoes." In other words, before you go searching for patterns in a database, it might be prudent to make sure the database conforms to Benford's pattern. *Geology*, 2012. DOI: 10.1130/G32787.1 (About DOIs).

<http://phys.org/news/2012-04-results-cattle-lyme-disease.html>

Surprising study results: More cattle means less Lyme disease

The abundance of cattle is the primary influence on the prevalence of two tick-borne pathogens, according to a paper in the April Applied and Environmental Microbiology.

Phys.org - One of these, *Anaplasma phagocytophilum*, causes human granulocytic anaplasmosis, and the other, *Borrelia burgdorferi*, causes Lyme disease. Although other studies have examined the effect of hosts on tick and tick-borne pathogen dynamics, this is the first to clarify the role of host abundance on prevalence of the two pathogens in their natural habitat, where wildlife and domestic livestock coexist.

The impetus for the research was the fact that in recent decades, gamekeepers in the study area, a wildlife preserve in the northern Iberian peninsula, had suffered Lyme disease, and had noticed an increasing abundance of ticks, says first author Francisco Ruiz-Fons, of the Instituto de Investigación en Recursos Cinegéticos, Ciudad Real, Spain. "Our working hypothesis was that wild and domestic ungulates would be primary drivers of the abundance of *Ixodes ricinus* ticks, and of the prevalence of pathogens transmitted by this tick species, in natural foci where they coexist and where *B. burgdorferi* and *A. phagocytophilum* are endemic."

That hypothesis held up, but somewhat differently from expected, having opposite effects on *A. phagocytophilum* and Lyme bacterium, *B. burgdorferi*, in reducing prevalence of the latter. That seemingly counterintuitive finding stemmed from the fact that "cattle and wild ungulates may act as diluters of borrelias by diverting infected ticks' bites from competent reservoirs such as birds or small mammals," says Ruiz-Fons. Thus, more cattle meant reduced numbers of Lyme disease bacteria.

"The most important application of our findings is that if we want to reduce the risk of animals and humans becoming infected by [tick-borne] pathogens we should control the infestation by ticks in cattle—and perhaps in wildlife—rather than by reducing cattle or wild host abundance in areas where wild and domestic animals coexist," says Ruiz-Fons.

More information: F. Ruiz-Fons, I.G. Fernandez-de-Mera, P. Acevedo, C. Gortazar, and J. de la Fuente, 2012. Factors driving the abundance of Ixodes ricinus ticks and the prevalence of zoonotic I. ricinus-borne pathogens in natural foci. Appl. Environ. Microbiol. 78:2669-2676 Provided by American Society for Microbiology

<http://www.scientificamerican.com/article.cfm?id=how-colors-get-their-name>

Hierarchy of Color Naming Matches the Limits of Our Vision System

The time needed for us to reach consensus on a color name falls into a hierarchy that matches the human vision system's sensitivity to red over blue, and so on

By Charles Q. Choi and LiveScience | Monday, April 16, 2012 | 2

The order in which colors are named worldwide appears to be due to how eyes work, suggest computer simulations with virtual people. These findings suggest that wavelengths of color that are easier to see also get names earlier in the evolution of a culture.

A common question in philosophy is whether or not we all see the world the same way. One strategy that scientists have for investigating that question is to see what colors get names in different cultures. Intriguingly, past research has found that colors familiar to one culture might not have names in another, suggesting different cultures indeed have distinct ways of understanding the world.

One mystery scientists have uncovered is that color names always seem to appear in a specific order of importance across cultures—black, white, red, green, yellow and blue.

"For example, if a population has a name for red, it also has a name for black and for white; or, if it has a name for green, it also has a name for red," said researcher Francesca Tria, a physicist at the ISI Foundation in Turin, Italy. But if a population has a name for black and white, that doesn't necessarily mean they have a name for red.

To solve the puzzle of this color-name hierarchy, Tria and her colleagues devised a computer simulation with pairs of virtual people, or "agents," who lacked the knowledge of names for colors. One agent, the speaker, is shown two or more objects, invents a name for a color to describe one of the objects, and refers to the item by that color. The other agent, the hearer, then has to guess which item, and thus color, the speaker referred to. Scientists repeated this until all the agents came to a consensus on color names.

A key feature of this simulation was its adherence to the limits of human vision. Our eyes are more sensitive to some wavelengths of light, or colors, than others. The agents in the simulation were not required to distinguish between hues that a human eye could not tell apart. "Roughly speaking, human eyes can tell apart two colors only if their wavelengths differ at least by a certain amount - the 'just noticeable difference,'" Tria said. The researchers found the time agents needed to reach consensus on a color name fell into a distinct hierarchy - red, magenta-red, violet, green-yellow, blue, orange and cyan, in that order. This hierarchy approximately matches the color name order seen in real cultures. This hierarchy of colors also matches the limits of human vision, with the human eye being more sensitive to red wavelengths than those for blue, and so on.

"Our approach suggests a possible route to the emergence of hierarchical color categories," Tria told LiveScience. "Humans tend to react most saliently to certain parts of the spectrum, often selecting exemplars for them, and finally comes the process of linguistic color naming, which adheres to universal patterns resulting in a neat hierarchy."

Tria and her colleagues Vittorio Loreto and Animesh Mukherjee detailed their findings online April 16 in the Proceedings of the National Academy of Sciences.

http://www.eurekalert.org/pub_releases/2012-04/lhri-acc041712.php

A common cholesterol medication may impact kidney health New study by Lawson Research cautions further research is needed

LONDON, ON - Older patients taking a common cholesterol medication should be cautious of the impact on their kidney health. In a new study by Dr. Amit Garg, Scientist at the Lawson Health Research Institute and the Institute for Clinical Evaluative Sciences (ICES), and colleagues, one in 10 new older fibrate users experienced a 50 per cent increase in their serum creatinine.

Fibrates are a group of medications commonly used to treat high cholesterol. Recent evidence from clinical trials and case reports suggests fibrates can cause an increase to serum creatinine, an indicator of kidney health measured by a blood test, which indicates a loss of kidney health. After a number of similar experiences in their renal clinics, Dr. Garg and his colleagues felt these events merited further examination.

In a large, "real practice" study, the team examined more than 20,000 older Ontario residents with new prescriptions for fibrates. Throughout the first 90 days of their prescription, they monitored the renal outcomes in this population, and compared them to patients taking ezetimide, another cholesterol agent not known to have any renal effects.

Results show new fibrate users were more likely to experience an increase in serum creatinine; one in 10 users experienced a 50 per cent increase in the first 90 days of their prescription. As a result, these users were also more likely to consult a kidney specialist or to be hospitalized during this time.

The exact mechanism by which fibrates influence kidney function remains unclear and requires further research. This study proves that fibrates have important acute effects on kidney function and/or its measurement, to a greater extent than described in existing clinical trials data.

"At the end of the day, we want to prescribe medication with the highest benefit and the least amount of adverse events," Dr. Garg says. "When a physician decides to start a fibrate in a new patient, especially an older patient, given the information we have today they should start the patient on a dose that's appropriate, closely monitor their kidney function, and, if the kidney function goes off, either lower the dose or discontinue the drug."

The full study is published in the Annals of Internal Medicine, [available here](#)

Dr. Garg is also a Nephrologist and Director of the Kidney Clinical Research Unit at London Health Sciences Centre. He is also a Professor in the Departments of Medicine and Epidemiology and Biostatistics at Western University's Schulich School of Medicine & Dentistry.

To view the appointments and affiliations of the full study team, click here: <http://www.annals.org/content/156/8/560.abstract>

Some stars capture rogue planets

New research suggests that billions of stars in our galaxy have captured rogue planets that once roamed interstellar space.

The nomad worlds, which were kicked out of the star systems in which they formed, occasionally find a new home with a different sun. This finding could explain the existence of some planets that orbit surprisingly far from their stars, and even the existence of a double-planet system. "Stars trade planets just like baseball teams trade players," said Hagai Perets of the Harvard-Smithsonian Center for Astrophysics. The study, co-authored by Perets and Thijs Kouwenhoven of Peking University, China, will appear in the April 20th issue of *The Astrophysical Journal*.

To reach their conclusion, Perets and Kouwenhoven simulated young star clusters containing free-floating planets. They found that if the number of rogue planets equaled the number of stars, then 3 to 6 percent of the stars would grab a planet over time. The more massive a star, the more likely it is to snag a planet drifting by.

They studied young star clusters because capture is more likely when stars and free-floating planets are crowded together in a small space. Over time, the clusters disperse due to close interactions between their stars, so any planet-star encounters have to happen early in the cluster's history.

Rogue planets are a natural consequence of star formation. Newborn star systems often contain multiple planets. If two planets interact, one can be ejected and become an interstellar traveler. If it later encounters a different star moving in the same direction at the same speed, it can hitch a ride.

A captured planet tends to end up hundreds or thousands of times farther from its star than Earth is from the Sun. It's also likely to have an orbit that's tilted relative to any native planets, and may even revolve around its star backward. Astronomers haven't detected any clear-cut cases of captured planets yet. Imposters can be difficult to rule out. Gravitational interactions within a planetary system can throw a planet into a wide, tilted orbit that mimics the signature of a captured world.

Finding a planet in a distant orbit around a low-mass star would be a good sign of capture, because the star's disk wouldn't have had enough material to form the planet so far out.

The best evidence to date in support of planetary capture comes from the European Southern Observatory, which announced in 2006 the discovery of two planets (weighing 14 and 7 times Jupiter) orbiting each other without a star. "The rogue double-planet system is the closest thing we have to a 'smoking gun' right now," said Perets. "To get more proof, we'll have to build up statistics by studying a lot of planetary systems."

Could our solar system harbor an alien world far beyond Pluto? Astronomers have looked, and haven't found anything yet. "There's no evidence that the Sun captured a planet," said Perets. "We can rule out large planets. But there's a non-zero chance that a small world might lurk on the fringes of our solar system."

http://www.eurekalert.org/pub_releases/2012-04/uomm-sht041512.php

Study: Helicopter transport improves trauma patient survival compared to ground transport

University of Maryland researcher leads sophisticated analysis of national trauma data

Baltimore, MD If you are severely injured, a helicopter flight to a top-level trauma center will boost your chance of survival over ground transport. That's the conclusion of a rigorous, national comparison of the effectiveness of helicopter versus ground emergency medical services, published in the April 18, 2012, issue of the *Journal of the American Medical Association*.

Survival after trauma has increased in recent years with improvements in emergency medical services coupled with the rapid transportation of trauma patients to centers capable of providing the most advanced care. What has not been clear until this study, is the effectiveness of helicopter emergency medical services (HEMS), a limited and expensive resource, compared to its alternative, ground emergency medical services (GEMS).

"We looked at the sickest patients with the most severe injuries and applied sophisticated statistical analyses to the largest aggregation of trauma data in the world," says the study's principal investigator, Samuel M. Galvagno Jr., D.O., Ph.D., assistant professor, Department of Anesthesiology, Divisions of Trauma Anesthesiology and Critical Care Medicine, University of Maryland School of Medicine. "We were careful at every step to balance all the potential other factors that could explain any benefit of the helicopter. After all that, the survival advantage of helicopters remained," says Galvagno.

Dr. Galvagno is on the staff of the R Adams Cowley Shock Trauma Center at the University of Maryland Medical Center, where many of the life-saving practices in modern trauma medicine were pioneered. The Shock Trauma Center was the first fully integrated trauma center in the world, and remains the epicenter for trauma research and training both nationally and internationally today.

"The use of helicopter emergency medical services in the United States has been a controversial subject over the last decade or so, centering on the costs and the potential for crashes, says Thomas M. Scalea, M.D., the Francis X. Kelly Professor of Trauma in the Department of Surgery; director of the Program in Trauma, University of Maryland School of Medicine; and physician-in-chief at the R Adams Cowley Shock Trauma Center. "Previous studies have found a survival benefit by using helicopters, but the studies were small and left some doubt. This study in JAMA is very robust," says Dr. Scalea.

"Dr. Galvagno's research demonstrates how statistics and technology can be used to help researchers mine huge databases for useful information to help determine best care for patients and appropriate utilization of limited health care funds," says E. Albert Reece, M.D., Ph.D., M.B.A., vice president for medical affairs, University of Maryland; the John Z. and Akiko K. Bowers Distinguished Professor; and dean, University of Maryland School of Medicine. "Advances in the emerging science of comparative effectiveness, coupled with the expertise of physician researchers who have direct patient care experience, will deliver data that clinicians, policy-makers and ultimately the public can use to make informed decisions."

For this study, Dr. Galvagno developed the most rigorous comparison of helicopter and ground transport to date. He and his team tracked patients meeting certain criteria who were listed in the 2007-2009 version of the American College of Surgeons (ACS) National Trauma Data Bank (NTDB). The NTDB contains more than 1.8 million patient records from more than 900 centers in the United States.

To meet the study's criteria, patients had to be adults (over age 15) who sustained blunt or penetrating trauma with an injury severity score greater than 15 (critically injured). They had to be taken to either an ACS Level I or Level II trauma center in the United States (centers that meet certain high standards of care, with Level I the highest). The main outcome measure was survival to discharge from the hospital.

"Dr. Galvagno is an anesthesiologist whose specialties include intraoperative care of people who have suffered trauma and the care of critically ill patients. He not only treats patients but also has major research interests in the improvement of patient safety and care," says Peter Rock, M.D., M.B.A., the Dr. Martin A. Helrich Professor and chair of the Department of Anesthesiology at the School of Medicine. "His experience as a former paramedic and now in the Air Force Reserve where he assists in the evacuation of wounded warriors has taught him that saving lives is a combination of quality care both in the field and on the way to a trauma center, coupled with rapid transport and expertise at the hospital. All of those factors come together in this study."

