http://www.eurekalert.org/pub_releases/2011-01/s-hco010311.php

Hair color of unknown offenders is no longer a secret

Increasing number of appearance traits extractable from DNA

The hair color of an unknown perpetrator who has committed a crime will soon no longer be a secret for forensic investigators. Erasmus MC scientists, in collaboration with their Polish colleagues, have discovered that DNA can be used to predict people's probable hair color. Their findings[1] will be published today in the Springer journal Human Genetics.

The research findings demonstrate that on the basis of DNA information it is possible to determine with an accuracy of more than 90 percent whether a person has red hair, with a similarly high accuracy whether a person has black hair, and with an accuracy of more than 80 percent whether a person's hair color is blond or brown. This new DNA approach even allows differentiating hair colors that are similar, for example, between red and reddish blond, or between blond and dark blond hair. The necessary DNA can be taken from blood, sperm, saliva or other biological materials relevant in forensic case work.

Prof. Manfred Kayser, Chair of the Department of Forensic Molecular Biology at Erasmus MC, who led the study, said, "That we are now making it possible to predict different hair colors from DNA represents a major breakthrough because, so far, only red hair color, which is rare, could be estimated from DNA. For our research we made use of the DNA and hair color information of hundreds of Europeans and investigated genes previously known to influence the differences in hair color. We identified 13 'DNA markers' from 11 genes that are informative to predict a person's hair color."

Prof. Ate Kloosterman of the Department of Human Biological Traces at the Netherlands Forensic Institute (NFI) said: "This research lays the scientific basis for the development of a DNA test for hair color prediction. A validated DNA test system for hair color shall become available for forensic research in the not too distant future. These researchers have previously published articles on predicting eye color and estimating age on the basis of DNA material. This type of objective information can be used to refine the description of an unknown but wanted person. This new development results in an important expansion of the future DNA toolkit used by forensic investigators to track down unknown offenders."

The current study was directed at the predictability of the color of the hair on the head. Further research would be necessary to predict the color of body hair.

References

1. Branicki W, Kayser M et al. (2011). Model-based prediction of human hair color using DNA variants. Human Genetics; DOI 10.1007/s00439-010-0939-8

2. The Erasmus MC research was made possible by subsidies granted by the Netherlands Forensic Institute (NFI) and the Netherlands Genomics Initiative (NGI) / Netherlands Organization for Scientific Research (NWO) in the framework of the Forensic Genomic Consortium Netherlands (FGCN).

http://www.bbc.co.uk/news/science-environment-11979934

Sabretooth cats threatened most ancient human ancestor

By Paul Rincon Science reporter, BBC News

Humankind's oldest known ancestor probably lived in fear of several large sabretooth cats that roamed the same ancient lakeside habitat in Africa.

Palaeontologists have identified two new sabretooth species among fossils unearthed at Toros Menalla in Chad.

In 2001, a team unearthed remains of a seven million-year-old human-like creature - or hominid - known as "Toumai" at the central African site. Its discoverers argue that Toumai is the oldest hominid known to science.

The fossilised skull of Toumai (which means "hope of life" in the local Dazaga language of Chad) was found in the Djurab desert by a team led by Michael Brunet of the University of Poitiers, France.

The position of a hole at the bottom of the skull called the foramen magnum suggests that Toumai (Sahelanthropus tchadensis) walked upright - an important signature of the human lineage.

The brainstem enters and exits the skull through this hole; in great apes, it is positioned more towards the back of the skull. But in hominids - including Toumai - it is placed more towards the front of the skull.

Sahelanthropus may have inhabited the gallery forest where the trees offered some protection

The ancient fossil caused a worldwide sensation when it was unveiled in the pages of Nature journal in 2002.

However, the interpretation of Toumai as a human relative is controversial. The skull was distorted and, if any other parts of the skeleton happen to exist, none has yet been published in the scientific literature. It is also older than the date when genetics says that the human and chimp lineages diverged.

Predator's playground

Nevertheless, palaeontologists have been busy studying the abundant fossil material unearthed at the site, steadily building a picture of the environment in which Sahelanthropus eked out its existence.

In Late Miocene times, this area of Chad must have had a lake, because palaeontologists have found the fossilised remains of fish, amphibians and crocodiles. But they have also found evidence of grasslands, gallery forest and a desert. Researchers have discovered the fossilised remains of a wide variety of carnivorous mammals at Toros Menalla. Ending up in the sharp jaws of a predator must have been an ever-present threat for primates like Toumai. Palaeontologists had already reported finding remains of a large sabretooth cat from Toros Menalla known as Machairodus kabir which weighed in at 350-490kg.

Writing in the journal Comptes Rendus Palevol, Louis de Bonis from Poitiers University and colleagues add two new sabretooth species to the growing list of carnivores that stalked this region of central Africa in late Miocene times.

The big cat remains were unearthed during recent field expeditions and have been identified as new species belonging to the genus Lokotunjailurus and the genus Megantereon.

Forest refuge

Patrick Vignaud, director of Poitier University's Institute of Palaeo-primatology and Human Palaeontology, told BBC News the cats were about the same size as modern lions.

"With our present data, we don't know what precisely the interactions were between a primate and a big carnivore. But probably these interactions were not so friendly," said Professor Vignaud.

He told BBC News: "Sabretooths hunted all mammals; bovids, equids... and primates. The interactions were also more 'psychological', exercising a stress on potential prey. We can't prove it but it's probably important because in that case, primates had to live near closed environments like gallery forest."

While ancient primates like Sahelanthropus tchadensis gave sabretooth cats a wide berth, they may also have depended on these big carnivores - and others - for their survival.

Sabretooths would have hunted large herbivorous mammals, and probably left enough meat on their kills for scavengers like the jackal-sized Hyaenictherium and perhaps even primates like Sahelanthropus.

Some researchers have proposed that Toumai is more closely related to chimpanzees or gorillas. Even if this were the case, the discovery would be of great significance, as virtually no fossil ancestors of these great apes are known from Africa.

http://www.eurekalert.org/pub_releases/2011-01/uoc-att010311.php

A toast to history: 500 years of wine-drinking cups mark social shifts in ancient Greece

How commonly used items – like wine drinking cups – change through time can tell us a lot about those times, according to University of Cincinnati research to be presented Jan. 7 by Kathleen Lynch, UC associate professor of classics, at the annual meeting of the Archaeological Institute of America.

Lynch will present the research at the event's Gold Medal Session, when archaeology's most distinguished honor will be bestowed on her mentor, Susan Rotroff of Washington University.

UC's Lynch will present a timeline of wine drinking cups used in ancient Athens from 800 B.C. to 323 B.C. and will discuss how changes to the drinking cups marked political, social and economic shifts.

BACKGROUND

Lynch's specific area of study, which will result in a forthcoming book, is what's known as the "symposium" in ancient Athens. These were gatherings held for nearly a millennia where communal drinking of wine was a means for cementing cultural norms and social bonds that carried over into the world of politics and business.

Think of these symposia as the ancient world's ultimate cocktail parties, with established rituals and rules. An important aspect of any symposium was the wine cup, and the form of and the imagery on the cups reflected the shared culture of participants, as well as the larger social realities and changes in their world during the following periods:

* Iron Age (1,100-700 B.C.)
* The Archaic Period (700-480 B.C.)
* The Late Archaic Period (525-480 B.C.)
* The High Classical Period (480-400 B.C.)
* The Late Classical Period (400-323 B.C.)
* The Hellenistic Period (323-31 B.C)

Basic rules of Athenian symposia:

* Couches or mattresses used by reclining participants were set in a circle or square. So, there was no formal position of status or group "head."

* Drinkers imbibed in rounds, so consumption of wine (mixed with water) was equitable. In other words, everyone got drunk at about the same rate. No teetotalers permitted.

* Said Lynch, "The focus was on drinking communally and in equal amounts. Inhibitions were lost. In-group bonds were formed. "

Why study these items? "Because," stated Lynch, "People's things tell you about those people and their times. In the same way that the coffee mug with 'World's Greatest Golfer' in your kitchen cabinet speaks to your values and your culture, so too do the commonly used objects of the past tell us about that past. And, often, by studying the past, we learn about ourselves."

IRON AGE SYMPOSIA AND DRINKING CUPS (1,100-700 B.C.)

* The drinking gatherings (symposia) were reserved for the elite, probably allowing political factions to consolidate power and set themselves apart from the population at large. In other words, the drinking gatherings were for the "in" crowd.