A total of 223,475 adult patients met the criteria: ground transport accounted for 161,566, while 61,909 patients were flown in helicopters. Overall, 7,813 (12.6 percent) HEMS patients died compared to 17,775 (11 percent) transported by GEMS. This raw data shows a significant difference, but Dr. Galvagno says the odds for helicopter transport improved when statistical models were applied to the numbers to factor in so-called "confounding" factors that could lead to a misinterpretation of the data. These include injury severity, age, vital signs, type of injury, gender and trauma center. The researchers also developed statistical models to account for missing data in the NTDB, including travel time and distance to trauma centers, key bits of information that could impact survival.

Dr. Galvagno and his team conclude that the helicopter is associated with a 16 percent increased rate of survival for the 156,511 patients transported to Level I trauma centers. That percentage means 65 patients must be transported to save one life. The 64,964 patients who went by chopper to Level II trauma centers had a 15 percent survival advantage, meaning 69 must be transported to save one life.

Dr. Galvagno says the study raises many questions that need to be explored further. "The benefits of the helicopter, we believe, are related to multiple factors. Certainly time and crew expertise play a role. Beyond that, we're not sure. More study is warranted," says Dr. Galvagno. Trauma is the leading cause of death and disability among young people around the world, according to the researchers. In the United States, more than 50 million people are injured each year, resulting in approximately 169,000 deaths annually and a lifetime cost of \$406 billion.

http://www.eurekalert.org/pub_releases/2012-04/m-lrf041712.php

Licorice root found to contain anti-diabetic substance

Researchers discover promising anti-diabetic substance in the amorfrutin class of natural substances

It provides the raw material for liquorice candy, calms the stomach and alleviates diseases of the airways: liquorice root. Chosen as the "Medicinal plant 2012", the root has been treasured in traditional healing since ancient times. Researchers at the Max Planck Institute for Molecular Genetics in Berlin have now discovered that liquorice root also contains substances with an anti-diabetic effect. These amorfrutins not only reduce

blood sugar, they are also anti-inflammatory and are very well tolerated. Thus, they may be suitable for use in the treatment of complex metabolic disorders.

Natural substances have a surprising and often largely unexploited potential in the prevention and treatment of common diseases. For example, liquorice root *Glycyrrhiza* contains different substances that help to alleviate disorders of the airways and digestive system. It has been used for millennia in traditional healing and is mainly administered in the form of tea. A team of researchers working with Sascha Sauer from the Max Planck Institute for Molecular Genetics in Berlin has now discovered that the plant from the papilionaceae or leguminous family might also be effective in the treatment of adult (type 2) diabetes. The scientists identified a group of natural substances with an anti-diabetic effect, the amorfrutins, in the plant's edible root.

The substances, which have a simple chemical structure, are not only found in liquorice root, but are also in the fruit of the *Amorpha fruticosa* bush. The new anti-diabetic agents were named after this plant, which is native to the US, Canada and Mexico. As the researchers demonstrated using diabetic mice, the amorfrutins not only have characteristics that reduce blood sugar, they are also anti-inflammatory in their effect. Moreover, they also prevent fatty liver – a common disease caused by excessively fat-rich nutrition.

"The health-beneficial effects are based on the fact that the amorfrutin molecules dock directly onto a receptor in the nucleus called PPAR γ ," explains Sascha Sauer. PPAR γ plays an important role in the cell's fat and glucose metabolism. The binding of the amorfrutin molecules activates various genes that reduce the plasma concentration of certain fatty acids and glucose. The reduced glucose level prevents the development of insulin resistance – the main cause of adult diabetes.

"Although there are already drugs on the market that affect the PPAR γ receptor, they are not selective enough in their effect and cause side effects like weight gain and cardio-vascular problems," says Sascha Sauer. In contrast, as demonstrated by the studies carried out to date, the amorfrutins are very well tolerated. "However, drinking liquorice tea or eating liquorice will not help to treat diabetes," explains the scientist. "The concentration of the substances in the tea and liquorice is far too low to be effective." The researchers therefore developed special extraction processes to obtain the amorfrutins from the plant in sufficient concentrations. This could be used to produce amorfrutin extracts on an industrial scale.

The newly discovered active substances not only seem to hold great promise for the treatment of complex metabolic disorders, they may also be suitable for prophylactic use. "The amorfrutins can be used as functional nutritional supplements or as mild remedies that are individually tailored to the patient," says Sascha Sauer. "In view of the rapid spread of metabolic diseases like diabetes, it is intended to develop these substances further so that they can be used on humans in the future." To do this, the researchers must now test the effect of the substances and the plant amorfrutin extracts in clinical studies on diabetes patients.

http://www.eurekalert.org/pub_releases/2012-04/bumc-mac041712.php

Moderate alcohol consumption before and after heart attack associated with lower mortality

The Health Professionals Follow-up Study (HPFS) is a prospective cohort study of 51,529 US male health professionals.

During the follow up of these men between 1986 to 2006, published in the European Heart Journal, 1,818 men were confirmed with incident non-fatal myocardial infarction (MI) – a non fatal heart attack. Among heart attack survivors, 468 deaths were documented during up to 20 years of follow up. Repeated reports were obtained on alcohol consumption every four years. Average alcohol consumption was calculated prior to and then following the MI.

The overall results show that, in comparison with no alcohol consumption, the pre-MI and the post-MI intakes of light (0.1-9.9 g/day of alcohol, or up to one small typical drink) and moderate (10.0-29.9 g/d, or up to about 2 ½ to 3 drinks) amounts of alcohol were both associated with lower risk of all-cause mortality and cardiovascular mortality among these men.

The significant reductions in all-cause mortality risk (22% lower for 0.1-9.9 g/day and 34% lower for 10.0 – 29.9 g/day, in comparison with non-drinkers) were no longer present for those who drank more than 30 g/day; for this highest consumer group, the adjusted hazard ratio was 0.87 with 95% CI of 0.61-1.25.

There are a number of other informative and interesting results described from this study. First, there was little change in reported alcohol intake prior to and following the MI: drinkers tended to remain drinkers of similar amounts. Few non-drinkers began to drink after their heart attack; among heavier drinkers, there was a tendency to reduce drinking (but very few stopped drinking completely). Further there were no significant differences in outcome according to type of beverage consumed although, interestingly, lower hazard ratios were seen for consumers of beer and liquor than of wine. While the authors state that the effects of alcohol were

stronger for the association with non-anterior MI's, the relative risk (versus non-drinkers) for all-cause mortality were little different: among the moderately drinking men the relative risks were 0.58 for anterior MI and 0.51 for other types of MI.

Even though exposures (such as alcohol) for cardiovascular events (such as MI) may be different after a person has an event than it was before the event, in this study the reductions in risk were almost the same. For example, both for alcohol intake reported prior to a MI, and that after a non-fatal MI, the risk of mortality was about 30% lower for moderate drinkers than it was for abstainers. This suggests that, in terms of reducing cardiovascular disease, alcohol may have relatively short-term effects, suggesting that frequent but moderate consumption (of under 30g a day for men) may result in the best health outcomes.

Reference: Pai JK, Mukamal KJ, Rimm EB. Long-term alcohol consumption in relation to all-cause and cardiovascular mortality among survivors of myocardial infarction: the Health Professionals Follow-up Study. European Heart Journal 2012; doi:10.1093/eurheartj/ehs047

http://www.eurekalert.org/pub_releases/2012-04/uota-nrc041812.php

New research could mean cellphones that can see through walls

Team finds new possibilities in untapped terahertz range with implications for a host of devices

Comic book hero superpowers may be one step closer to reality after the latest technological feats made by researchers at UT Dallas. They have designed an imager chip that could turn mobile phones into devices that can see through walls, wood, plastics, paper and other objects.

The team's research linked two scientific advances. One involves tapping into an unused range in the electromagnetic spectrum. The other is a new microchip technology.

The electromagnetic spectrum characterizes wavelengths of energy. For example, radio waves for AM and FM signals, or microwaves used for cell phones or the infrared wavelength that makes night vision devices possible.

But the terahertz band of the electromagnetic spectrum, one of the wavelength ranges that falls between microwave and infrared, has not been accessible for most consumer devices.

"We've created approaches that open a previously untapped portion of the electromagnetic spectrum for consumer use and life-saving medical applications," said Dr. Kenneth O, professor of electrical engineering at UT Dallas and director of the Texas Analog Center of Excellence (TxACE). "The terahertz range is full of unlimited potential that could benefit us all."

Using the new approach, images can be created with signals operating in the terahertz (THz) range without having to use several lenses inside a device. This could reduce overall size and cost.

The second advance that makes the findings applicable for consumer devices is the technology used to create the microchip. Chips manufactured using CMOS (Complementary Metal-Oxide Semiconductor) technology form the basis of many consumer electronic devices used in daily life such as personal computers, smart phones, high definition TV and game consoles.

"CMOS is affordable and can be used to make lots of chips," Dr. O said. "The combination of CMOS and terahertz means you could put this chip and receiver on the back of a cellphone, turning it into a device carried in your pocket that can see through objects."

Due to privacy concerns, Dr. O and his team are focused on uses in the distance range of less than four inches.

Consumer applications of such technology could range from finding studs in walls to authentication of important documents. Businesses could use it to detect counterfeit money. Manufacturing companies could apply it to process control. There are also more communication channels available in terahertz than the range currently used for wireless communication, so information could be more rapidly shared at this frequency.

Terahertz can also be used for imaging to detect cancer tumors, diagnosing disease through breath analysis, and monitoring air toxicity.

"There are all kinds of things you could be able to do that we just haven't yet thought about," said Dr. O, holder of the Texas Instruments Distinguished Chair.

The research was presented at the most recent International Solid-State Circuits Conference (ISSCC). The team will work next to build an entire working imaging system based on the CMOS terahertz system.

Other authors of the paper include Ruonan Han and Yaming Zhang, former students of Professor O, Yongwan Kim and Dae Yeon Kim, TxACE members, and Hisashi Sam Shichijio, research professor at TxACE.

The work was supported by the Center for Circuit & System Solutions (C2S2 Center) and conducted in the TxACE laboratory at UT Dallas, which is funded by the Semiconductor Research Corporation (SRC), the state through its Texas Emerging Technology Fund, Texas Instruments Inc., The UT System and UT Dallas.

Kidney stone mystery solved

Kidney stones strike an estimated 1 million Americans each year, and those who have experienced the excruciating pain say it is among the worst known to man (or woman).

Now, new research by scientists at Washington University School of Medicine in St. Louis provides evidence to explain why some people are more prone to develop the condition than others. Their discovery opens the door to finding effective drug treatments and a test that could assess a person's risk of kidney stones.

"Now, we finally have a more complete picture detailing why some people develop kidney stones and others do not," says senior author Jianghui Hou, PhD, assistant professor of medicine. "With this information, we can begin to think about better treatments and ways to determine a person's risk of the condition, which typically increases with age." The research, in mice, is now available online in the EMBO Journal, published by the European Molecular Biology Organization.

Because kidneys function the same way in mice as in humans, the new findings can help scientists understand the root causes of kidney stones in patients. The mouse model used in the study can also serve as a platform for the preclinical testing of novel treatments for the condition, the researchers say. Most kidney stones form when the urine becomes too concentrated, allowing minerals like calcium to crystallize and stick together. Diet plays a role in the condition - not drinking enough water or eating too much salt (which binds to calcium) also increases the risk of stones. But genes are partly to blame. A common genetic variation in a gene called claudin-14 recently has been linked to a substantial increase in risk - roughly 65 percent - of getting kidney stones. In the new study, the researchers have shown how alterations in the gene's activity influence the development of stones.

Typically, the claudin-14 gene is not active in the kidney. The new research shows that its expression is dampened by two snippets of RNA, a sister molecule of DNA, that essentially silence the gene. When claudin-14 is idled, the kidney's filtering system works like it's supposed to. Essential minerals in the blood like calcium and magnesium pass through the kidneys and are reabsorbed back into the blood, where they are transported to cells to carry out basic functions of life. But when people eat a diet high in calcium or salt and don't drink enough water, the small RNA molecules release their hold on claudin 14. An increase in the gene's activity prevents calcium from re-entering the blood, the study shows.

Hou and his team have found that claudin-14 blocks calcium from entering passageways called tight junctions in cells that line the kidney and separate blood from urine. Without a way back to the bloodstream, excess calcium goes into the urine. Too much calcium in the urine can lead to stones in the kidneys or bladder. Intense pain develops when a large stone gets stuck in the bladder, ureter or urethra and blocks the flow of urine.

Hou's research supports the theory that people with a common variation in claudin-14 lose the ability to regulate the gene's activity, increasing the risk of kidney stones. He is optimistic, however, that drugs could be developed to target the short stretches of RNA that are intimately linked to claudin 14. Drugs that mimic these so-called microRNAs could keep the activity of claudin-14 in check and reduce the likelihood that stones would form. Also, it may one day be possible to develop a diagnostic test to measure levels of the claudin-14 protein excreted in urine. Elevated levels would indicate an increased risk of stones, and people could take steps to prevent stones by modifying their diet. "Many genes likely play a role in the formation of kidney stones," Hou says. "But this study gives us a better idea of the way one of the major players work."

Now that we understand the physiology of the condition, we can start to think about better treatments or even ways to prevent stones from developing in the first place." Hou is working with Washington University's Office of Technology Management on an invention related to work described in the paper.

The research was funded, in part, by the National Institutes of Health (NIH) and the American Heart Association.