* At this time, even grave markers for the very wealthy came in the form of the mixing bowls (kraters) used to blend wine with water during symposia. In other words, the ability to sponsor these drinking events was what people wanted to be remembered for.

* The drinking cups during this period were simply decorated and rested directly on a base (no stem).

This three-foot-high Iron Age gravemarker is in the form of a mixing vessel (water and wine) used at symposia. It signals the importance of the symposia in Athenian society. People wanted to be remembered for their ability to sponsor these gatherings. **Credit: The Metropolitan Museum of Art website: metmuseum.org/toah/works-of-art/14.130.14**

THE ARCHAIC PERIOD (700-480 B.C.)

* After the turn of the 6th century B.C., changes in the fashion of drinking cups began, corresponding with Athens' rising political power and rising dominance in the ceramic market. Variety and quality were high during this period. It was the beginning of black-figured pottery production as well as plain, black-glazed versions. Stemmed cups became more popular, probably because they were easier to hold while reclining.

* The middle of the 6th century B.C. saw a rapid proliferation of cup types: Komast cups, Siana cups, Gordion cups, Lip cups, Band cups, Droop cups, Merry-thought cups and Cassel cups – last only a few decades in terms of popularity. Some of these remain popular for only a few decades.

* Explained Lynch, "Possessing what was newest in terms of mode and style of drinking cups was likely equated with knowledge and status. The elites may have been seeking cohesion and self definition in the face of factional rivalries and populist movements. This hypothesis underscores how the drinking symposia – and specific cup forms identified with specific factions – might have been used by aristocratic blocs to cement group bonds in the politically charged environment of the time."

LATE ARCHAIC PERIOD (525-480 B.C.)

* The overall number of wine-drinking vessels increased dramatically during this period, pointing to the democratization of the symposium, as well as the democratization of the political and social arenas. The masses had become the political, if not the social, equals of the elites, and these masses were now enjoying symposia of their own.

* It's estimated that drinking vessels for symposia comprised up to 60 percent of the terra cotta fineware (collection of dishes) in the typical Athenian home of this period. "The typical home had few useful dishes for eating in contrast to many vessels designed for drinking wine in communal settings," explained Lynch.

* This period ends with the devastating Persian Wars, which Greece won. The proliferation of cup types fell, with red-figured drinking cups, introduced around 525 B.C., becoming the most popular.

HIGH CLASSICAL PERIOD (480-400 B.C.)

* Red-figured cups (cups decorated with red figures vs. black) remain popular through the first part of this period of cultural development in Athens, but the cups grow taller and shallower.

* By the end of the 5th century B.C., Athens was weathering the Peloponnesian Wars and plague, and people were searching for an escape. This came in the form of an aesthetic restlessness. Fads in drinking cups came and went, but few developed into long-lived styles.

* These new cup innovations tended to emulate the fineness commonly found in silver work at the time. For instance, there were many more plain, black clay cups with shiny surfaces. And delicate stamped and incised designs in clay cup interiors imitated metal prototypes on the cheap. In other words, the common terra cotta cups were "designer knock-offs" of the "high-

* Stemmed cups had finally run their course, being 200 years old at this point, and a stemless form became more popular.

* Said Lynch, "People may have been seeking a visual antidote to the struggles of the period and a yearning for luxury at odds with daily conditions."

LATE CLASSICAL PERIOD (400-323 B.C.)

* Trends toward pseudo luxury (designer knock-offs) in drinking cups continued; however, the variety of these "silver-inspired" clay cup designs diminished after the turn of the 4th century B.C., probably because the forms were impractical. For instance, one clay cup – modeled on a silver drinking vessel – featured delicate high-swung handles that served no useful purpose in clay.

* Also "running out of steam" in this period was the tradition of decorating cups with human figures. A decorative innovation, called West Slope, became popular at this time. It consisted of colored clay applied atop black-glazed surfaces to create the effects of garlands and wreaths. Human figures were no longer depicted.

* Finally, as Athens fell under the sway of Philip of Macedon and his son, Alexander the Great, the symposium came full circle. It began in the Iron Age as a practice of the elite. Then, with the movement toward democratization in Athens, participation in symposia broadened. Now, in Athens' Hellenistic period, the practice was again the prerogative of the elites as a luxury and display of ostentatious consumption. Equality was no longer important in a state that was no longer democratic but monarchical.

Lynch's research on symposia of ancient Greece received funding from the Louise Taft Semple Fund of the Department of Classics at UC; the Samuel H. Kress Foundation; and the Sheldon H. Solow Foundation, Inc.

http://www.physorg.com/news/2011-01-infant-hydrocephalus-seasonal-linked-farm.html

Infant hydrocephalus, seasonal and linked to farm animals in Uganda

Hydrocephalus in Ugandan children and other developing countries is seasonal, linked to farm animals and in part, caused by previous bacterial infection, according to an international team of researchers from Uganda and the United States, who believe that the best approach to this problem is prevention.

"Hydrocephalus in infants in developing countries is a grand medical mystery," said Steven Schiff, the Brush Chair professor of engineering and director, Penn State Center for Neural Engineering.

Hydrocephalus is a build up of the fluid that normally surrounds the brain. The increased pressure causes the head to swell and damages brain tissue. Treatment includes placing a shunt to drain the fluid, but inevitably these shunts become plugged and require emergency care, not always available in rural Africa and other resource-limited regions of the developing world. Surgeons vigorously explore the use of new brain endoscopes to divert fluid buildup internally in such children, but this approach addresses the fluid and does not fix previous infection damage to the brain.

"Brains of children with hydrocephalus can be completely or mostly destroyed either by the scarring from the disease or by the pressure of the cerebrospinal fluid that cannot escape," said Schiff. "Many of these children with the worst aftereffects of infection will be mentally deficient and survive only as long as their mothers can adequately care for them. Understanding the causes could eliminate or prevent the enormous costs to lives and families that hydrocephalus brings."

Hydrocephalus in infants in sub-Saharan Africa is thought to be caused most often by meningitis-type infections during the first month of life. The U.S. and Ugandan researchers looked at the fluid from the brains of three sets of 25 consecutive infant hydrocephalus patients during January, July and October to try to determine the cause of the disease. By the time parents bring infants with rapidly growing heads to the CURE Children's Hospital in Mbale, Uganda, the underlying infection is gone. The researchers were unable to culture any bacteria from the samples.

To identify traces of previous bacterial infection, the researchers used DNA sequencing to look for 16S ribosomal DNA that exists in all bacteria. They reported their findings in the current issue of the Journal of Neurosurgery: Pediatrics, showing that 94 percent of the samples contained bacterial remnants. The researchers found a seasonal difference between samples representing infection during the dry season that were predominantly Betaproteobacteria and Gammaproteobacteria, that resulted from rainy season infection. Acinetobacter appeared in the majority of patients following rainy season infection.

Some sequences that appeared in the DNA analysis were from unknown bacteria and in many cases the bacterial fragments were not identifiable as to the type of Acinetobacter they represented.

In the United States and other industrialized countries, infant hydrocephalus is usually due to either a congenital anomaly or, in low birthweight premature infants, due to brain hemorrhages from immature blood vessels. At one time, Group B Streptococcus was a common cause of postinfectious hydrocephalus in infants in industrialized countries, but now physicians test mothers for the infection and treat with antibiotics before they give birth and the infections are rare. Surprisingly, according to Schiff, in Uganda, none of the remnant DNA in the infants was from Group B Streptococcus.

Looking for the source of the neonatal infections, the researchers targeted the living environment from infants with evidence of prior acinetobacter infection and located patients' homes. What they found were villages of huts where cow dung was pounded into the hut floors to keep water and ants out and used in patios around the huts where vegetation is cleared to protect against snakes. Newborns enter an environment where they not only live near animals, but also are surrounded by their material.

The researchers sampled both the cow dung floors and excrement from cattle, goats and chickens. They found similar genetic sequences from the bacteria retrieved from the infants as in the hut floors and nearby dung.

"It is really hard to keep infants to an adequate standard of cleanliness in this environment," said Schiff. "The bacteria we found reflects, I think, a significant environmental influence."

While the researchers have not yet proven that these bacterial infections are the cause of the devastating hydrocephalus occurrences, they believe that in part, bacterial infections from animals are the cause.

Historically, certain East African peoples have applied cow dung to stem bleeding in umbilical cord stumps, which caused newborn infections. Although such infections are now rare, the scope of newborn bacterial infections related to living in close proximity to domestic animals remains poorly categorized.