Gong Y, Renigunta V, Himmerkus N, Zhang J, Renigunta A, Bleich M, Hou J. Claudin-14 regulates renal Ca^{++} transport in response to CaSR signaling via a novel microRNA pathway. The EMBO Journal. Advance online publication Feb. 28, 2012.

http://www.eurekalert.org/pub_releases/2012-04/plos-mel041612.php

Meat eating led to earlier weaning, helped humans spread across globe

When early humans became carnivores, their higher-quality diet allowed mothers to wean babies earlier and have more children, with potentially profound effects on population dynamics and the course of human evolution, according to a study published Apr. 18 in the open access journal PLoS ONE.

In a comparison of 67 mammalian species, including humans, apes, mice, and killer whales, among many others, researchers from Lund University in Sweden found a clear correlation between carnivory and earlier weaning.

"Eating meat enabled the breast-feeding periods and thereby the time between births to be shortened," said Elia Psouni, lead author of the study. "This must have had a crucial impact on human evolution."

Among natural fertility societies, the average duration of breast-feeding is 2 years and 4 months. This is not much in relation to the maximum lifespan of our species, around 120 years. It is even less if compared to our closest relatives: female chimpanzees suckle their young for 4 to 5 years, whereas the maximum lifespan for chimpanzees is only 60 years.

Many researchers have tried to explain the relatively shorter breast-feeding period of humans based on social and behavioral theories of parenting and family size. But the Lund group has now shown that the young of all species stop suckling when their brains have reached a particular developmental stage. The difference is that carnivores – categorized as species for which at least 20 per cent of the energy content of their diet comes from meat – reach this point earlier than herbivores or omnivores due to their higher quality diet. Therefore, the different weaning times for humans and the great apes seems to result simply from the fact that, as a species, humans are carnivores, whereas gorillas, orangutans and chimpanzees are herbivores or omnivores.

"That humans seem to be so similar to other animals can of course be taken as provocative," Psouni says. "We like to think that culture makes us different as a species. But when it comes to breast-feeding and weaning, no social or cultural explanations are needed; for our species as a whole it is a question of simple biology."

She is careful to emphasize that their results provide insight into how carnivory may have contributed to early humans spreading on Earth, and says nothing about what humans today should or should not eat.

Citation: Psouni E, Janke A, Garwicz M (2012) Impact of Carnivory on Human Development and Evolution Revealed by a New Unifying Model of Weaning in Mammals. *PLoS ONE* 7(4): e32452. doi:10.1371/journal.pone.0032452

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http://www.eurekalert.org/pub_releases/2012-04/jhmi-nmo041612.php

New medication offers hope to patients with frequent, uncontrollable seizures

A new type of anti-epilepsy medication that selectively targets proteins in the brain that control excitability may significantly reduce seizure frequency in people whose recurrent seizures have been resistant to even the latest medications, new Johns Hopkins-led research suggests.

"Many other drugs to treat frequent seizures have been released in the last 10 years and for many people, they just don't work," says study leader Gregory L. Krauss, M.D., a professor of neurology at the Johns Hopkins University School of Medicine. "For a drug-resistant population that has run out of options, this study is good news. These are patients who are tough to treat and are fairly desperate."

Perampanel is the first in a new class of drugs that appears to blunt an excitatory response in the brain by inhibiting a specific form of glutamate receptor called an AMPA receptor and therefore reducing seizures without causing major side effects. Other drugs targeting all three forms of glutamate receptors in the brain have tended to make patients too sleepy to function, even putting them in comas, Krauss says. But this new medication, he says, may potentially offer relief not only to people with epilepsy, but to those struggling with drug addiction problems or the neurodegenerative disorder ALS. "For years, people have been trying to modify glutamate receptors to cure disease," he says. "It's been a very difficult area to develop new drugs in."

In a multinational, blinded, placebo-controlled trial of more than 700 people with uncontrolled partial-onset seizures, roughly one-third of participants saw the frequency of their seizures fall by more than 50 percent when they were given 8 milligrams a day of perampanel. Partial-onset seizures - the most common form in epilepsy - begin in one part of the brain, occurring when there is an injury or abnormality in one of the brain's electrical networks. They can involve anything from the twitching of a limb to confusion to convulsions. Those in this trial typically had roughly 10 seizures a day at baseline. One in 200 Americans have epilepsy and more than half have partial-onset seizures.

The participants in the study, being reported this week in the journal *Neurology*, were all taking one to three anti-epileptic drugs before adding perampanel (or a placebo) to their regimen. Krauss and his colleagues assigned each to receive a placebo, two milligrams, four milligrams or eight milligrams per day of the drug. The lowest effective dose was four milligrams per day and the higher the dose, they found, the better the results. Another trial is currently looking at a 12 milligram per day dose. The most common side effect was dizziness, Krauss says.

The study was paid for by Eisai Inc., a New Jersey-based pharmaceutical firm. Krauss says he believes the U.S. Food and Drug Administration will review perampanel in the next year. H.B. Edwards, B.S., of Johns Hopkins was also involved in the research.

http://www.hopkinsmedicine.org/neurology_neurosurgery/experts/profiles/team_member_profile/8C81D4C308D919948A2C8CD3006E9565/Gregory_Krauss http://www.hopkinsmedicine.org/neurology_neurosurgery/specialty_areas/epilepsy/
http://www.eurekalert.org/pub_releases/2012-04/uow-efa041612.php

Evidence for a geologic trigger of the Cambrian explosion

The oceans teemed with life 600 million years ago, but the simple, soft-bodied creatures would have been hardly recognizable as the ancestors of nearly all animals on Earth today.

MADISON - Then something happened. Over several tens of millions of years – a relative blink of an eye in geologic terms – a burst of evolution led to a flurry of diversification and increasing complexity, including the expansion of multicellular organisms and the appearance of the first shells and skeletons.

The results of this Cambrian explosion are well documented in the fossil record, but its cause – why and when it happened, and perhaps why nothing similar has happened since – has been a mystery.

New research shows that the answer may lie in a second geological curiosity – a dramatic boundary, known as the Great Unconformity, between ancient igneous and metamorphic rocks and younger sediments.

"The Great Unconformity is a very prominent geomorphic surface and there's nothing else like it in the entire rock record," says Shanan Peters, a geoscience professor at the University of Wisconsin–Madison who led the new work. Occurring worldwide, the Great Unconformity juxtaposes old rocks, formed billions of years ago deep within the Earth's crust, with relatively young Cambrian sedimentary rock formed from deposits left by shallow ancient seas that covered the continents just a half billion years ago.

Named in 1869 by explorer and geologist John Wesley Powell during the first documented trip through the Grand Canyon, the Great Unconformity has posed a longstanding puzzle and has been viewed – by Charles Darwin, among others – as a huge gap in the rock record and in our understanding of the Earth's history.

But Peters says the gap itself – the missing time in the geologic record – may hold the key to understanding what happened.

In the April 19 issue of the journal *Nature*, he and colleague Robert Gaines of Pomona College report that the same geological forces that formed the Great Unconformity may have also provided the impetus for the burst of biodiversity during the early Cambrian. "The magnitude of the unconformity is without rival in the rock record," Gaines says. "When we pieced that together, we realized that its formation must have had profound implications for ocean chemistry at the time when complex life was just proliferating." "We're proposing a triggering mechanism for the Cambrian explosion," says Peters. "Our hypothesis is that biomineralization evolved as a biogeochemical response to an increased influx of continental weathering products during the last stages in the formation of the Great Unconformity."

Peters and Gaines looked at data from more than 20,000 rock samples from across North America and found multiple clues, such as unusual mineral deposits with distinct geochemistry, that point to a link between the physical, chemical, and biological effects.

During the early Cambrian, shallow seas repeatedly advanced and retreated across the North American continent, gradually eroding away surface rock to uncover fresh basement rock from within the crust. Exposed to the surface environment for the first time, those crustal rocks reacted with air and water in a chemical weathering process that released ions such as calcium, iron, potassium, and silica into the oceans, changing the seawater chemistry. The basement rocks were later covered with sedimentary deposits from those Cambrian seas, creating the boundary now recognized as the Great Unconformity.

Evidence of changes in the seawater chemistry is captured in the rock record by high rates of carbonate mineral formation early in the Cambrian, as well as the occurrence of extensive beds of glauconite, a potassium-, silica-, and iron-rich mineral that is much rarer today. The influx of ions to the oceans also likely posed a challenge to the organisms living there. "Your body has to keep a balance of these ions in order to function properly," Peters explains. "If you have too much of one you have to get rid of it, and one way to get rid of it is to make a mineral."

The fossil record shows that the three major biominerals – calcium phosphate, now found in bones and teeth; calcium carbonate, in invertebrate shells; and silicon dioxide, in radiolarians – appeared more or less simultaneously around this time and in a diverse array of distantly related organisms.

The time lag between the first appearance of animals and their subsequent acquisition of biominerals in the Cambrian is notable, Peters says. "It's likely biomineralization didn't evolve for something, it evolved in response to something – in this case, changing seawater chemistry during the formation of the Great Unconformity. Then once that happened, evolution took it in another direction." Today those biominerals play essential roles as varied as protection (shells and spines), stability (bones), and predation (teeth and claws).

Together, the results suggest that the formation of the Great Unconformity may have triggered the Cambrian explosion. "This feature explains a lot of lingering questions in different arenas, including the odd occurrences of many types of sedimentary rocks and a very remarkable style of fossil preservation. And we can't help but think this was very influential for early developing life at the time," Gaines says.

Far from being a lack of information, as Darwin thought, the gaps in the rock record may actually record the mechanism as to why the Cambrian explosion occurred in the first place, Peters says.

"The French composer Claude Debussy said, 'Music is the space between the notes.' I think that is the case here," he says. "The gaps can have more information, in some ways, about the processes driving Earth system change, than the rocks do. It's both together that give the whole picture."

The work was supported by the National Science Foundation.

http://www.eurekalert.org/pub_releases/2012-04/aha-npt041312.php

No proof that gum disease causes heart disease or stroke

Despite popular belief, gum disease hasn't been proven to cause heart disease or stroke, and treating gum disease hasn't been proven to prevent heart disease or stroke

Despite popular belief, gum disease hasn't been proven to cause atherosclerotic heart disease or stroke, and treating gum disease hasn't been proven to prevent heart disease or stroke, according to a new scientific statement published in *Circulation*, an American Heart Association journal.

Keeping teeth and gums healthy is important for your overall health. However, an American Heart Association expert committee - made up of cardiologists, dentists and infectious diseases specialists - found no conclusive scientific evidence that gum disease, also known as periodontal disease, causes or increases the rates of cardiovascular diseases.

Current data don't indicate whether regular brushing and flossing or treatment of gum disease can cut the incidence of atherosclerosis, the narrowing of the arteries that can cause heart attacks and strokes.

Observational studies have noted associations between gum disease and cardiovascular disease, but the 500 journal articles and studies reviewed by the committee didn't confirm a causative link.

"There's a lot of confusion out there," said Peter Lockhart, D.D.S., co-chair of the statement writing group and professor and chair of oral medicine at the Carolinas Medical Center in Charlotte, N.C. "The message sent out by some in healthcare professions that heart attack and stroke are directly linked to gum disease, can distort the facts, alarm patients and perhaps shift the focus on prevention away from well known risk factors for these diseases."

Gum disease and cardiovascular disease both produce markers of inflammation such as C-reactive protein, and share other common risk factors as well, including cigarette smoking, age and diabetes mellitus.

These common factors may help explain why diseases of the blood vessels and mouth occur in tandem. Although several studies appeared to show a stronger relationship between these diseases, in those studies researchers didn't account for the risk factors common to both diseases.

"Much of the literature is conflicting," Lockhart said, "but if there was a strong causative link, we would likely know that by now."

A large, long-term study would be needed to prove if dental disease causes heart disease and stroke, he said.

Such a study isn't likely to be done in the near future, and it's most important to let patients know "what we know now, and what we don't know," Lockhart said.

For more than a century, doctors have proposed that infected gums lead to systemic problems like heart disease, and we know that mouth bacteria frequently enter the blood stream during dental procedures and during naturally occurring events such as tooth brushing.

"We already know that some people are less proactive about their cardiovascular health than others. Individuals who do not pay attention to the very powerful and well proven risk factors, like smoking, diabetes or high blood pressure, may not pay close attention to their oral health either" Lockhart said.

Statements that imply a cause and effect relationship between periodontal disease and cardiovascular disease, or claim that dental treatment may prevent heart attack or stroke are "unwarranted," at this time, the statement authors said.

The American Dental Association Council on Scientific Affairs and World Heart Federation endorsed the statement.

The statement's writing group was co-chaired by Ann F. Bolger, M.D. Other co-authors are Panos N. Papapanou, D.D.S., Ph.D.; Olusegun Osinbowale, M.D.; Maurizo Trevisan, M.D.; Matthew E. Levison, M.D.; Kathryn A. Taubert, Ph.D.; Jane W. Newburger, M.D., M.P.H.; Heather L. Gornik, M.D., M.H.S.; Michael H. Gewitz, M.D.; Walter R. Wilson, M.D.; Sidney C. Smith Jr., M.D.; and Larry M. Baddour, M.D. Author disclosures are on the manuscript.

<http://www.bbc.co.uk/news/health-17748165>

'Blind' mice eyesight treated with transplanted cells
British scientists have restored the sight of blind mice by transplanting light-sensitive photoreceptor cells into their eyes.

The work is a step towards a new treatment for patients with degenerative eye diseases. Scientists at University College London Institute of Ophthalmology injected cells from young healthy mice directly into the retinas of adult mice that had night-blindness. The cells transplanted were immature rod-photoreceptor cells, which are especially important for seeing in the dark. After four to six weeks up to one in six of the transplanted cells had formed the connections needed to transmit visual information to the brain. The findings are published in Nature.

Hidden platform

The researchers tested the vision of the treated mice in a dimly-lit water maze. Those mice with transplanted rod cells were able to see a visual cue to find a hidden platform to enable them to get out of the water. This was in contrast to untreated mice who found the platform only by chance after lengthy exploration of the maze.

Prof Robin Ali, at UCL Institute of Ophthalmology and Moorfields Eye Hospital, who led the research said: "We've shown for the first time that transplanted photoreceptor cells can integrate successfully with the existing retinal circuitry and truly improve vision. "We're hopeful that we will soon be able to replicate this success with photoreceptors derived from embryonic stem cells and eventually to develop human trials."

Prof Ali said the behavioural maze test was "ultimate proof" that a significant amount of vision had been restored in the treated mice. But although the results appear promising, there are still many steps to go before such a treatment might be suitable for patients.

There are two types of photoreceptor in the eye - rods and cones. It has so far proved harder to transplant cone photoreceptors - which are crucial for human sight and tasks like reading. The scientists also plan to experiment with photoreceptors derived from embryonic stem cells. Prof Ali said such cell lines already exist but the question is how efficiently they can transplant them. Loss of photoreceptors is the cause of blindness in many human eye diseases including age-related macular degeneration (AMD), retinitis pigmentosa and diabetes-related blindness. But many more animal studies will be needed before such a technique would be tried with humans.

'Great encouragement'

Dr Rob Buckle, head of regenerative medicine at the MRC said: "This is a landmark study that will inform future research across a wide range of fields including vision research, neuroscience and regenerative medicine. "It provides clear evidence of functional recovery in the damaged eye through cell transplantation, providing great encouragement for the development of stem cell therapies to address the many debilitating eye conditions that affect millions worldwide." The research was funded by the Medical Research Council, the Wellcome Trust, the Royal Society the British Retinitis Pigmentosa Society, Alcon Research Institute and The Miller's Trust.

There are already a number of research programmes aiming to treat blindness using cell transplants. Last year, the same research group were given the go-ahead to carry out Europe's first clinical trial involving human embryonic stem cells at Moorfields Eye Hospital. That study involves patients with Stargardt's disease, one of the main causes of blindness in young people. Early results suggest the technique is safe but reliable results will take several years.

<http://www.sciencedaily.com/releases/2012/04/120418135045.htm>

How Selective Hearing Works in the Brain: 'Cocktail Party Effect' Explained
The longstanding mystery of how selective hearing works - how people can tune in to a single speaker while tuning out their crowded, noisy environs - is solved this week in the journal Nature by two scientists from the University of California, San Francisco (UCSF).

ScienceDaily - Psychologists have known for decades about the so-called "cocktail party effect," a name that evokes the Mad Men era in which it was coined. It is the remarkable human ability to focus on a single speaker in virtually any environment -- a classroom, sporting event or coffee bar -- even if that person's voice is seemingly drowned out by a jabbering crowd.

To understand how selective hearing works in the brain, UCSF neurosurgeon Edward Chang, MD, a faculty member in the UCSF Department of Neurological Surgery and the Keck Center for Integrative Neuroscience, and UCSF postdoctoral fellow Nima Mesgarani, PhD, worked with three patients who were undergoing brain surgery for severe epilepsy.

Part of this surgery involves pinpointing the parts of the brain responsible for disabling seizures. The UCSF epilepsy team finds those locales by mapping the brain's activity over a week, with a thin sheet of up to 256

electrodes placed under the skull on the brain's outer surface or cortex. These electrodes record activity in the temporal lobe -- home to the auditory cortex.

UCSF is one of few leading academic epilepsy centers where these advanced intracranial recordings are done, and, Chang said, the ability to safely record from the brain itself provides unique opportunities to advance our fundamental knowledge of how the brain works.

"The combination of high-resolution brain recordings and powerful decoding algorithms opens a window into the subjective experience of the mind that we've never seen before," Chang said. In the experiments, patients listened to two speech samples played to them simultaneously in which different phrases were spoken by different speakers. They were asked to identify the words they heard spoken by one of the two speakers.

The authors then applied new decoding methods to "reconstruct" what the subjects heard from analyzing their brain activity patterns. Strikingly, the authors found that neural responses in the auditory cortex only reflected those of the targeted speaker. They found that their decoding algorithm could predict which speaker and even what specific words the subject was listening to based on those neural patterns. In other words, they could tell when the listener's attention strayed to another speaker. "The algorithm worked so well that we could predict not only the correct responses, but also even when they paid attention to the wrong word," Chang said.

Speech Recognition by the Human Brain and Machines

The new findings show that the representation of speech in the cortex does not just reflect the entire external acoustic environment but instead just what we really want or need to hear. They represent a major advance in understanding how the human brain processes language, with immediate implications for the study of impairment during aging, attention deficit disorder, autism and language learning disorders.

In addition, Chang, who is also co-director of the Center for Neural Engineering and Prostheses at UC Berkeley and UCSF, said that we may someday be able to use this technology for neuroprosthetic devices for decoding the intentions and thoughts from paralyzed patients that cannot communicate.

Revealing how our brains are wired to favor some auditory cues over others it may even inspire new approaches toward automating and improving how voice-activated electronic interfaces filter sounds in order to properly detect verbal commands.

How the brain can so effectively focus on a single voice is a problem of keen interest to the companies that make consumer technologies because of the tremendous future market for all kinds of electronic devices with voice-active interfaces. While the voice recognition technologies that enable such interfaces as Apple's Siri have come a long way in the last few years, they are nowhere near as sophisticated as the human speech system.

An average person can walk into a noisy room and have a private conversation with relative ease -- as if all the other voices in the room were muted. In fact, said Mesgarani, an engineer with a background in automatic speech recognition research, the engineering required to separate a single intelligible voice from a cacophony of speakers and background noise is a surprisingly difficult problem. Speech recognition, he said, is "something that humans are remarkably good at, but it turns out that machine emulation of this human ability is extremely difficult."

This work was funded by the National Institutes of Health and the Ester A. and Joseph Klingenstein Foundation. *The above story is reprinted from materials provided by University of California - San Francisco. The original article was written by Jason Bardi.*

Journal Reference: Nima Mesgarani, Edward F. Chang. Selective cortical representation of attended speaker in multi-talker speech perception. *Nature*, 2012; DOI: 10.1038/nature11020

<http://phys.org/news/2012-04-tokyo-mega-quake.html>

Tokyo mega-quake 'would kill over 9,000'

More than 9,600 people would die with nearly 150,000 injured if a mega-quake struck Tokyo, a disaster that would also level large parts of the Japanese capital, a government projection said Wednesday.

The frightening simulation was released by the Tokyo Metropolitan Government as Japan slowly rebuilds its northeast coast, which was devastated by a magnitude 9.0 quake in March last year that unleashed a deadly tsunami. The disaster killed some 19,000 people and triggered the worst nuclear accident in a generation.

Tokyo was largely spared from the damage, but if a smaller 7.3-magnitude quake struck the sprawling metropolis it would leave about 9,600 dead and 147,000 people with injuries, including 21,900 seriously, the projection said.

About 5.2 million people would be unable to go home owing to electricity and transportation damage while the temblor would flatten or seriously damage some 378,000 buildings with about 188,000 structures burning to the ground. A huge tsunami would strike isolated Pacific Ocean islands several hundred kilometres outside

Tokyo, which are considered part of the municipality, but was not likely to cause damage or fatalities in the metropolis itself.

The biggest city in earthquake-prone Japan lies at the intersection of four tectonic plates and there is a 50 percent chance it will be struck by a magnitude-7.0 or higher quake in the next four years, according to the University of Tokyo's Earthquake Research Institute. The government projection does not include fatalities and damage in outlying prefectures that make up Greater Tokyo, home to about 35.0 million people.

In 1923, Tokyo and surrounding areas were struck by a 7.9 magnitude quake that left more than 140,000 people dead and destroyed much of the city.

<http://phys.org/news/2012-04-astronomers-coolest-radio-star.html>

Astronomers detect coolest radio star

Astronomer, have discovered flaring radio emission from an ultra-cool star, not much warmer than the planet Jupiter

Phys.org - Astronomers using the world's largest radio telescope, at Arecibo, Puerto Rico, have discovered flaring radio emission from an ultra-cool star, not much warmer than the planet Jupiter, shattering the previous record for the lowest stellar temperature at which radio waves were detected.

The team from Penn State University's Department of Astronomy and Astrophysics and the Center for Exoplanets and Habitable Worlds has been using the giant 305-m (1000-ft) telescope to look for radio signals from a class of objects known as brown dwarfs. These are small, cold stars that bridge the gap between Jupiter-like giant planets and normal, more massive, hydrogen-fusing stars. They hit the jackpot with a star named J1047+21, a brown dwarf 33.6 light years away in the constellation Leo, in a result that could boost the odds of discovering life elsewhere in the universe.

Matthew Route, a graduate student at Penn State and the lead author of the discovery paper, said, "This object is the coolest brown dwarf ever seen in the radio - it's half the temperature of the previous record holder, making it only about five times hotter than Jupiter." The new radio-star is much smaller and colder than our Sun. With a surface temperature not much higher than that of a giant planet, and a size comparable to Jupiter's, it is scarcely visible in optical light. Yet the radio flares seen at Arecibo show it must have a strong magnetic field, implying that the same could be true of other similar stars.

Dr. Alex Wolszczan, who is leading the project, said, "This is a really exciting result. We hope that in the future we'll be able to detect yet colder brown dwarfs, and possibly even giant planets around other stars."

The possibility that young, hot planets around other stars could be detected in the same manner - because they still maintain strong magnetic fields - has implications for the chances of finding life elsewhere in the Galaxy, Dr. Wolszczan explained. "The Earth's field protects life on its surface from harmful particles of the solar wind. Knowing whether planetary magnetic fields are common or not throughout the Galaxy will aid our efforts to understand chances that life may exist beyond the Solar System."

The discovery of radio signals from J1047+21 dramatically broadens the window through which astronomers can study the atmospheres and interiors of these tiny stars, using the radio detection of their magnetic fields as a tool. At the temperature of this brown dwarf, its atmosphere must be made of neutral gas, which would not give off radio signals like those seen. The energy to drive the signals is likely to come from magnetic fields deep inside the star, similar to the field that protects the Earth from dangerous high-energy particles. By monitoring the radio flares from J1047+21, astronomers will be able to tell how stable the magnetic field is over time, and, from flare duration, they can infer the size of the emitter itself.

The results were published in the March 10 edition of the Letters section of the *Astrophysical Journal*.

More information: <http://dx.doi.org/10.1088/2041-8205/747/2/L22>

<http://www.scientificamerican.com/article.cfm?id=dinosaurs-grew-outpace-their-young>

Dinosaurs Grew to Outpace Their Young

Ancient reptiles owed huge size more to their eggs than to a benign environment.

By Matt Kaplan of Nature magazine

Some dinosaurs grew to gigantic sizes to avoid competition from their own young, rather than to take advantage of abundant oxygen, high temperatures and large territorial ranges, say two studies. But their largeness may also have proved their undoing.

Some have argued that dinosaurs were able to grow quickly and fuel large bodies when temperatures were warm, oxygen levels were high, and land masses such as the supercontinent Gondwana provided abundant living space.

But although the idea that certain environmental conditions favored the growth of enormous dinosaurs has been popular among paleontologists, there is little evidence for it.

Friendly environment

To see whether the link could be supported, Roland Sookias, a biologist at the Ludwig Maximilian University of Munich in Germany, and his colleagues examined whether changes in body size followed changes in environmental factors. Their findings are published in *Biology Letters* today¹.

The team used thigh-bone lengths to work out the body sizes of more than 400 species alive during the Permian, Triassic and Jurassic periods (299 million to 145 million years ago). This included dinosaurs and their predecessors, as well as contemporaries such as flying pterosaurs and the ancestors of mammals.

The researchers compared body sizes with records of atmospheric oxygen levels, temperatures and the available area of dry land when these animals were alive, and found no correlation. To check their results, they also compared the same environmental variables with the sizes of mammals living between the Palaeocene and Pleistocene epochs (65 million to 0.01 million years ago). Again, there was no connection.

"Our results support the idea that biological factors, such as growth rates, were more important in governing maximum body size" than were environmental factors, says Richard Butler, a paleontologist at the Ludwig Maximilian University of Munich and a co-author of the study.

"The model is good and the findings are probably right but we must remember this is very broad-brush stuff," says Paul Barrett, a paleontologist at the Natural History Museum in London. "We mustn't take this to mean we can start entirely ignoring the interactions that exist between environmental factors and physiology."

Bigger and bigger

In another study published in *Biology Letters* today², Daryl Codron, a zoologist at the University of Zurich in Switzerland, and his colleagues argue that the key to some dinosaurs' vast size lies in the limitations of egg laying. Large eggs must have thick shells, which make it difficult for the developing embryo to 'breathe' by exchanging gases with the outside world. This places an upper limit on egg size.

So, even though they grew into giants as adults, dinosaurs were forced to produce relatively tiny young. Titanosaur hatchlings, for example, were nearly 2,500 times smaller than the 4-tonne adults. By contrast, the live-born calf of an Asian elephant (*Elephas maximus*) is about 25 times smaller than its mother.

When the young of large animals start out small, they must grow through a large size range before reaching adulthood, and compete with species of many different sizes as they do so.