"As far as we can tell, these types of environmental newborn infections are the dominant cause of hydrocephalus on the planet," said Schiff. "We may be dealing with bacteria that we can't culture, viruses or parasites, and we may be dealing with different organisms in different locations"

The researchers are continuing their work and forming an African Hydrocephalus Consortium with Rwanda, Kenya, Tanzania and Zambia. They are conducting follow-up clinical trials at the Mbarara University of Science and Technology in southwest Uganda on mother-infant pairs with new neonatal infections, and at the CURE Children's Hospital of Uganda on older infants with postinfectious hydrocephalus. These trials use next generation technologies and high quality microbiology to sort out the causative agents affecting these infants. They are also continuing to explore the environmental connection so that public health strategies toward preventing the initial infections might be found. *Provided by Pennsylvania State University*

http://www.eurekalert.org/pub_releases/2011-01/ccon-pbc010311.php

Parallels between cancers, infection suppression reported

Same proteins involved, but cancer takes hold when response gets out of control, CCNY biologists report

Tiny parasitoid wasps can play an important role in controlling the populations of other insect species by laying their eggs inside the larvae of these species. A newly hatched wasp gradually eats the host alive and takes over its body.

The host insect is far from defenseless, however. In Drosophila (fruit flies), larvae activate humoral immunity in the fat body and mount a robust cellular response that encapsulates and chokes off the wasp egg.

New research by Dr. Shubha Govind, professor of biology at The City College of New York, and colleagues reveals parallels between how this mechanism fights the wasp infection and the way blood cancer develops. "There are fundamental similarities in the processes," she explains. "The response to wasp infection is similar to acute inflammation while the cancer is akin to chronic inflammation in mammals, where regulation of the response to an infection also goes out of control."

Professor Govind reports that the immune system that counters wasp egg infection is highly restrained. The system works like a thermostat, with certain proteins detecting the infection and triggering the immune reactions. Once the egg has been destroyed the immune reactions come to a halt.

However, when the regulating mechanism goes haywire, cancer can develop. Through sumoylation, the correct balance between positive and negative factors is achieved, Professor Govind and colleagues report.

"There is strong evidence that the fundamental mechanism of regulation uncovered in flies also works in humans," she notes. "Because of the molecular similarities between flies and mammals, it may be possible to use flies to test drugs for potential anti-inflammatory effects in human disease." While such drugs would not cure cancer, they could control inflammation and, perhaps, delay cancer progression.

Other potential applications are in pest control for agriculture. Instead of using insecticides, parasitoids with the ability to suppress the hosts' immune systems could be used to kill insect pests. Also, insecticides could be

developed that, at very low concentrations, would weaken the immune systems of host insects and enable parasitoid eggs to succeed, Professor Govind adds.

The findings were published last month in PLoS Pathogens, a peer-reviewed, open-access journal published by the Public Library of Science. Contributing scientists were: Indira Paddibhatla, Mark J. Lee, Marta E. Kalamarz and Roberto Ferrarese. The work was funded by the National Institute of General Medicine, U.S. Department of Agriculture and PSC-CUNY.

http://www.eurekalert.org/pub_releases/2011-01/kki-nrr010311.php

New research reveals unexpected biological pathway in glaucoma

Study is first to pinpoint the precise anatomical location where vision loss appears to occur in glaucoma

Baltimore, MD - In a study published today in the Proceedings of the National Academy of Sciences (Early Edition ahead of print), a team of researchers from the Kennedy Krieger Institute and four collaborating institutions, identified a new and unexpected biological pathway that appears to contribute to the development of glaucoma and its resulting vision loss.

Prior research has suggested that the optic nerve head, the point where the cables that carry information from the eye to the brain first exit the eye, plays a role in glaucoma. In this study, researchers report a series of findings that offer novel insights into cellular and molecular mechanisms operating at the optic nerve head in two mouse models of glaucoma. Most notably, they discovered that at a specific location within the optic nerve head, there is a unique class of cells called astrocytes that demonstrate properties that appear to make them a critical factor in the visual blinding that occurs in glaucoma.

Further, at this same site, researchers found abnormal forms of a protein called gamma synuclein that is similar to abnormal forms of alpha synuclein, a related protein known for its key role in cell loss in Parkinson's disease. The findings suggest that a biological process similar to Parkinson's disease unfolds in glaucoma at the specific anatomical location pinpointed in this study for the first time.

Finally, researchers discovered that at this anatomical location, there is a surprising process whereby astrocytes remove the debris of neurons, the cells that die in neurodegenerative disorders such as glaucoma. It is likely that this newly discovered process involving removal of the debris of one cell by a neighboring cell is important not only in glaucoma and Parkinson's disease, but also for many neurodegenerative diseases.

"These findings are very exciting because they give us several novel targets for future interventions," said Dr. Nicholas Marsh-Armstrong, senior study author and a research scientist at Kennedy Krieger Institute. "I believe these findings put us on the cusp of discovering a treatment for glaucoma that may also have relevance for a number of other neurodegenerative diseases."

Future studies will examine this novel pathway and molecular/cellular mechanism to understand precisely what steps go awry in glaucoma and what can be controlled pharmacologically to identify interventions that slow the disease progression.

Dr. Marsh-Armstrong and other scientists at Kennedy Krieger Institute collaborated on this study with colleagues at the Johns Hopkins University School of Medicine, University of California at San Diego, Cardiff University in England, and the University of Murcia in Spain.

This research was principally supported by the Melza M. and Frank Theodore Barr Foundation through the Glaucoma Research Foundation, with additional grant funding provided in part by the International Retinal Research Foundation and the National Eye Institute of the National Institutes of Health.

About Glaucoma

Glaucoma is a neurodegenerative disorder that causes blindness by damaging the optic nerve, which sends signals from the eye to the brain. It affects more than 60 million people and is the second leading cause of blindness worldwide. While older individuals are at higher risk for the disease, babies and children are also susceptible to glaucoma, especially those with certain neurological disorders.

http://www.eurekalert.org/pub_releases/2011-01/wfub-pdo010311.php

Peptide delivers 1-2 punch to breast cancer in pre-clinical study

WINSTON-SALEM, N.C. -- ***Researchers at Wake Forest University Baptist Medical Center (WFUBMC) have discovered what may become a new weapon in the fight against breast cancer. For the first time, a peptide found in blood and tissue has been shown to inhibit the growth of human breast tumors in mice, according to a study recently published in the journal Cancer Research.***

Patricia E. Gallagher, Ph.D., and E. Ann Tallant, Ph.D., scientists in the Hypertension and Vascular Research Center at WFUBMC, demonstrated that the peptide angiotensin-(1-7) attacked breast cancer in two ways: by inhibiting the growth of the breast cancer cells themselves and by inhibiting the growth of cancer-associated fibroblasts (CAFs), cells found in the tumor microenvironment -- the tissue surrounding the tumor. CAFs play a vital role in tumor initiation, growth and metastases by providing structural support for the tumor cells and by producing growth factors that help the tumor cells grow.

In this study, mice were injected with human breast cancer cells to form the two most common types of breast tumors -- estrogen-receptor and HER2 sensitive. In women with breast cancer, an estimated 50 to 60 percent have estrogen-receptor sensitive tumors and 20 to 30 percent have HER2 sensitive tumors.

Once the tumors grew, the mice were injected with either angiotensin-(1-7) or saline for 18 days. In the mice treated with angiotensin-(1-7), there was a 40 percent reduction in tumor size as compared to the saline-injected mice, whose tumors grew three times their size at the initiation of treatment. Breast tumor fibrosis also was reduced by 64 to 75 percent in the mice treated with the peptide as compared to the saline-injected mice. Fibrosis is the thickening of the breast tissue around and within the tumor that acts as a scaffold to support the spread of cancer cells.

"This is the first study to show that angiotensin-(1-7) not only inhibits the growth of tumors, but also inhibits breast tumor fibrosis," Gallagher said. "Think of it as a seed and the soil around it – the seed being the tumor and the soil being the fibrosis. You can attack the seed, or you can attack the soil, or do both, and our drug does both."

The tumor microenvironment is especially important when the cancer has metastasized, Tallant said, because drugs that are effective for treating the primary tumor often are not effective in treating a tumor growing in a different part of the body. "Our findings also suggest that angiotensin-(1-7) may enhance the effect of chemotherapeutic agents when administered in combination with other drugs by altering the microenvironment in which the tumor grows," she said.