Codron and his colleagues developed a model that suggests that there was intense competition among small and medium-sized dinosaurs, so that it was difficult for medium-sized adults to make a living, and adults had to keep growing until they reached very large sizes to gain a competitive edge. In comparison to mammals, relatively few dinosaurs are known with adult sizes between 1 kilogram and 1,000 kilograms.

But being big also has drawbacks. When an asteroid impact at the end of the Cretaceous period (65 million years ago) wiped out most large-bodied animals, there were so few small dinosaur species that the group was almost obliterated, with only the birds surviving. However, the many small mammals alive at the time were well suited to a world that favored diminutive species.

"I love the idea that egg-laying led dinosaurs to engage in such different niche occupation from mammals and that this played a part in their eventual extinction," says John Hutchinson, an evolutionary biologist at the Royal Veterinary College in Hatfield, UK. "But we are going to see a lot of debate over how accurate these models really are," he adds.

http://www.eurekalert.org/pub_releases/2012-04/ip-1ct041912.php

19th century therapy for Parkinson's disease may help patients today ***Research published in the Journal of Parkinson's Disease***

Amsterdam, NL - In the 19th century, the celebrated neurologist, Jean-Martin Charcot, developed a "vibration chair" to relieve symptoms of Parkinson's disease. He reported improvements in his patients, but he died shortly thereafter and a more complete evaluation of the therapy was never conducted. Now a group of scientists at Rush University Medical Center have replicated his work, and they report that while vibration therapy does significantly improve some symptoms of Parkinson's disease, the effect is due to placebo or other nonspecific factors, and not the vibration. Their study is published in the April issue of *Journal of Parkinson's Disease*.

"We attempted to mimic Charcot's protocol with modern equipment in order to confirm or refute an historical observation," explains lead investigator Christopher G. Goetz, MD, director of the Rush University Medical Center Parkinson's disease and movement disorders program. "Both the treated group and the control group improved similarly, suggesting other factors had an effect on Parkinson's disease motor function."

Charcot's patients told him that during long carriage rides or train journeys, uncomfortable or painful symptoms of Parkinson's disease seemed to disappear, and the relief lasted quite some time after the journey. He developed a chair that mimicked the continuous jerking of a carriage or train. In their current study, Dr.

Goetz and his colleagues randomly assigned 23 patients to either a vibrating chair or the same chair without vibration. During the treatment sessions, both groups listened to a relaxation CD of nature sounds. They underwent daily treatment for a month.

The patients in the vibration treatment group showed significant improvement in motor function after daily 30-minute treatments for four weeks. Although not as high, motor function scores for the no vibration group also improved significantly. Both groups showed similar and significant improvement in depression, anxiety, fatigue, and nighttime sleep and both groups reported similar high satisfaction with their treatment.

"Our results confirm Charcot's observation of improvement in Parkinson's disease symptomology with chronic vibration treatment, but we did not find the effect specific to vibration," Dr. Goetz says. "Instead, our data suggest that auditory sensory stimulation with relaxation in a lounge chair or simply the participation in a research protocol has equivalent benefit as vibration on motor function."

Dr. Goetz concludes, "While we can agree that our results may not change scientific thinking on treatment mechanisms, our results will allow clinicians to guide patients to at least one apparatus that is safe and associated with objective changes in parkinsonian impairment scores. Charcot's advice to colleagues resonates as one places vibration therapy in the context of potential options for patients. It is no small gain to be able to relieve the sufferers of paralysis agitans."

http://www.eurekalert.org/pub_releases/2012-04/uow-pic041512.php

Payment innovation cuts depression time in half

Quality-of-care incentives to community health clinics shortened the duration of depression in a socioeconomically vulnerable patient population

When 25 percent of the payments to community health clinics were based on quality of care, patients received better care and had better depression outcomes. The results of this initiative will be published in the April 19 issue of the American Journal of Public Health in the paper, "Quality Improvement with Pay-for-Performance Incentives in Integrated Behavioral Health Care."

University of Washington researchers examined records from almost 8,000 patients treated for depression in 29 community health clinics in the Washington State Mental Health Integration Program before and after the implementation of a pay-for-performance incentive. After the incentive was started, patients were seen more quickly and were more likely to receive consultation from a psychiatrist. They were also more likely to show improvements in their depression. "The time for depression to improve in the majority of patients was cut by more than half, from over 60 weeks to less than 25 weeks," said Dr. Jürgen Unützer, professor and vice chair of the UW Department of Psychiatry and Behavioral Sciences, the lead author of the study.

The Washington State Mental Health Integration Program provides medical and mental health services for low-income adults who are temporarily disabled due to a physical or mental health condition and expected to be unemployed for at least 90 days. This program is funded by the State of Washington and administered by the Community Health Plan of Washington, a non-profit managed care plan.

In King County the program also covers military veterans and their family members, the uninsured, low-income mothers and their children, and low-income older adults. Services in King County are also funded by a voter-approved levy that is administered by Public Health – Seattle & King County.

Treatment for depression and other common mental disorders is provided through an innovative team approach in which primary care providers are supported by a trained mental health care coordinator and a consulting psychiatrist. Expert faculty members from the UW's Advancing Integrated Mental Health Solutions (AIMS) Center provide training, technical assistance, and a web-based tracking system to support systematic outcome tracking and quality improvement.

Unützer said he was struck by the program's effectiveness with this highly vulnerable group of patients.

"Most of the program participants were unemployed due to a medical or a mental health problem and more than half had problems with stable housing," he said. "Despite these challenges, we found that the majority of program participants achieved improvements in their depression."

Unützer pointed to the Mental Health Integration Program and its pay-for-performance component as a prime example of achieving the healthcare "triple aim." The triple aim seeks to enhance the patients' experience of care and improve health outcomes while containing or reducing healthcare costs.

Each year more than 20 million Americans suffer from depression. The World Health Organization ranks depression among the leading causes of disease burden worldwide. Most patients with depression seek help in primary care clinics, but in many cases their depression is not recognized or treated effectively.

Ineffectively treated depression leads to tremendous suffering in patients and their loved ones, increased mortality and higher healthcare costs. The researchers noted that today's study demonstrates that with the right

incentives and support community health clinics can substantially improve care and clinical outcomes for vulnerable patients with depression.

http://www.eurekalert.org/pub_releases/2012-04/aaon-suo041012.php

Use of drug following first sign of possible MS reduces likelihood of progression to MS
People who received injections of the multiple sclerosis (MS) drug interferon beta-1a soon after their first signs of possible MS were less likely to progress to clinically definite MS than people who switched to interferon beta-1a from placebo, according to new phase three results of the three-year

New Orleans - People who received injections of the multiple sclerosis (MS) drug interferon beta-1a soon after their first signs of possible MS were less likely to progress to clinically definite MS than people who switched to interferon beta-1a from placebo, according to new phase three results of the three-year REFLEXION clinical trial that will be presented as part of the Emerging Science program (formerly known as Late-Breaking Science) at the American Academy of Neurology's 64th Annual Meeting in New Orleans, April 21 - 28, 2012.

The trial was conducted with the human serum albumin-free formulation of interferon beta-1a, which is now available in all European Union countries, Australia, Canada and Switzerland, as well as a number of countries in Asia, Latin America, Africa and the Middle East. It is not available in the United States.

"While we've known it's beneficial to start MS drugs as soon as possible, this is the first trial to show a benefit of early injections of interferon beta-1a treatment at three years," said Mark Freedman, MD, with the University of Ottawa in Ontario, Canada, and a Fellow of the American Academy of Neurology.

The three-year clinical trial involved 517 people who had experienced a first clinical episode suggestive of a demyelinating event, such as tingling, numbness, muscle weakness or problems with balance, along with having at least two clinically silent brain lesions detected by a brain MRI scan.

For two years, one-third of the participants received 44 mcg of interferon beta-1a subcutaneously three times a week; one-third received 44 mcg of the drug once a week, which is an unapproved dosage; and another one-third received placebo for two years or until experiencing a second clinical episode, at which point they were switched to three-times-weekly dosing. After the two years were over, the 133 people who were still receiving the placebo were switched to the three-times-weekly dose and the others continued their originally allocated dosages. The participants started their treatment an average of 58 days after their first symptoms.

After the third year of the study, the researchers found that those who had been receiving the drug three times a week or once a week for the duration of the trial were less likely to be diagnosed with clinically definite MS (defined as having a second clinical attack or a sustained increase in the Expanded Disability Status Scale disability score of greater than 1.5) than those who had initially received the placebo. The cumulative probability of being diagnosed with clinically definite MS by the end of the third year was 41 percent for the delayed treatment group (people who had switched from placebo to three-times-weekly treatment), 28 percent for those who had received once-a-week treatment all three years, and 27 percent for those who had three-times-weekly treatment for three years.

The study also found that those who had received the treatment for the full three years were less likely to meet the McDonald criteria for a MS diagnosis, a different measure than the one for clinically definite MS that includes an evaluation of MRI. A total of 87 percent of people who had switched from placebo to the treatment after two years met the McDonald criteria for MS after three years, compared with 79 percent of those who had received the weekly treatment and 67 percent of those who had treatments three times a week.

"While doses three times a week and once a week equally delayed a clinically definite MS diagnosis without MRI measures, there were significantly more benefits in taking the drug three times a week compared with once a week when it came to brain lesion changes and other McDonald criteria for diagnosing MS," said Freedman. The REFLEXION trial is ongoing and will provide long-term data out to five years. The most common adverse events in the trial were flu-like symptoms, injection site reactions and headache.

The study was supported by Merck Serono S.A. – Geneva, Switzerland.

http://www.eurekalert.org/pub_releases/2012-04/plos-ftr041612.php

Finding the roots and early branches of the tree of life
Tracing the tree of life back to a single ancestral form

A study published in PLoS Computational Biology maps the development of life-sustaining chemistry to the history of early life. Researchers Rogier Braakman and Eric Smith of the Santa Fe Institute traced the six methods of carbon fixation seen in modern life back to a single ancestral form.

Carbon fixation – life's mechanism for making carbon dioxide biologically useful – forms the biggest bridge between Earth's non-living chemistry and its biosphere. All organisms that fix carbon do so in one of six ways.

These six mechanisms have overlaps, but it was previously unclear which of the six types came first, and how their development interweaved with environmental and biological changes.

The authors used a method that creates "trees" of evolutionary relatedness based on genetic sequences and metabolic traits. From this, they were able to reconstruct the complete early evolutionary history of biological carbon-fixation, relating all ways in which life today performs this function.

The earliest form of carbon fixation identified achieved a special kind of built-in robustness – not seen in modern cells – by layering multiple carbon-fixing mechanisms. This redundancy allowed early life to compensate for a lack of refined control over its internal chemistry, and formed a template for the later splits that created the earliest major branches in the tree of life. For example, the first major life-form split came with the earliest appearance of oxygen on Earth, causing the ancestors of blue-green algae and most other bacteria to separate from the branch that includes Archaea, which are outside of bacteria the other major early group of single-celled microorganisms.

"It seems likely that the earliest cells were rickety assemblies whose parts were constantly malfunctioning and breaking down," explains Smith. "How can any metabolism be sustained with such shaky support? The key is concurrent and constant redundancy."

Once early cells had more refined enzymes and membranes, giving greater control over metabolic chemistry, minimization of energy (ATP) used to create biomass, changes in oxygen levels and alkalinity directed life's unfolding. In other words, the environment drove major divergences in predictable ways, in contrast to the common belief that chance dominated evolutionary innovation – and that rewinding and replaying the evolutionary tape would lead to an irreconcilably different tree of life. "Mapping cell function onto genetic history gives us a clear picture of the physiology that led to the major foundational divergences of evolution," explains Braakman. "This highlights the central role of basic chemistry and physics in driving early evolution."

With the ancestral form uncovered, and evolutionary drivers pinned to branching points in the tree, the researchers now want to make the study more mathematically formal and further analyze the early evolution of metabolism.

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PLoS Comput Biol 8(4): e1002455. doi:10.1371/journal.pcbi.1002455

http://www.eurekalert.org/pub_releases/2012-04/eaft-gmt041712.php

Gut microbiota transplantation may prevent development of diabetes and fatty liver disease

Data presented today shows the gut microbiota's causal role in the development of diabetes and non-alcoholic fatty liver disease, independent of obesity.

Barcelona, Spain - Exciting new data presented today at the International Liver Congress™ 2012 shows the gut microbiota's causal role in the development of diabetes and non-alcoholic fatty liver disease (NAFLD), independent of obesity.¹ Though an early stage animal model, the French study highlights the possibility of preventing diabetes and NAFLD with gut microbiota transplantation – the engrafting of new microbiota, usually through administering faecal material from a healthy donor into the colon of a diseased recipient.²

In the 16 week study, two groups of germ free mice received gut microbiota transplants; one set from donor mice displaying symptoms of insulin resistance and liver steatosis (responders), the other from normal mice (non responders). The donor mice were selected due to their response to being fed a high fat diet.

The germ free group that received microbiota from symptomatic mice (responder receivers - RR) showed higher levels of fat concentration in the liver as well as being insulin resistant. The germ free group that received microbiota from healthy mice (non-responder-receivers – NRR) maintained normal glucose levels and sensitivity to insulin.

EASL Scientific Committee Member Dr Frank Lammert said: "The factors leading to Non-Alcoholic Fatty Liver Disease (NAFLD) are poorly understood, but it is known that NAFLD and Type 2 diabetes are characterised, respectively, by liver inflammation and metabolic disorders like insulin resistance."

"This study shows that different microbiota cause different metabolic responses in animals. By implanting microbiota from healthy mice, the study authors prevented the development of liver inflammation and insulin resistance, both indications of liver disease and diabetes. Thus, gut microbiota transplants could have a therapeutic role in the development of these diseases."