"Because the safety of angiotensin-(1-7) was established here at Wake Forest Baptist in a recently completed trial in patients with different types of solid tumors, we hope to go to clinical trials for breast cancer relatively soon," Gallagher said.

Gallagher's and Tallant's initial research conducted at the Comprehensive Cancer Center at Wake Forest Baptist showed that angiotensin-(1-7) inhibited the growth of vascular smooth muscle cells, the cells that surround blood vessels and regulate blood pressure. Previous studies showed that patients treated with drugs to reduce blood pressure and increase angiotensin-(1-7), also had a smaller chance of developing cancer. Based on this information, Gallagher and Tallant studied the effect of the peptide on lung cancer and discovered that angiotensin-(1-7) inhibited the growth of lung tumors in mice, as well as reduced the supply of blood vessels to the growing tumor. Their latest study, as reported in Cancer Research, now shows additional effects of angiotensin-(1-7).

Both scientists and Wake Forest University Baptist Medical Center hold a patent on the use of angiotensin-(1-7) for the treatment of cancer.

http://www.scientificamerican.com/blog/post.cfm?id=did-big-babies-help-bring-human-anc-2011-01-03

Did big babies help bring human ancestors down from the trees?

By Katherine Harmon | Monday, January 3, 2011 | 1

Relative to our ape brethren, humans give birth to really big babies. This especially substantial infant size—along with newborns' large heads and general helplessness—helped to spur the development of more advanced social systems to help mother and child safe, researchers think.

A new study examines the evolution of this trend to try to pinpoint when in human evolution this growth spurt occurred—and how it might have signaled a shift in social dynamics as well.

Modern humans give birth to babies that are generally about 6.1 percent of the mother's body weight, whereas chimpanzee babies are usually closer to 3.3 percent of the mother's mass. And as anthropologist Jeremy DeSilva of Boston Univeristy pointed out in his new paper, "carrying a relatively large infant both pre- and postnatally has important ramifications for birthing strategies, social systems, energetics, and locomotion."

Using models that estimate neonatal brain and body mass, DeSliva estimated that the 4.4-million-year-old hominin Ardi (Ardipithecus ramidus) likely would have borne an infant between 2.1 and 3.2 percent of the mother's body mass, which would place it closer to more primitive hominin traits. By about 3.2 million years ago, however, "females of the genus Australopithecus were giving birth to relatively large infants" of about 5 to 6 percent of maternal body mass, DeSilva noted in his paper.

For modern chimpanzees—and likely early human ancestors—with relatively small infants, strong grasping toes and long, clingable body hair, taking babies into the trees with them both before and after birth is not too taxing. But as these traits seem to disappear with Australopithecus, arboreal living would be "a more dangerous activity," DeSilva wrote, noting that bipedal, less hairy mothers would have to park their young or actively carry them. And these bigger, probably more helpless babies of Australopithecus might not have started walking until six or seven months after birth, he estimated.

Saddled with a large, nursing infant that is unable to walk, Australopithecus mothers could have used assistance from males as well as juveniles in caring for themselves and their babies (grandmothers are thought

to have come into the picture only after lifespan lengthened with the emergence of the Homo genus). This new need for extended postnatal care may have driven the emergence of a social structure different from that of chimpanzees, which "rarely will share their infants with other members of the group," DeSilva noted. And despite assertions that more modern rearing practices did not emerge until the evolution of early Homo species, the new assessment indicates that "the increased levels of shared infant care critical to infant survival in modern humans could have its roots in the genus Australopithecus," rather than in Homo, DeSilva hypothesized

DeSilva cautioned, however, that many of the adult body mass estimates derive from "only a small number of often taxonomically ambiguous fossil specimens," and he suggests his calculations be retested as new hominin bones are uncovered.

His analysis was published online January 3 in Proceedings of the National Academy of Sciences.

http://www.nytimes.com/2011/01/04/health/04really.html

The Claim: Taking a Walk Can Help Reduce Cravings

By ANAHAD O'CONNOR

THE FACTS ***Do your New Year's resolutions tend to fizzle like a glass of chilled Champagne?***

If your goal is to break a bad habit or cut back on food and shed a few pounds, then a simple but overlooked trick could come in handy: go for a walk. As far as weight-loss strategies go, it is not the most glamorous, but studies have found that a brisk walk around the block can significantly dampen cravings, whether the urge for junk food or the desire to light up a cigarette.

In a 2008 study, researchers recruited a group of "regular chocolate eaters" — people who ate at least two chocolate bars a day — and had them abstain for three days. They then divided them into groups, put them to work on difficult cognitive tests to raise their stress levels, and tempted them with unwrapped chocolate bars.

The researchers found that if the subjects walked for 15 minutes on a treadmill at a pace that was brisk but not tiring, they were far less likely to suffer cravings, and even showed lower blood pressure when handling the chocolate bars.

In other studies, scientists looked at the effects of brief walks on cigarette cravings. One in 2005 found that smokers who were told to abstain for a day had rapid reductions in the urge to smoke when they took "self-paced, low-intensity" walks lasting about 15 minutes.

Another study in 2007 showed that brief walks not only beat back cravings, but also reduced withdrawal symptoms and increased the time between cigarettes smoked.

THE BOTTOM LINE Studies show that a brisk walk can ease cravings and help break some habits.

http://www.wired.com/wiredscience/2011/01/zapotec-thighbones/

Decline of an Empire Seen in Zapotec Thighbones

By Brandon Keim

A newly excavated Zapotec burial site has yielded a fresh interpretation of the ancient, grisly Mesoamerican custom of removing thighbones from the dead.

Across pre-Hispanic Mesoamerica, femurs were believed to contain an individual's power. Aztecs treated them as war trophies, while Zapotec royalty are thought to have used them like sceptres, as symbols of ancestral political might.

Burial site at Mitla, with arrow pointing to missing femur./Antiquity.

The new excavation, in a relatively humble residential dwelling in the ancient city of Mitla, suggests that ancestral thighbone-wielding "may not have been a practice limited to rulers," wrote researchers led by Field Museum archaeologist Gary Feinman in a study published in December in Antiquity.

Thighbone customs of the Zapotec civilization, which reigned from the late 6th century BC to the early 16th century in what is now the Oaxaca valley of Mexico, are best known from a pair of burial sites.

The first, a 16th-century tomb in the city of Monte Alban that was excavated in the 1930s, yielded the remains of nine individuals, along with three extra femurs. These had been cut and painted, suggesting Aztec-style trophy use.

In the 1970s, another 16th-century tomb was excavated, this time in the smaller city of Lambityeco. It was part of a palatial residence, clearly occupied by rulers, six of whom had been buried there — but only three of their thighbones remained. The rest were missing.

Friezes on the wall depicted men holding what appeared to be femurs, giving rise to the interpretation of thighbones as scepters. Subsequent burial excavations have supported this hypothesis, but the sites have tended to be poorly preserved, with skeletons missing many bones.

The tomb excavated by Feinman at Mitla was extremely well-preserved, and had never been disturbed — except, that is, by someone who broke open the tomb, removed a thighbone, then carefully resealed it, leaving a bowl as an offering.

According to Feinman's team, that offering suggests a veneration for the deceased. As the tomb was part of a residence — Zapotec dead were commonly buried in this fashion, with dwellings occupied for generations — it had likely been opened by a descendant.

Meanwhile, the upper portion of the skeleton was in slight disarray, while the lower portion was undisturbed except for the missing femur. The researchers interpret this as evidence of a generational gap. Whoever opened the tomb knew where it was, but not how it was aligned; they accidentally broke into the top part first, realized their mistake, then gently removed the thighbone.

Frieze at Lambityeco depicting man holding a femur./Antiquity.

The residence was located on a terraced hillside Mitla, part of a relatively nondescript neighborhood, well down from the dwellings of rulers at the top. It was, however, in the center of the neighborhood, atop a rocky promontory that would have made it an ideal lookout. The researchers think it belonged to someone like a ward boss, revered by his descendants.

To Feinman, the generational gap hinted at by this site, and the common dating of Zapotec thighbone customs to its Late Classic period, shortly before conquest by the Aztecs and then the Spanish, suggest the structure of society and tenor of life at the empire's end.