The RR mice also showed lower levels of microorganisms than usually found in the healthy gut. Lachnospiraceae was identified as the species most important in developing fatty liver and insulin resistance.

At present, the intestinal microbiota is considered to constitute a "microbial organ": one that has pivotal roles in the body's metabolism as well as immune function. Therefore transplantation aims to restore gut functionality and re-establish a certain state of intestinal flora.

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McMaster researchers find potential for new uses of old drug New evidence is helping explain additional health benefits of aspirin

Researchers in Canada, Scotland and Australia have discovered that salicylate, the active ingredient in aspirin, directly increases the activity of the protein AMPK (AMP-activated protein kinase), a key player in regulating cell growth and metabolism. AMPK which is considered a cellular fuel-gauge is switched on by exercise and the commonly used anti-diabetic medication metformin. The research from scientists at McMaster University, the University of Dundee and the University of Melbourne will be published in today's issue of the journal *Science*.

"We're finding this old dog of aspirin already knows new tricks," said Dr. Greg Steinberg, a co-principal investigator of the study. "In the current paper we show that, in contrast to exercise or metformin which increase AMPK activity by altering the cells energy balance, the effects of salicylate is totally reliant on a single Ser108 amino acid of the beta 1 subunit.

"We show that salicylate increases fat burning and reduces liver fat in obese mice and that this does not occur in genetically modified mice lacking the beta1 subunit of AMPK," he said. Steinberg is an associate professor of medicine in the Michael G. DeGroot School of Medicine at McMaster University and the Canada Research Chair in Metabolism and Obesity. These findings are important as a large clinical trial is currently underway testing whether salicylate (a well-tolerated aspirin derivative), can prevent Type 2 diabetes.

Salicylate, which is derived from willow bark, and is the active ingredient in aspirin, is believed to be one of the oldest drugs in the world with first reports of its use dating back to an Egyptian papyrus in 1543 BC.

An anti-inflammatory drug first used as a painkiller more than a century ago, aspirin is now given to people at risk of heart attacks and strokes as well as patients with vascular disease. McMaster scientists played a key role in that previous research. Three studies published last month in the medical journal *The Lancet* reported that taking an aspirin every day may significantly reduce the risk of many cancers and prevent tumors from spreading. The unanswered question was how this anti-cancer benefit occurs.

With many recent studies showing that metformin may be important for cancer prevention the authors' study raise the interesting possibility that aspirin may also be working in a similar manner; however, further studies are needed as the concentrations of salicylate used in the current study were higher than the cancer trials. Nonetheless, the researchers' results show the one thing that salicylates and metformin hold in common is their ability to activate AMPK.

http://www.eurekalert.org/pub_releases/2012-04/nu-nbi041912.php

New brain-machine interface moves a paralyzed hand New technology bypasses spinal cord and delivers electrical signals from brain directly to muscles

CHICAGO - A new Northwestern Medicine brain-machine technology delivers messages from the brain directly to the muscles -- bypassing the spinal cord -- to enable voluntary and complex movement of a paralyzed hand. The device could eventually be tested on, and perhaps aid, paralyzed patients. "We are eavesdropping on the natural electrical signals from the brain that tell the arm and hand how to move, and sending those signals directly to the muscles," said Lee E. Miller, the Edgar C. Stuntz Distinguished Professor in Neuroscience at Northwestern University Feinberg School of Medicine and the lead investigator of the study, which was published in *Nature*. "This connection from brain to muscles might someday be used to help patients paralyzed due to spinal cord injury perform activities of daily living and achieve greater independence."

The research was done in monkeys, whose electrical brain and muscle signals were recorded by implanted electrodes when they grasped a ball, lifted it and released it into a small tube. Those recordings allowed the researchers to develop an algorithm or "decoder" that enabled them to process the brain signals and predict the patterns of muscle activity when the monkeys wanted to move the ball.

These experiments were performed by Christian Ethier, a post-doctoral fellow, and Emily Oby, a graduate student in neuroscience, both at the Feinberg School of Medicine. The researchers gave the monkeys a local anesthetic to block nerve activity at the elbow, causing temporary, painless paralysis of the hand. With the help of the special devices in the brain and the arm – together called a neuroprosthesis -- the monkeys' brain signals were used to control tiny electric currents delivered in less than 40 milliseconds to their muscles, causing them to contract, and allowing the monkeys to pick up the ball and complete the task nearly as well as they did before.

"The monkey won't use his hand perfectly, but there is a process of motor learning that we think is very similar to the process you go through when you learn to use a new computer mouse or a different tennis racquet. Things are different and you learn to adjust to them," said Miller, also a professor of physiology and of physical medicine and rehabilitation at Feinberg and a Sensory Motor Performance Program lab chief at the Rehabilitation Institute of Chicago.

Because the researchers computed the relationship between brain activity and muscle activity, the neuroprosthesis actually senses and interprets a variety of movements a monkey may want to make, theoretically enabling it to make a range of voluntary hand movements. "This gives the monkey voluntary control of his hand that is not possible with the current clinical prostheses," Miller said.

The Freehand prosthesis is one of several prostheses available to patients paralyzed by spinal cord injuries that are intended to restore the ability to grasp. Provided these patients can still move their shoulders, an upward shrug stimulates the electrodes to make the hand close, a shrug down stimulates the muscles to make the hand open. The patient also is able to select whether the prosthesis provides a power grasp in which all the fingers are curled around an object like a drinking glass, or a key grasp in which a thin object like a key is grasped between the thumb and curled index finger.

In the new system Miller and his team have designed, a tiny implant called a multi-electrode array detects the activity of about 100 neurons in the brain and serves as the interface between the brain and a computer that deciphers the signals that generate hand movements. "We can extract a remarkable amount of information from only 100 neurons, even though there are literally a million neurons involved in making that movement," Miller said. "One reason is that these are output neurons that normally send signals to the muscles. Behind these neurons are many others that are making the calculations the brain needs in order to control movement. We are looking at the end result from all those calculations."

The research was supported by the National Institutes of Health/NINDS grant #NS053603, the Chicago Community Trust through the Searle Program for Neurological Restoration at the Rehabilitation Institute of Chicago, and the Fonds de recherche en santé du Québec.

<http://nyti.ms/HHKpx9>

Beneath That Beguiling Smile, Seeing What Leonardo Saw

UNTIL recently, the Prado's copy of the Mona Lisa - one of dozens made over the centuries - was not much of a draw. Then, Ana González Mozo took an interest.

Samuel Aranda for The New York Times

Over the last two years, Ms. González, a researcher in the museum's technical documentation department, has used all manner of modern-day techniques — X-rays, infrared reflectography and high-resolution digital images, among others — to make, and then document, an unlikely finding.

It turns out that the Prado's Mona Lisa is not just any 500-year-old copy. It was most likely painted by someone who was sitting right next to Leonardo da Vinci, trying to duplicate his every brush stroke, as he produced his famous lady with the enigmatic smile. When Leonardo adjusted the size of the Mona Lisa's head or corrected her hands or slimmed her bosom or lowered her bodice, so did whoever was painting the Prado's Mona Lisa. "It had to be painted at the same time," Ms. González said. "Someone who copies doesn't make corrections because they haven't ever seen the changes. They can see only the surface of the painting."

The discovery is primarily important for what it reveals about the real Mona Lisa, a painting that has been darkened by layers of aging lacquer. The copy, now restored, offers details that are obscured in the original Mona Lisa. For instance, the copy shows an armrest where none can be seen in the original, and reflectographs show a much clearer image of her waistline. "What is really important about the copy is that we can see how Leonardo worked," Ms. González said. "We know something new about his creative process."

The copy, which also shows a much younger-looking figure, has once again ignited a debate about whether Leonardo's Mona Lisa should be restored as well. Ms. González says this is a hard call for the Louvre because people are so used to the way the painting looks now. But she cannot help being curious.

Most of the time, Ms. González spends her hours looking beneath the surface of the Prado's masterpieces, searching for insights into the artists' methods and thinking. And there, she said, she has found great treasures.

Many important paintings have sketches or first tries — adjusted and reworked — under the final image. Sometimes, she said, the work underneath is even more fascinating than the painting itself.

“I get to see what only the artist saw,” she said. “And he saw it five centuries ago.”

ON a recent visit, Ms. González’s work space was as cool and tidy as any computer lab. Only a messy pool of life-size images of the Louvre’s Mona Lisa and the Prado’s copy spread out on a table suggested her purpose.

She ran her hands over the photographs, pausing over the similarities; they were clear even to the untrained eye. What was she thinking when she made these discoveries? Was she in awe?

She shrugged off such questions. “Other people have asked me that,” she said, by way of an answer. “I am very calm, very prudent. When I made the discovery, I talked to the curator here.”

Some art magazines have speculated that the Prado’s Mona Lisa was painted by Leonardo’s lover. But Ms. González has no patience for such gossipy talk. “That is irrelevant,” she said. “We don’t know that. And that is not what the work here is about.” Until two years ago, the Prado, which inherited the painting with the rest of the royal collection in 1819, displayed it but never suspected its significance. It was catalogued without fanfare as an anonymous copy, painted on poplar.

The copy’s background was black, and the painting was covered in a layer of dark varnish, which gave it a yellowish glow and further diminished its vibrancy. But the Louvre was planning a special exhibition of Leonardo’s work and, because it did not want to move the original Mona Lisa from its protected area, wanted to borrow the Prado painting as a stand-in. A casual comment by one of the Louvre curators, asking whether the painting had ever been studied, got Ms. González thinking.

The next day she took her infrared camera into the gallery and got to work. Just the first pictures were enough for her to conclude that the two paintings had been produced in tandem. After that, it was just a question of watching the evidence pile up.

Perhaps the most exciting discovery was that the painting’s original background had been obscured by a layer of black paint, a practice sometimes used in the 18th century. Luckily, a layer of lacquer protected what was under it. So, once the paint was removed, the same Tuscan background as in Leonardo’s painting appeared, offering a tantalizing preview of what might be seen if Leonardo’s Mona Lisa were restored.

THERE is no doubt, however, that the Prado painting was not a copy made by Leonardo himself. While the corrections are identical, the lines are not. “Like I write an A and you write an A, you can tell it is not the same,” Ms. González said. Parts of the Prado copy are beautiful, she said, like the hands. But in general, it is not nearly so fine a painting. Just why it was made remains an open question. It could have been simply for a pupil’s instruction or a double commission.

Ms. González started working at the Prado 16 years ago, when she was completing her doctorate, one of the first art researchers to focus on the use of computer techniques to study paintings. “We did not even have Windows when I started as a student,” she said. But little by little, she said, computer techniques of all kinds have become important tools in studying paintings.



The Copy’s Restoration *A layer of black paint covered the background of the copy of the Mona Lisa, left. The black layer was removed during a recent restoration, revealing a preserved background.*

Everyone in her family is a scientist, she said. Her choice to get a fine arts degree was a sort of rebellion. When she begins studying a painting, she does a drawing of it, she said, as a way to familiarize herself with the work.

Ms. González seems somewhat indifferent to the attention her recent discovery is getting. She said she had participated in far more spectacular discoveries. For instance, she said, X-rays and infrared reflectographs show that Tintoretto sketched nude figures under his clothed ones. But, somehow, it is the copy of the Mona Lisa that everyone is talking about. “It has grabbed people’s imagination,” she said. “She is an icon.”

The Prado’s Mona Lisa is on loan to the Louvre until June.

Rachel Chaundler contributed reporting.

Strange cousins: Molecular alternatives to DNA, RNA offer new insight into life's origins
Living systems owe their existence to a pair of information-carrying molecules: DNA and RNA.

These fundamental chemical forms possess two features essential for life: they display heredity—meaning they can encode and pass on genetic information, and they can adapt over time, through processes of Darwinian evolution. A long-debated question is whether heredity and evolution could be performed by molecules other than DNA and RNA.

John Chaput, a researcher at ASU's Biodesign Institute, who recently published an article in *Nature Chemistry* describing the evolution of threose nucleic acids, joined a multidisciplinary team of scientists from England, Belgium and Denmark to extend these properties to other so-called Xenonucleic acids or XNA's.

The group demonstrates for the first time that six of these unnatural nucleic acid polymers are capable of sharing information with DNA. One of these XNAs, a molecule referred to as anhydrohexitol nucleic acid or HNA was capable of undergoing directed evolution and folding into biologically useful forms.

Their results appear in the current issue of *Science*.

The work sheds new light on questions concerning the origins of life and provides a range of practical applications for molecular medicine that were not previously available.

Nucleic acid aptamers, which have been engineered through in vitro selection to bind with various molecules, act in a manner similar to antibodies—latching onto their targets with high affinity and specificity. "This could be great for building new types of diagnostics and new types of biosensors," Chaput says, pointing out that XNAs are heartier molecules, not recognized by the natural enzymes that tend to degrade DNA and RNA. New therapeutics may also arise from experimental Xenobiology.

Both RNA and DNA embed data in their sequences of four nucleotides—information vital for conferring hereditary traits and for supplying the coded recipe essential for building proteins from the 20 naturally occurring amino acids. Exactly how (and when) this system got its start however, remains one of the most intriguing and hotly contested areas of biology.

According to one hypothesis, the simpler RNA molecule preceded DNA as the original informational conduit. The RNA world hypothesis proposes that the earliest examples of life were based on RNA and simple proteins. Because of RNA's great versatility—it is not only capable of carrying genetic information but also of catalyzing chemical reactions like an enzyme—it is believed by many to have supported pre-cellular life.

Nevertheless, the spontaneous arrival of RNA through a sequence of purely random mixing events of primitive chemicals was at the very least, an unlikely occurrence. "This is a big question," Chaput says. "If the RNA world existed, how did it come into existence? Was it spontaneously produced, or was it the product of something that was even simpler than RNA?"

This pre-RNA world hypothesis has been gaining ground, largely through investigations into XNAs, which provide plausible alternatives to the current biological regime and could have acted as chemical stepping-stones to the eventual emergence of life. The current research strengthens the case that something like this may have taken place.

Threose nucleic acid or TNA for example, is one candidate for this critical intermediary role. "TNA does some interesting things," Chaput says, noting the molecule's capacity to bind with RNA through antiparallel Watson-Crick base pairing. "This property provides a model for how XNAs could have transferred information from the pre-RNA world to the RNA world."

Nucleic acid molecules, including DNA and RNA consist of 3 chemical components: a sugar group, a triphosphate backbone and combinations of the four nucleic acids. By tinkering with these structural elements, researchers can engineer XNA molecules with unique properties. However, in order for any of these exotic molecules to have acted as a precursor to RNA in the pre-biotic epoch, they would need to have been able to transfer and recover their information from RNA. To do this, specialized enzymes, known as polymerases are required.

Nature has made DNA and RNA polymerases, capable of reading, transcribing and reverse transcribing normal nucleic acid sequences. For XNA molecules, however; no naturally occurring polymerases exist. So the group, led by Phil Holliger at the MRC in England, painstakingly evolved synthetic polymerases that could copy DNA into XNA and other polymerases that could copy XNA back into DNA. In the end, polymerases were discovered that transcribe and reverse-transcribe six different genetic systems: HNA, CeNA, LNA, ANA, FANA and TNA. The experiments demonstrated that these unnatural DNA sequences could be rendered into various XNAs when the polymerases were fed the appropriate XNA substrates.

Using these enzymes as tools for molecular evolution, the team evolved the first example of an HNA aptamer through iterative rounds of selection and amplification. Starting from a large pool of DNA sequences, a synthetic polymerase was used to copy the DNA library into HNA. The pool of HNA molecules was then incubated with an arbitrary target. The small fraction of molecules that bound the target were separated from the unbound pool, reverse transcribed back into DNA with a second synthetic enzyme and amplified by PCR. After many repeated rounds, HNAs were generated that bound HIV trans-activating response RNA (TAR) and hen egg lysosome (HEL), which were used as binding targets.) "This is a synthetic Darwinian process," Chaput says. "The same thing happens inside our cells, but this is done in vitro."

The method for producing XNA polymerases draws on the path-breaking work of Holliger, one of the lead authors of the current study. The elegant technique uses cell-like synthetic compartments of water/oil emulsion to conduct directed evolution of enzymes, particularly polymerases. By isolating self-replication reactions from each other, the process greatly improves the accuracy and efficiency of polymerase evolution and replication. "What nobody had really done before," Chaput says, "is to take those technologies and apply them to unnatural nucleic acids."

Chaput also underlines the importance of an international collaboration for carrying out this type of research, particularly for the laborious effort of assembling the triphosphate substrates needed for each of the 6 XNA systems used in the study: "What happened here is that a community of scientists came together and organized around this idea that we could find polymerases that could be used to open up biology to unnatural polymers. It would have been a tour de force for any lab to try to synthesize all the triphosphates, as none of these reagents are commercially available."

The study advances the case for a pre-RNA world, while revealing a new class of XNA aptamers capable of fulfilling myriad useful roles. Although many questions surrounding the origins of life persist, Chaput is optimistic that solutions are coming into view: "Further down the road, through research like this, I think we'll have enough information to begin to put the pieces of the puzzle together."

The research group consisted of investigators from the Medical Research Council (MRC) Laboratory of Molecular Biology, Cambridge, led by Philipp Holliger; the Institute, Katholieke Universiteit Leuven, Belgium, led by Piet Herdewijn; the Nucleic Acid Center, Department of Physics and Chemistry, University of Southern Denmark, led by Jesper Wengel; and the Biodesign Institute at Arizona State University, led by John Chaput.

In addition to his appointment at the Biodesign Institute, John Chaput is an associate professor in the Department of Chemistry and Biochemistry, in the College of Liberal Arts & Sciences.

<http://phys.org/news/2012-04-antibiotics.html>

Pinpointing how antibiotics work

Penicillin and other antibiotics have revolutionized medicine, turning once-deadly diseases into easily treatable ailments. However, while antibiotics have been in use for more than 70 years, the exact mechanism by which they kill bacteria has remained a mystery.

Now a new study by MIT and Boston University researchers reveals the killing mechanism behind all three major classes of antibiotics: The drugs produce destructive molecules that fatally damage bacterial DNA through a long chain of cellular events. Understanding the details of this mechanism could help scientists improve existing drugs, according to the researchers. Few new antibiotics have been developed in the past 40 years, and many strains of bacteria have become resistant to the drugs now available. "One could enhance the killing efficacy of our current arsenal, reduce the required doses or resensitize strains to existing antibiotics," says James Collins, a professor of biomedical engineering at BU, who collaborated with Graham Walker, MIT professor of biology, on a study appearing in the April 20 issue of *Science*.

Lead author of the paper is James Foti, a postdoc in Walker's lab. Other authors are MIT postdoc Babho Devadoss and Jonathan Winkler, a recent PhD recipient in Collins' lab.

Destructive radicals

In 2007, Collins showed that three classes of antibiotics — quinolones, beta-lactams and aminoglycosides — kill cells by producing highly destructive molecules known as hydroxyl radicals. At the time, he and others suspected that the radicals launch a general attack against any cell components they encounter.

"They react with almost everything," Walker says. "They'll go after lipids, they can oxidize proteins, they can oxidize DNA." However, most of this damage is not fatal, the researchers found in the new study.

What proves deadly to bacteria is hydroxyl-induced damage to guanine, one of the four nucleotide bases that constitute DNA. When this damaged guanine is inserted into DNA, cells try to repair the damage but end up hastening their own death. This process "doesn't account for all of the killing, but it accounts for a rather remarkable amount of it," says Walker, who is an American Cancer Society Professor.

Walker's studies of DNA repair enzymes led the researchers to suspect that this damaged guanine, known as oxidized guanine, might play a role in antibiotic-mediated cell death. In the first phase of their research, they showed that a specialized DNA-copying enzyme called DinB — part of a cell's system for responding to DNA damage — is very good at utilizing the oxidized-guanine building block to synthesize DNA.

However, DinB not only inserts oxidized guanine opposite its correct base partner, cytosine, on the complementary strand when DNA is being copied, but also opposite its incorrect partner, adenine. The researchers found that, when too many oxidized guanines had been incorporated into new DNA strands, the cell's unsuccessful efforts to remove these lesions resulted in death.

Based on these very basic DNA-repair studies, Walker and his colleagues hypothesized that the hydroxyl radicals produced by antibiotics might be setting off the same cascade of DNA damage. This turned out to be the case.

Once oxidized guanine caused by antibiotic treatment is inserted into DNA, a cellular system designed to repair DNA kicks into action. Specialized enzymes known as MutY and MutM make snips in the DNA to initiate repair processes that normally help the cells deal with the presence of oxidized guanine in their DNA. However, this repair is risky because it requires opening up the DNA double helix, severing one of its chains while the incorrect base is replaced. If two such repairs are undertaken in close proximity on opposite DNA strands, the DNA suffers a double-strand break, which is usually fatal to the cell. "This system, which normally should be protecting you and keeping you very accurate, becomes your executioner," Walker says.

Deborah Hung, a professor of microbiology and immunobiology at Harvard Medical School, says that the new study represents "the next important chapter as we're going through a renaissance of understanding how antibiotics work. We used to think we knew, and now we've realized that all our simple assumptions were wrong, and it's much more complex," says Hung, who was not part of this study.

New targets

In some cases of antibiotic-induced DNA damage, the bacterial cell is able to save itself by repairing the double-strand break using a process called homologous recombination. Disabling the enzymes required for homologous recombination could increase bacteria's sensitivity to antibiotics, the researchers say.

"Our work would suggest that proteins involved in repairing double-stranded DNA breaks could be very interesting targets to go after as a means to affect the killing efficacy of drugs," Collins says.

The researchers, whose work was funded by the National Institutes of Health and Howard Hughes Medical Institute, also showed that an additional mechanism may be involved in cell deaths caused by one of the antibiotic classes, aminoglycosides: In cells treated with these antibiotics, oxidized guanine is incorporated into messenger RNA, resulting in incorrect proteins that, in turn, trigger more hydroxyl-radical production and hence more oxidized guanine. The researchers are now working to further advance their understanding of how antibiotics kill cells.

<http://phys.org/news/2012-04-scanning-brain-impending-error.html>

Scanning the brain for impending error

UA computer science doctoral student Federico Cirett is using new technology to predict, in advance, when people will make a mistake.

Phys.org - He's been testing subjects taking the SAT exam in math.

Our bodies and brains tend to give us good cues about when we are becoming stressed, fatigued or overwhelmed. But what if, with near exact precision, you could predict when heightened levels of fatigue were about to cause you to make a mistake? University of Arizona doctoral student Federico Cirett believes he's found a way – and with about 80 percent accuracy.

Cirett had been working on the Animal Watch tutoring program with Carole Beal, a professor in the UA's School of Information: Science, Technology and Arts, or SISTA. Noticing English language learners were having more difficulty answering problems, Cirett set out on an investigation for his dissertation work.

"There are so many things going on where students may be getting distracted, but it wasn't clear," said Cirett, a computer science department student working on his dissertation. "So, I thought to measure brain states of students as they were working on the material."

Using electroencephalography, or EEG, technology, Cirett began studying specific brain wave activity in students taking the math portion of the popular, but challenging, SAT exam. Measuring the activity, Cirett was able to detect with 80 percent accuracy whether a student – all of them university students – would answer a question incorrectly about 20 seconds after they began the question.

The findings have important implication for students and educators, said Beal, also Cirett's adviser and collaborator. With the findings, Beal and Cirett co-authored "EEG estimates of engagement and cognitive

workload predict math problem solving outcomes." The paper has since been accepted for presentation during the User Modeling, Adaptation and Personalization conference to be held in Montreal, Canada in July.

"He's done this great project, and his contribution is applying this research to education," said Beal, who has spent years developing AnimalWatch, a Web-based tutoring system centered on algebra readiness.

But how is this done?

During his work on AnimalWatch, Cirett said he noticed that the English language learners – the majority of them who spoke Spanish as their primary language – seemed to be having a more difficult time answering problems than did their primarily English-speaking peers. Cirett found that the students performed at comparable levels on the math problems, but the English learners stumbled a bit. "It was the language barrier."

"We want students to be able to solve these problems," Cirett said, "but we have to make these problems easier for them to read, but we have to give them better opportunities."

Mexico-born Cirett, who came to the UA to study alongside his wife, spoke with Beal about using the EEG technology, which she had employed in the past. For his research, which was partially funded by the National Science Foundation, Cirett employs a headset developed by the San Diego-based Advanced Brain Monitoring Technologies, which is generally used to monitor high-stress and fatigue in military personnel.

"Some of it is pre-programmed," Beal said, noting that the technology and algorithms had already been designed. "But what Federico did was to look at these patterns to create a classification," Beal said. "His algorithm is much better than chance and it's much better than knowing the number of correct answers people typically get on the exam."

With its nine sensors, the device records information about individual attention levels and cognitive workload as they completed multiple-choice math questions, some easy; some hard. For instance, the measurements were directly correlated to how engaged students were in their work and how they felt in the process.

During the study, students also reported on feelings of frustration and their perceived difficulty of the math problems. Cirett analyzed the data to try and figure out if the EEG data specific to students' attention levels and their cognitive workload could predict when they would answer a question with the correct answer.

Though his research involved college students, Cirett intends for his work to inform efforts to improve tutoring programs, especially for English language learners. Cirett and Beal point to developments over the last decade in intelligent tutoring systems, those modeled after human behavior, which have resulted in more adaptive systems.

But what Cirett wants to see are intelligent educational technologies that would intervene at important moments in students' learning, aiding them in ways an educator might not. The end goal, he said, is to optimize learning at the individual level, especially in the area of math, an increasingly important subject.

"There are different ways to solving this problem," Cirett said. "But if we can detect when they are going to fail, maybe we can change the text or switch the question to give them another one at a different level of difficulty, but also to keep them engaged," Cirett said. "Brain wave data is the nearest thing we have to really know when the students are having problems."

Provided by University of Arizona

<http://news.discovery.com/human/airplane-ufo-or-venus-120420.html>

Airplane, UFO or Venus?

On Jan. 14 of last year, an Air Canada pilot flying from Toronto to Zurich, Switzerland, woke up from a nap to see an alarming sight out the cockpit window: what appeared to be a flying object (presumably another plane) flying directly at him.

Analysis by Benjamin Radford

He immediately took evasive action, sending the jet into a steep, sudden dive that injured 16 people and almost resulted in a midair collision with another aircraft flying 1,000 feet lower.

It was a terrifying, bizarre event over the Atlantic Ocean, but what makes it even stranger is that, according to a new report from the Transportation Safety Board of Canada, the pilot was reacting to an optical illusion. The pilot thought it was a UFO -- quite literally, an unidentified object flying at the plane. Yet there was no aircraft, identified or otherwise: The pilot had instead seen reflected sunlight from the planet Venus.

Depending on when you measure it (since everything in the universe is in constant motion), Venus is between about 25 million and 162 million miles away. Yet the pilot thought that it was close enough to pose an imminent threat of collision. How could the pilot's estimate of the light's distance to the plane be off by at least 25 million miles? How could an experienced airline pilot mistake a planet for a plane?