As Zapotec power dwindled, so did its centralized authorities. Prominent local families gained power. This was rooted in personal and lineal networks, rather than in political tradition. And to demonstrate their legitimacy, families showed off their ancestors' bones.

http://www.eurekalert.org/pub_releases/2011-01/luhs-edm010311.php

Experimental drug more potent, longer lasting than morphine

Less likely to cause constipation

MAYWOOD, Ill. - A little-known morphinelike drug is potentially more potent, longer lasting and less likely to cause constipation than standard morphine, a study led by a Loyola University Health System anesthesiologist has found. The drug, morphine-6-0-sulfate, has a similar chemical structure to standard morphine. Dr. Joseph Holtman Jr. and colleagues reported that a study they performed in rats "demonstrated potential clinical advantages of morphine-6-0-sulfate compared to morphine." Holtman is first author of the study, published in the December 2010 issue of the European Journal of Pharmacology.

Holtman is medical director of Loyola's Pain Specialty Service and a professor in the departments of Anesthesiology and Molecular Pharmacology and Therapeutics of Loyola University Chicago Stritch School of Medicine. He directed the study while he was at the University of Kentucky's College of Medicine. He joined Loyola on March 1, 2010.

Opioids, such as morphine, oxycodone and hydrocodone, are standard drugs for treating moderate to severe pain, including cancer pain. But these drugs can have significant side effects, including constipation, nausea, vomiting, drowsiness, cognitive dysfunction and slowed breathing and heart rates. And while opioids work well for conditions such as back pain and post-operative pain, the drugs are less effective against neuropathic pain, such as tingling, burning or shooting pain.

Constipation is a common side effect of morphine and can be so uncomfortable that some patients limit their use of the drug. Doctors typically do not discharge surgery patients until they have had a bowel movement and this can extend hospital stays.

Holtman and colleagues tested standard morphine and morphine-6-0-sulfate on rats. The animals received the drugs three ways -- by mouth, by IV and by injection into the space surrounding the spinal cord. The rats underwent several well-established tests to determine their sensitivity to pain. In one such test, researchers focused a very warm light beam on the tail and measured how long it took for the rat to flick the tail.

In this tail-flick test, morphine-6-0-sulfate was 10 times more potent than standard morphine when administered in the space surrounding the spinal cord, five times more potent when administered by IV and two

times more potent when given by mouth. Morphine-6-0-sulfate maintained its maximum effect for three hours, compared with 1½ hours for standard morphine. And it took rats 25 days to build tolerance to morphine-6-0-sulfate, compared with 10 days with standard morphine.

Morphine-6-0-sulfate also was more potent than standard morphine for neuropathic and inflammatory pain.

Researchers found that morphine-6-0-sulfate could cause constipation, but only at doses 10 to 20 times higher than the effective doses. The findings suggest that morphine-6-0-sulfate "may be an interesting potential drug for further study," Holtman and colleagues wrote.

Co-authors of the study, all at the University of Kentucky, are Peter Crooks, Jaime Johnson-Hardy and Elzbieta Wala.
The study was funded by Insys Therapeutic, Inc., which is has a license to develop the drug for possible use in humans.

http://www.eurekalert.org/pub_releases/2011-01/aafc-emh122810.php

Estrogen may help precancerous cells spread in oral cavity

PHILADELPHIA — *Head and neck cancer is the sixth most common type of cancer and is on the rise in some demographic groups, including young women without any known risk factors. Now, researchers at Fox Chase Cancer Center report that estrogen may increase the movement of precancerous cells in the mouth and thus promote the spread of the disease within the oral cavity.*

The new results, published in the January issue of Cancer Prevention Research, a journal of the American Association for Cancer Research, may lead to novel chemoprevention strategies in the future.

Margie Clapper, Ph.D., co-leader of the Cancer Prevention and Control Program at Fox Chase Cancer Center and Cancer Prevention Research editorial board member, and colleagues had previously reported that estrogen metabolism changes following smoke exposure in the lungs and may contribute to lung cancer. This study on estrogen and lung cancer first appeared in the June 3, 2010, issue of Cancer Prevention Research.

To find out if this female hormone influences development of head and neck cancer, Ekaterina Shatalova, Ph.D., a postdoctoral fellow at Fox Chase Cancer Center and researcher on this study, examined the impact of estrogen on precancerous and cancerous cells.

They found that estrogen induces the expression of an enzyme called cytochrome P450 1B1 (CYP1B1), which is responsible for breaking down toxins and metabolizing estrogen. Interestingly, CYP1B1 induction occurred only in precancerous cells, which are neither totally normal nor cancerous. Surprisingly, estrogen did not induce CYP1B1 in cancer cells.

With closer investigation, the researchers found that depleting the expression of CYP1B1 diminished the ability of precancerous cells to move and divide, as compared to similar cells with normal levels of CYP1B1. Estrogen also reduced cell death in the precancerous cells, irrespective of the amount of CYP1B1 present.

"In the future, we would like to find a natural or dietary agent to deplete the CYP1B1 enzyme and see if we can prevent oral cancer at the precancerous stage," said Shatalova.

"Our previous studies showed that the CYP1B1 enzyme sits at the hub of changes that occur in the lungs after smoke exposure. We were now able to look at its role in a more direct fashion by removing it from precancerous cells of the oral cavity," Clapper said. "We found that cells lacking it move slower. CYP1B1 could be a wonderful target in precancerous lesions of the head and neck, because by attacking it, we might stop these lesions from progressing or moving to a more advanced stage."

In addition, patients diagnosed with head and neck cancer are at a high risk of developing a second primary tumor, which is associated with poorer overall survival. Finding a way to reduce these subsequent tumors could improve patients' survival.

These results may help researchers to "understand factors that cause head and neck cancer, in addition to the traditional risk factors of tobacco and alcohol exposure," said Jennifer R. Grandis, M.D., professor and director of the Head and Neck Cancer Program at the University of Pittsburgh School of Medicine, and an editorial board member for Cancer Prevention Research.

However, because these results are limited to a single premalignant cell line, said Grandis, further studies are needed to validate these findings in head and neck cancer in a human population.

http://www.eurekalert.org/pub_releases/2011-01/cp-bbi122910.php

Birch bark ingredient comes with many metabolic benefits

An ingredient found in abundance in birch bark appears to have an array of metabolic benefits, according to new studies in animals that are reported in the January issue of Cell Metabolism, a Cell Press publication.

In mice, the compound known as betulin lowered cholesterol, helped prevent diet-induced obesity, and improved insulin sensitivity. Betulin-treated mice were also more resistant to developing atherosclerotic plaques in their arteries.

Betulin works by targeting so-called sterol regulatory element-binding proteins (SREBPs), transcription factors that are known to be important for activating the expression of genes involved in the biosynthesis of cholesterol, fatty acids, and triglycerides.

"Our study shows that the SREBP pathway is a good target for several metabolic diseases," said Bao-Liang Song of the Shanghai Institutes for Biological Sciences. "We also identify a leading compound."

In the new study, Song and his colleagues went in search of a compound that might act directly on SREBP. That chemical screen turned up betulin as a top contender. They then confirmed in cells that betulin lowered the activity of genes that are normally switched "on" by SREBP. It also lowered lipid levels within cells.

Song's team then treated mice on a high-fat, Western diet with betulin, the cholesterol-lowering statin known as lovastatin, or a placebo (saline) for 6 weeks. Compared to placebo, both drugs led the mice to gain less weight on the high-fat diet, though by different means. Betulin caused the animals to burn more calories while lovastatin appeared to reduce the amount of lipid taken up from the diet.

Further investigations showed that betulin also lowered lipid levels in blood, liver, and fat tissue. Betulin also made the animals more sensitive to insulin. Mice with a mutation that makes them prone to develop atherosclerosis showed fewer plaques when treated with either lovastatin or betulin.

"Betulin has several major metabolic effects," Song said.

The researchers say that their findings suggest that betulin may have similar or even better effects than lovastatin, a member of the most widely prescribed drug class for treating high cholesterol. For instance, in their studies betulin decreased lipids in liver and fat to a greater extent than lovastatin did. Betulin also improved insulin resistance through its effects on fatty acid and triglyceride synthesis.

Song notes that betulin is a readily available compound and is already in use as a precursor in the manufacture of other drugs.