It's actually not that difficult to understand and has implications for other UFO sightings. As this incident shows, accurately judging the size, speed and distance of unknown lights in the night sky is virtually impossible.

A light in the sky might be small and 100 yards away, medium-sized and a few miles away, or even planet-sized and tens of millions of miles away -- and there is no way to know the difference. John Nance, a former commercial pilot and ABC News aviation analyst, said that such a mistake, while seemingly inexplicable to the average person, was "not outlandish ... a bright light, which can be a planet like Venus, can be very startling, and you can mistake it for an airplane."

Venus and UFOs

The strange fact is that Venus has been responsible for many UFO sightings over the years. This skeptical explanation causes discomfort for many UFO believers, who claim that eyewitness UFO reports by pilots are the most reliable in the world. After all, they claim, experienced pilots are familiar with normal lights in the night sky -- surely no pilot could possibly mistake a planet for a nearby flying object!

Robert Sheaffer, a columnist for Skeptical Inquirer magazine and veteran UFO investigator, told Discovery News that "there is a long history of Venus (or some other bright planet or star) being perceived as something that it isn't. Even the UFO proponent Jacques Vallee wrote back in 1966 that "no single object has been misinterpreted as a 'flying saucer' more often than the planet Venus."

In fact, Sheaffer noted, "During World War II, B-29 crews making night bombing raids in Japan reported being followed by a 'ball of fire' that turned out to be Venus. Since then, numerous police officers and pilots have made the same mistake, as did Jimmy Carter, who reported seeing a UFO back in 1969 that turned out to be in exactly the same place as Venus."

This case came to light primarily because the Air Canada pilot's reaction caused injuries to the passengers (several were hospitalized). While it's impossible to know exactly how often pilots and others mistake Venus (or other ordinary lights in the night sky) for UFOs, it's likely to be underestimated and underreported.

Pilots may be reluctant or embarrassed to admit they mistook a planet for a plane and not report the error (in fact Air Canada initially claimed that the incident was the result of "severe turbulence").

Though this size/distance miscalculation effect is strongest at night, the phenomenon also occurs in daylight when unknown objects are sighted (or recorded) in the sky at an unknown distance from the camera. Whether night or day, without some way of establishing the scale or distance of a flying object, it is impossible to determine its size or speed accurately. (In one recently released UFO video taken at an Air Force base in Chile, it seems that insects -- probably bees -- were captured in the foreground on videocameras and mistaken for high-speed extraterrestrial spacecraft in the skies.)

Of course Venus does not explain all UFO sightings. But this case proves that even experienced pilots can (and do) mistake our neighboring planet for unexplained lights in the sky.

<http://www.sciencedaily.com/releases/2012/04/120420105330.htm>

Body Cooling Cuts In-Hospital Cardiac Arrest Patient Deaths Nearly 12 Percent *Therapeutic hypothermia has reduced in-hospital deaths among sudden cardiac arrest patients nearly 12 percent between 2001 and 2009*

ScienceDaily - Forced body cooling known as therapeutic hypothermia has reduced in-hospital deaths among sudden cardiac arrest patients nearly 12 percent between 2001 and 2009, according to a Mayo Clinic study being presented at the upcoming American Academy of Neurology 2012 Annual Meeting in New Orleans.

The goal of therapeutic cooling is slowing the body's metabolism and preventing brain damage or death. It is believed that mild therapeutic hypothermia suppresses harmful chemical reactions in the brain and preserves cells. Two key studies published in 2002 found therapeutic hypothermia more effective for sudden cardiac arrest patients than traditional therapies.

Mayo researchers analyzed a database covering more than 1 million patients and found mortality rates among in-hospital sudden cardiac arrest patients dropped from 69.6 percent in 2001 -- the year before the studies appeared -- to 57.8 percent in 2009, the most recent data available.

"Because we reviewed such a large number of cases, we are confident that the reduction in mortality among in-hospital sudden cardiac arrest patients is significant and sustained," says co-author Alejandro Rabinstein, M.D., a Mayo Clinic neurologist.

"We continue to seek answers to the questions: Why did this trend develop, and how can we accelerate it," says co-author Jennifer Fugate, D.O.

These measures are important because disease accumulates in the cortex over time, and inflammation in the cortex is a sign the disease has progressed.

Making a Home on Plesiosaurs

A dead whale is more than a rotting mass of flesh and bone. In the deep sea, the descent of a leviathan is a fortuitous bonanza for the battalions of scavengers which gradually break down the cetacean's body.

By Brian Switek

And, for as long as the body lasts, the whale becomes a little island of diversity in the deep – a temporary deconstruction site where the remains of one life are distributed among the many.

But whales may only be the latest creatures to so enrich the sea floor. Long before whales, the carcasses of marine crocodiles, sea turtles, mosasaurs, ichthyosaurs, and plesiosaurs settled on the bottom. (Even the occasional dinosaur carcass floated out into deep water.) Unfortunately, we can't study the decomposition of these Mesozoic creatures in real time, but the circumstances of preservation have created snapshots of marine communities which briefly sprung up around the bodies of these animals. For the most part, the associated fossils include bivalves and shark teeth, but at least two plesiosaur skeletons appear to have been colonized by a few archaic forerunners of today's whalefall communities.

Paleontologist Andrzej Kaim and co-authors described the specimens in 2008. Each had been discovered in slightly different Late Cretaceous deposits near Hokkaido, Japan. One, dubbed the Turonian skeleton, was about 93 to 89 million years old, and the other – called the Conician skeleton – was about 89 to 85 million years old. And despite the fact that these skeletons were found at different sites and were not of the same species, both were found associated with numerous gastropod fossils.

By itself, the association between reptile and mollusk isn't remarkable. Fossils of gastropods, as well as bivalves, are often found associated with skeletons of marine reptiles. But the identity of the invertebrates was a clue that ephemeral ecosystems had developed on the plesiosaur carcasses.

The small snails collected from the rock between and near the plesiosaur bones were provannids – a group of gastropods which are still around today, and are found in relatively short-lived habitats such as hydrothermal vents, cold seeps, and whalefalls. Indeed, Kaim and colleagues pointed out that similar snails also turned up at sites of Cretaceous cold seeps – places where methane and other chemicals oozed out into pools and nourished chemosynthetic communities of bacteria which formed the basis for islands of life on the seafloor. The snails on the skeletons were specialists at colonizing short-lived sites of localized resources.



The skull of a large plesiosaur. Around 90 million years ago, bacteria and snails colonized a creature similar to this one. Photographed at the Wyoming Dinosaur Center by the author.

Snails weren't the only organisms to take up in the plesiosaur carcasses. Some of the plesiosaur bones were shot through with tiny boreholes similar to those found on decomposing whale skeletons. Exactly what creature or process creates this pattern of damage is unclear, but the tiny tubes may have been left behind by bacteria. This may also explain why the snails were present on the carcasses. As mats of bacteria spread over the picked-over plesiosaur skeletons, the snails grazed on the proliferation of bacteria – a situation similar to the final “sulfophilic stage” of modern deadfalls.

What remains unclear is whether the plesiosaur skeletons represent the true beginnings of dedicated deadfall communities, or whether the bacteria and snails were just opportunists. The plesiosaur carcasses settled in an area pocked by prehistoric methane seeps known to host such snails, and, hence, the bacteria on which the snails fed. Perhaps the creatures from a nearby seep simply took advantage of an easy meal, and distinct deadfall communities did not originate until the origin of entirely aquatic whales, around 45 million years ago. But, even if this is the case, the connection between the plesiosaur skeletons, colonizers, and methane seeps hints that bodies which fell in the vicinity of methane seeps or hydrothermal vents might have provided a stepping stone for deadfall communities to develop.

As Kaim and colleagues pointed out, we don't have enough information to know whether modern whale falls are extensions of plesiosaur, ichthyosaur, and mosasaur falls of the past. No one even expected that such communities might exist until they were discovered in the modern seas. Now that paleontologists know what to look for, though, additional evidence of deep sea decay will hopefully be dredged from the rock.

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Seeking HIV treatment clues in the neem tree

Preliminary data hint at how extracts from the tree, abundant in tropical and subtropical areas, may stop the virus from multiplying

Tall, with dark-green pointy leaves, the neem tree of India is known as the "village pharmacy." As a child growing up in metropolitan New Delhi, Sonia Arora recalls on visits to rural areas seeing villagers using neem bark to clean their teeth. Arora's childhood memories have developed into a scientific fascination with natural products and their power to cure illnesses.

Now an assistant professor at Kean University in New Jersey, Arora is delving into understanding the curative properties of the neem tree in fighting the virus that causes AIDS. She will be presenting her data at a poster session at 12:25 p.m. Sunday, April 22, at the Experimental Biology 2012 meeting in San Diego. Her preliminary results seem to indicate that there are compounds in neem extracts that target a protein essential for HIV to replicate. If further studies support her findings, Arora's work may give clinicians and drug developers a new HIV-AIDS therapy to pursue.

Extracts from neem leaves, bark and flowers are used throughout the Indian subcontinent to fight against pathogenic bacteria and fungi. "The farther you go into the villages of India, the more uses of neem you see," says Arora. Tree branches are used instead of toothpaste and toothbrushes to keep teeth and gums healthy, and neem extracts are used to control the spread of malaria.

Practitioners of Ayurvedic medicine, a form of traditional Indian alternative medicine, even prescribe neem extracts, in combination with other herbs, to treat cardiovascular diseases and control diabetes. The neem tree, whose species name is *Azadirachta indica* and which belongs to the mahogany family, also grows in east Africa.

Arora's scientific training gave her expertise in the cellular biology of cancer, pharmacology, bioinformatics and structural biology. When she established her laboratory with a new research direction at Kean University in 2008, Arora decided to combine her knowledge with her long-time fascination with natural products. The neem tree beckoned.

Arora dived into the scientific literature to see what was known about neem extracts. During the course of her reading, Arora stumbled across two reports that showed that when HIV-AIDS patients in Nigeria and India were given neem extracts, the amount of HIV particles in their blood dropped. Intrigued, Arora decided to see if she could figure out what was in the neem extract that seemed to fight off the virus.

She turned to bioinformatics and structural biology to see what insights could be gleaned from making computer models of HIV proteins with compounds known to be in neem extracts. From the literature, she and her students found 20 compounds present in various types of neem extracts. When they modeled these compounds against the proteins critical for the HIV life-cycle, Arora and her team discovered that most of the neem compounds attacked the HIV protease, a protein essential for making new copies of the virus.

Arora's group is now working on test-tube experiments to see if the computer models hold up with actual samples. If her work bears out, Arora is hopeful that the neem tree will give a cheaper and more accessible way to fight the HIV-AIDS epidemic in developing countries, where current therapies are priced at levels out of reach of many people. "And, of course," she notes, "there is the potential of discovering new drugs based on the molecules present in neem."

<http://www.bbc.co.uk/news/magazine-17774210>

Are North Koreans really three inches shorter than South Koreans?

It's often been reported that North Koreans are a few inches shorter than their counterparts south of the border. Is that true?

By Richard Knight BBC News

North Korea's recent failure to launch a long-range rocket was embarrassing for its new leader, Kim Jong-un. It was supposed to be a symbol of progress.

Renewed media interest in North Korea since Kim Jong-un replaced his father has prompted the re-emergence of a claim which appears to be a symbol not of progress, but of relative decline: that North Koreans are much shorter than South Koreans.

The Independent reported last week that "nothing is small in North Korea apart from the people, who are on average three inches shorter than their cousins in the South".

This statistic, or versions of it, have been quoted for some time. In 2010 the late Christopher Hitchens put the difference at six inches in an article in Slate titled "A Nation of Racist Dwarfs".

Senator John McCain referred to a three-inch gap in a 2008 presidential debate.

So what's the truth? Professor Daniel Schwekendiek from Sungkyunkwan University in Seoul has studied the heights of North Korean refugees measured when they crossed the border into South Korea.

He says North Korean men are, on average, between 3 - 8cm (1.2 - 3.1in) shorter than their South Korean counterparts.

A difference is also obvious between North and South Korean children.

"The height gap is approximately 4cm (1.6in) among pre-school boys and 3cm (1.2in) among pre-school girls, and again the South Koreans would be taller."

Schwekendiek points out that the height difference cannot be attributed to genetics, because the two populations are the same.

"We're dealing with the Korean people," he says, "and Korea is interesting because it basically hasn't experienced any immigration for many centuries."

Martin Bloem is head of nutrition at the World Food Programme, which has been providing food aid to North Korea since 1995. He says poor diet in the early years of life leads to stunted growth.

"Food and what happens in the first two years of life is actually critical for people's height later," he says.

In the 1990s North Korea suffered a terrible famine. Today, according to the World Food Programme, "one in every three children remains chronically malnourished or 'stunted', meaning they are too short for their age".

South Korea, in contrast, has experienced rapid economic growth. Bloem says "economic growth is one of the main determinants of height improvement".

So while North Koreans have been getting shorter, South Koreans have been getting taller.

"If you look at older Koreans," says Schwekendiek, "we now see a situation where the average South Korean woman is approaching the height of the average North Korean man.

"This is to my knowledge a unique situation, where women become taller than men."

The secretive nature of North Korea makes it difficult to find reliable data for analysis.

Schwekendiek has studied refugees, but he rejects the notion that people driven to cross the border to South Korea are the most disadvantaged and therefore most likely to be stunted.

The refugees, he says, "come from all social strata and from all regions".

He has also studied data collected by the North Korean government and by international organisations working in North Korea, which he says support his findings.

It seems that this height statistic reveals a tragic fact - that as South Koreans have got richer and taller, North Korean children are being stunted by malnourishment.