Although betulin appears to have very low toxicity, he says future studies will need to further investigate the safety of betulin and its metabolic effects. Researchers will also explore the possibility that a derivative of betulin might have even greater potency. "That may be the path forward to move this clinically," Song said.

http://www.eurekalert.org/pub_releases/2011-01/rpi-cs010311.php

'Nanoscoops' could spark new generation of electric automobile batteries

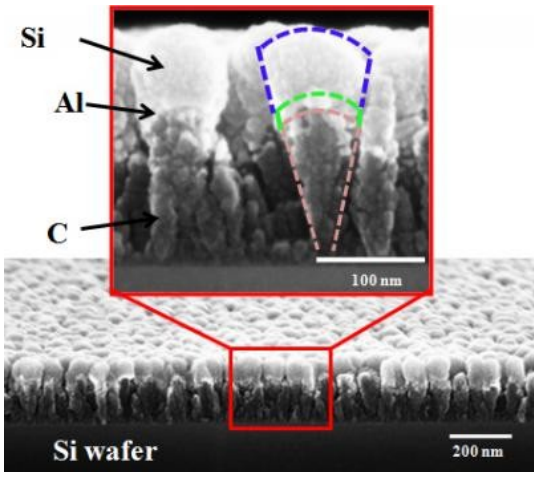
New nanoengineered batteries developed at Rensselaer exhibit remarkable power density, charging more than 40 times faster than today's lithium-ion batteries

Troy, N.Y. – An entirely new type of nanomaterial developed at Rensselaer Polytechnic Institute could enable the next generation of high-power rechargeable lithium (Li)-ion batteries for electric automobiles, as well as batteries for laptop computers, mobile phones, and other portable devices.

The new material, dubbed a "nanoscoop" because its shape resembles a cone with a scoop of ice cream on top, can withstand extremely high rates of charge and discharge that would cause conventional electrodes used in today's Li-ion batteries to rapidly deteriorate and fail. The nanoscoop's success lies in its unique material composition, structure, and size.

The Rensselaer research team, led by Professor Nikhil Koratkar, demonstrated how a nanoscoop electrode could be charged and discharged at a rate 40 to 60 times faster than conventional battery anodes, while maintaining a comparable energy density. This stellar performance, which was achieved over 100 continuous charge/discharge cycles, has the team confident that their new technology holds significant potential for the design and realization of high-power, high-capacity Li-ion rechargeable batteries.

"Charging my laptop or cell phone in a few minutes, rather than an hour, sounds pretty good to me," said Koratkar, a professor in the Department of Mechanical, Aerospace, and Nuclear Engineering at Rensselaer. "By using our nanoscoops as the anode architecture for Li-ion rechargeable batteries, this is a very real prospect. Moreover, this technology could potentially be ramped up to suit the demanding needs of batteries for electric automobiles."



Researchers at Rensselaer Polytechnic Institute developed an entirely new type of nanomaterial that could enable the next generation of high-power rechargeable lithium (Li)-ion batteries for electric automobiles, laptop computers, mobile phones and other devices. The material, called a "nanoscoop" because it resembles a cone with a scoop of ice cream on top, is shown in the above scanning electron microscope image. Nanoscoops can withstand extremely high rates of charge and discharge that would cause today's Li-ion batteries to rapidly deteriorate and fail.

Rensselaer/Koratkar

Batteries for all-electric vehicles must deliver high power densities in addition to high energy densities, Koatkar said. These vehicles today use supercapacitors to perform power-intensive functions, such as starting the vehicle and rapid acceleration, in conjunction with conventional batteries that deliver high energy density for normal cruise driving and other operations. Koratkar said the invention of nanoscoops may enable these two separate systems to be combined into a single, more efficient battery unit.

Results of the study were detailed in the paper "Functionally Strain-Graded Nanoscoops for High Power Li-Ion Battery Anodes," published Thursday by the journal Nano Letters. See the full paper at: http://pubs.acs.org/doi/abs/10.1021/nl102981d

The anode structure of a Li-ion battery physically grows and shrinks as the battery charges or discharges. When charging, the addition of Li ions increases the volume of the anode, while discharging has the opposite effect. These volume changes result in a buildup of stress in the anode. Too great a stress that builds up too quickly, as in the case of a battery charging or discharging at high speeds, can cause the battery to fail prematurely. This is why most batteries in today's portable electronic devices like cell phones and laptops charge very slowly – the slow charge rate is intentional and designed to protect the battery from stress-induced damage.

The Rensselaer team's nanoscoop, however, was engineered to withstand this buildup of stress. Made from a carbon (C) nanorod base topped with a thin layer of nanoscale aluminum (Al) and a "scoop" of nanoscale silicon (Si), the structures are flexible and able to quickly accept and discharge Li ions at extremely fast rates without sustaining significant damage. The segmented structure of the nanoscoop allows the strain to be gradually transferred from the C base to the Al layer, and finally to the Si scoop. This natural strain gradation provides for a less abrupt transition in stress across the material interfaces, leading to improved structural integrity of the electrode.

The nanoscale size of the scoop is also vital since nanostructures are less prone to cracking than bulk materials, according to Koratkar.

"Due to their nanoscale size, our nanoscoops can soak and release Li at high rates far more effectively than the macroscale anodes used in today's Li-ion batteries," he said. "This means our nanoscoop may be the solution to a critical problem facing auto companies and other battery manufacturers – how can you increase the power density of a battery while still keeping the energy density high?"

A limitation of the nanoscoop architecture is the relatively low total mass of the electrode, Koratkar said. To solve this, the team's next steps are to try growing longer scoops with greater mass, or develop a method for stacking layers of nanoscoops on top of each other. Another possibility the team is exploring includes growing the nanoscoops on large flexible substrates that can be rolled or shaped to fit along the contours or chassis of the automobile.

Along with Koratkar, authors on the paper are Toh-Ming Lu, the R.P. Baker Distinguished Professor of Physics and associate director of the Center for Integrated Electronics at Rensselaer; and Rahul Krishnan, a graduate student in the Department of Materials Science and Engineering at Rensselaer.

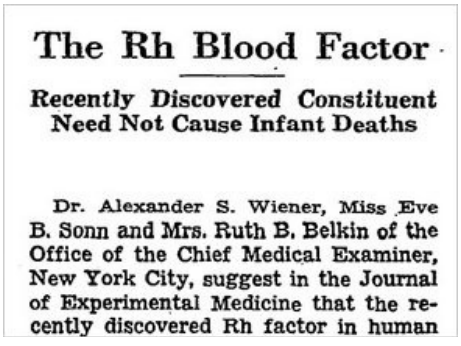
This study was supported by the National Science Foundation (NSF) and the New York State Energy Research and Development Authority (NYSERDA).

http://www.nytimes.com/2011/01/04/health/04first.html

Rh Factor, 1944

By NICHOLAS BAKALAR

The so-called Rh factor makes few headlines these days, but until the middle of the 20th century it was a serious public-health concern, implicated in the deaths and severe disabilities of 10,000 babies in this country every year.



The Rh Blood Factor

Recently Discovered Constituent Need Not Cause Infant Deaths

Dr. Alexander S. Wiener, Miss Eve B. Sonn and Mrs. Ruth B. Belkin of the Office of the Chief Medical Examiner, New York City, suggest in the Journal of Experimental Medicine that the recently discovered Rh factor in human

The presence or absence of the blood protein Rh (for rhesus, the monkey in which it was discovered) can lead to runaway immune reactions in Rh-positive babies born to Rh-negative mothers, or in people receiving transfusions of incompatible blood.

So in hindsight, The New York Times's first mention of the Rh factor, on Sunday, March 26, 1944, should have made bigger news than it did — in a brief article at the bottom of the "Science in Review" column on Page 9 of Section 4, The News of the Week in Review. "The recently discovered Rh factor in human blood," it said, "need not cause infant deaths and childless marriages."

The article quoted Dr. Alexander S. Wiener, who in 1940, along with his colleague Karl Landsteiner, first described the Rh factor in humans. "Dr. Wiener believes that some method may be developed to desensitize

mothers so that their babies may be saved," the article said. "Research based on this hope has already been started."

The first mention of a preventive treatment for newborns with Rh disease appeared on April 24, 1947, in a report of a conference at which Dr. Philip Levine said that "the destruction of red blood cells in new-born babies of mothers with an Rh disturbance in their blood may be controlled if the Rh negative pregnant woman receives only Rh negative blood in the event of a transfusion," and that her baby "may be saved by the transfusion of Rh negative blood."

Unfortunately, he added, blood tests for the Rh factor were not widely available to pregnant women. But they were available to lawyers and their clients. On July 21, 1947, The Times reported the first use of the Rh factor, an inherited trait, as a test of parentage in a court case. The judge decided that on the basis of the man's Rh test he could not be the father

It was not until Sept. 11, 1965, that the paper reported on clinical trials of a drug treatment for Rh disease. The article did not refer to the substance by name, but this was the first test of Rh immune globulin, a solution of antibodies derived from human plasma.

Injected into the Rh-negative mother, the antibodies bind to and destroy fetal Rh-positive blood cells that have passed from the fetus to the mother during birth. If the trials succeeded, the vaccine would prevent the maternal immune reaction that can cause Rh disease in babies.

The trials did succeed. On April 24, 1968, an article on Page 48 by Jane E. Brody began, "A New Jersey pharmaceutical laboratory announced yesterday that its vaccine to prevent Rh blood disease in infants had been approved for marketing and would be generally available in June."

On April 28, Ms. Brody reported that the drug, Rhogam, "will be made available to hospitals at $64.80 a dose" — about $407 in today's money. Rh immune globulin, or RhIg (Rhogam is one of several brands), now costs about $100 a dose. Rh blood disease is no longer a threat.

http://news.discovery.com/space/viking-mars-organics-experiment.html

Viking Found Organics on Mars, Experiment Confirms

Using Mars-like soil taken from Atacama Desert, a study confirms Mars has organics, and Viking found them.

By Irene Klotz | Tue Jan 4, 2011 07:57 AM ET

More than 30 years after NASA's Viking landers found no evidence for organic materials on Mars, scientists say a new experiment on Mars-like soil shows Viking did, in fact, hit pay dirt.

The new study was prompted by the August 2008 discovery of powerful oxygen-busting compounds known as perchlorates at the landing site of another Mars probe called Phoenix.

Scientists repeated a key Viking experiment using perchlorate-enhanced soil from Chile's Atacama Desert, which is considered one of the driest and most Mars-like places on Earth, and found telltale fingerprints of combusted organics -- the same chemicals Viking scientists dismissed as contaminants from Earth.

"Contrary to 30 years of perceived wisdom, Viking did detect organic materials on Mars," planetary scientist Christopher McKay, with NASA's Ames Research Center in California, told Discovery News. "It's like a 30-year-old cold case suddenly solved with new facts."

"If the Viking team had said 'Well, maybe there's perchlorate in the soil,' everybody would have said they're crazy -- why would there be perchlorates in the soil? It was only by having it pushed on us by Phoenix where we had no alternative but to conclude that there was perchlorate in the soil … Once you realize it's there, then everything makes sense," McKay added.

The Viking team's verdict that Mars lacked organics was the lynchpin argument against another Viking experiment that looked for signs of microbial life. In the experiment, a bit of nutrient-laced water was added to a sample of Martian soil.

The air above the soil was then monitored for signs that the nutrients had been metabolized. The instrument detected tracer gases the first time the experiment was done, but subsequent runs did not. The results were considered inconclusive and remain contested.

New evidence for organics on Mars does not mean Viking found life, cautions McKay. "Finding organics is not evidence of life or evidence of past life. It's just evidence for organics," he said.

But if NASA had realized there were organics on Mars, there might not have been a 20-year hiatus in sending landers for follow-up studies, said Rafael Navarro-González, with the Institute of Nuclear Science at the National Autonomous University in Mexico. "We might have had continuing missions," Navarro-González told Discovery News. NASA plans to launch a follow-up mission to look for organics on Mars in November.

The research appears in last month's Journal of Geophysical Research.

http://news.discovery.com/earth/atlantic-circulation-on-the-fasttrack-for-change.html

Atlantic Circulation On the Fasttrack for Change

By Tim Wall | Tue Jan 4, 2011 05:55 PM ET

Gorgonian coral Bell-bottom jeans, Abba, and the Labrador Current -- one of the three is not making a retro comeback. Temperature-tracking coral reefs indicate that the circulation of water in the Atlantic Ocean has changed dramatically since the 1970's.

That could be part of the reason areas in the northern hemisphere have had harsh winter storms and summer droughts say researchers. The evidence for changing currents comes from ancient gorgonian coral reefs growing off the coast of Nova Scotia. The reef was studied by a team of biochemists and oceanographers from Switzerland, Canada, and the United States.

Like most organisms, corals are what they eat and changes in their diet are recorded in the reef structure.

The Canadian corals showed that the cold, south-bound Labrador current is losing ground to the warm, north-bound Gulf Stream current. The corals of the deep north Atlantic have been feasting on nutrient-rich warm water since the 1970's. The researchers looked at the concentrations of a certain isotope of nitrogen, called delta 15. Different concentrations of the isotope allow scientists to trace a creature's food sources.

The levels the researchers found in the Nova Scotian coral indicated the coral had been feeding heavily on nutrients brought north from the subtropical regions for approximately 40 years.

The coral had been feeding primarily on subarctic nutrients for the previous 1800 years.

That abrupt change in food sources caused by changes in currents coincides closely with the onset of observable changes in Earth's climate caused by industrialization.

"The researchers suspect there is a direct connection between the changes in oceanic currents in the North Atlantic and global warming caused by human activities," said one of the participating institutions, the Swiss Federal Institute of Aquatic Sciences and Technology in a statement to the Associated Foreign Press.

The coral reef research was published recently in the Proceedings of the National Academy of Sciences.

Recent heavy snowfalls and harsh winter storms along with droughts and heat waves in the summer, could be explained by a changes in the circulation of the Atlantic's water, say climate researchers. A 2004 paper by NASA explained the possibility of melting arctic sea ice triggering colder weather in Europe and North America.

As my colleague, Mr. Cox, pointed out in the above blog post, the term global warming is a misnomer in many senses. Humanity faces global climate destabilization. The weather patterns our agriculture and industry have adapted to over the centuries are changing rapidly. In his book Collapse, Jared Diamond, points out many examples of civilizations failing to adapt to climatic changes. Weather changes in the north contributed heavily to the demise of the Vikings in Greenland, for example.

Other examples of civilizations crumbling under environmental pressures comes from 535 AD, as detailed by David Keys in Catastrophe: A Quest for the Origins of the Modern World. Civilizations from Mexico to Java to the Byzantine empire show evidence of a serious crisis at that time. An event, possibly a massive volcanic eruption, caused a world-wide reduction in temperature and darkening of the skies. Crops failed and famine followed.

History shows us that fluctuations in the climate can have serious effects on humans and the Earth's ecosystems. But the Earth has recovered from numerous calamities in the past. The real danger from climate change, biodiversity loss, deforestation, ocean acidification, and other phenomenon is to the stability for global human civilization. The Earth will be fine. It's humanity that needs to better plan for its future.

http://www.eurekalert.org/pub_releases/2011-01/idso-sct010311.php

Study confirms 2 vaccine doses protect children from chickenpox

Two doses of the varicella, or chickenpox, vaccine provide excellent protection in children against this highly contagious and, in some cases, severe disease.

To be published in the February 1 issue of The Journal of Infectious Diseases, the findings support the two-dose vaccine regimen recommended in the United States since 2006. (Please see below for a link to the study online.)

The Centers for Disease Control and Prevention (CDC) began recommending a single dose of varicella vaccine in children aged 1 to 13 years old in 1995. Although the incidence of varicella fell by 90 percent after introduction of the vaccine, there was a high rate of breakthrough varicella illness in immunized children and continuing outbreaks of varicella among children despite high rates of vaccination. Studies also showed that the single-dose vaccine's effectiveness was less than 90 percent. Given the evidence, CDC in 2006 began recommending a second dose of the vaccine for children 4 to 6 years old.

Although data suggest that two doses of varicella vaccine are associated with higher levels of antibody than is one dose, this study is the first to assess the clinical effectiveness of two doses of the vaccine in the general population. Eugene D. Shapiro, MD, and colleagues at Yale University and collaborators at Columbia University conducted active surveillance in an area in Connecticut and discovered 71 cases of varicella in children aged 4 or older. None of the children had received two doses of vaccine, 66 (93 percent) had received one dose, and 5 (7 percent) had received no vaccine.

The investigators then compared the effectiveness of two doses of vaccine versus one dose in a case-control study, using 140 matched controls. The effectiveness of one dose in preventing varicella was 86.0 percent, while the effectiveness of two doses was 98.3 percent. According to Dr. Shapiro, "The odds of developing varicella were 95 percent lower in children who had received two doses of the vaccine compared with those who had received only one."

The results of this study suggest that countries immunizing children with only one dose of varicella vaccine should consider changing to a two-dose regimen. But, the authors emphasized, "There should be continued monitoring of the effectiveness of two doses to assure that its high degree of effectiveness is sustained."

In an accompanying editorial, David W. Kimberlin, MD, of the University of Alabama at Birmingham, agreed with the study authors, noting that this study is the first to evaluate the effectiveness of two doses of varicella vaccine in a "real-world" setting. "The high effectiveness of 98.3 percent found in this investigation supports the programmatic change instituted four years ago," Dr. Kimberlin noted.

Fast Facts:

1) In this study, the odds of developing varicella (chickenpox) were 95 percent lower in children > 4 years of age who had received two doses of the varicella vaccine compared with those who had received only one dose.
2) Of the 71 cases of varicella noted in the study, none of the subjects had received two doses of vaccine.
3) The effectiveness of two doses of vaccine in protecting against varicella in the study population was 98.3 percent.
NOTE: The study and the accompanying editorial are available online. They are embargoed until 12:01 a.m. EST on Wednesday, Jan. 5, 2011:
"Effectiveness of 2 Doses of Varicella Vaccine in Children" http://www.oxfordjournals.org/our_journals/jid/jiq052.pdf
http://www.eurekalert.org/pub_releases/2011-01/wt-bss010511.php

Brain scans show children with ADHD have faulty off-switch for mind-wandering

Brain scans of children with attention-deficit/hyperactivity disorder (ADHD) have shown for the first time why people affected by the condition sometimes have such difficulty in concentrating.

The study, funded by the Wellcome Trust, may explain why parents often say that their child can maintain concentration when they are doing something that interests them, but struggles with boring tasks.

Using a 'Whac-a-Mole' style game, researchers from the Motivation, Inhibition and Development in ADHD Study (MIDAS) group at the University of Nottingham found evidence that children with ADHD require either much greater incentives – or their usual stimulant medication – to focus on a task. When the incentive was low, the children with ADHD failed to "switch off" brain regions involved in mind-wandering. When the incentive was high, however, or they were taking their medication, their brain activity was indistinguishable from a typically-developing non-ADHD child.

ADHD is the most common mental health disorder in childhood, affecting around one in 50 children in the UK. Children with ADHD are excessively restless, impulsive and distractible, and experience difficulties at home and in school. Although no cure exists for the condition, symptoms can be reduced by medication and/or behavioural therapy. The drug methylphenidate (more often known by the brand name Ritalin) is commonly used to treat the condition.

Previous studies have shown that children with ADHD have difficulty in 'switching-off' the default mode network (DMN) in their brains. This network is usually active when we are doing nothing, giving rise to spontaneous thoughts or 'daydreams', but is suppressed when we are focused on the task before us. In children with ADHD, however, it is thought that the DMN may be insufficiently suppressed on 'boring' tasks that require focused attention.

The MIDAS group researchers compared brain scans of eighteen children with ADHD, aged between nine and fifteen years old, against scans of a similar group of children without the condition as both groups took part in a task designed to test how well they were able to control their behaviour. The children with ADHD were tested when they were taking their methylphenidate and when they were off their medication. The findings are published in the Journal of Child Psychology and Psychiatry.

Whilst lying in a magnetic resonance imaging (MRI) scanner, which can be used to measure activity in the brain, the children played a computer game in which green aliens were randomly interspersed with less frequent black aliens, each appearing for a short interval. Their task was to 'catch' as many green aliens as possible,

while avoiding catching black aliens. For each slow or missed response, they would lose one point; they would gain one point for each timely response.

To study the effect of incentives, the reward for avoiding catching the black alien was then increased to five points, with a five-point penalty incurred for catching the wrong alien.

By studying the brain scans, the researchers were able to show that typically developing children switched off their DMN network whenever they saw an item requiring their attention. However, unless the incentive was high, or they had taken their medication, the children with ADHD would fail to switch off the DMN and would perform poorly. This effect of incentives was not seen in children without ADHD – activity in their DMN was switched off by items requiring their attention regardless of the incentive on offer.

Professor Chris Hollis, who led the study, says: "The results are exciting because for the first time we are beginning to understand how in children with ADHD incentives and stimulant medication work in a similar way to alter patterns of brain activity and enable them to concentrate and focus better. It also explains why in children with ADHD their performance is often so variable and inconsistent, depending as it does on their interest in a particular task."

Dr Martin Batty, co-author of the study, adds: "Using brain imaging, we have been able to see inside the children's heads and observe what it is about ADHD that is stopping them concentrating. Most people are able to control their 'daydreaming' state and focus on the task at hand. This is not the case with children with ADHD. If a task is not sufficiently interesting, they cannot switch off their background brain activity and they are easily distracted. Making a task more interesting – or providing methylphenidate – turns down the volume and allows them to concentrate."

Dr Elizabeth Liddle, first author of the study, says that these findings help explain one of the interesting characteristics of ADHD – that children with the condition appear able to control themselves much better when motivated to do so. "The common complaint about children with ADHD is that 'he can concentrate and control himself fine when he wants to', so some people just think the child is being naughty when he misbehaves," says Dr Liddle. "We have shown that this may be a very real difficulty for them. The off-switch for their 'internal world' seems to need a greater incentive to function properly and allow them to attend to their task."

http://www.eurekalert.org/pub_releases/2011-01/uom-bjc010511.php

Biological joints could replace artificial joints soon

University of Missouri researchers are part of team that has successfully regenerated complete shoulder joint surfaces using the patient's own cells

Artificial joint replacements can drastically change a patient's quality of life. Painful, arthritic knees, shoulders and hips can be replaced with state-of-the-art metal or ceramic implants, eliminating pain and giving a person a new lease on life. But, what if, instead of metal and plastic, doctors were able to take a patient's cells and grow an entirely new joint, replacing the old one with a fully functional biological joint? A team of University of Missouri and Columbia University researchers have found a way to create these biological joints in animals, and they believe biological joint replacements for humans aren't far away.

In a study published this fall in The Lancet, James Cook, a researcher in the MU College of Veterinary Medicine and Dept of Orthopaedic Surgery participated on a research team that created new cartilage in animals using a biological "scaffold" in the animals' joints. Cook assisted with the implant design and performed the surgeries to implant the biologic joint replacements. The study was led by Jeremy Mao of Columbia University.

The scaffold was implanted in rabbits with a surgical technique currently used for shoulder replacement in humans. The surgery removes the entire humeral head, or the ball part of the ball-and-socket shoulder joints. The scaffolds are infused with a growth factor, which encourages the host's own cells, including stem cells, to become cartilage and bone cells. The advantage to this technique is that it avoids the need to harvest and implant cells, which requires multiple surgeries.

"The device was designed with both biological and mechanical factors in mind," Cook said. "It is unique in design and composition and in how it stimulates the body's own cells. This is the first time we have seen cartilage regeneration using this type of scaffold."

The study found that the rabbits given the infused scaffolds resumed weight-bearing and functional use of their limbs faster and more consistently than those without. Four months later, cartilage had formed in the scaffolds creating a new, functional cartilage surface for the humeral head. The team observed no complications or adverse events after surgery; the new tissue regeneration was associated with excellent limb use and shoulder health, indicating the procedure is both safe and effective. Cook, who also was involved in the study design and data analysis, said the next step toward FDA approval and clinical use is to study the technique in larger animals.

"If we continue to prove the safety and efficacy of this biologic joint replacement strategy, then we can get FDA approval for use of this technology for joint replacements in people," Cook said. "We are still in the early phases of this process, but this study gives a big boost to its feasibility."

"We are continuing our concerted efforts in this arena," Cook said. "Our goal at Mizzou's Comparative Orthopaedic Laboratory is to do away with metal and plastic joints, and instead, regenerate a fully functional biologic joint for everyone who needs one. We think this is the future of orthopaedics and we hope that future is starting here and now."

http://www.eurekalert.org/pub_releases/2011-01/uonc-rps123010.php

Rifaximin provides significant relief of irritable bowel syndrome symptoms

CHAPEL HILL, N.C. – *A pair of clinical trials, conducted in part at the University of North Carolina at Chapel Hill, found that two weeks of treatment with rifaximin provides significant relief of irritable bowel syndrome symptoms including bloating, abdominal pain and loose or watery stools.*

In addition, the studies found that the benefits of treatment with rifaximin (brand name: Xifaxan) persisted for 10 weeks after patients stopped taking the broad-spectrum antibiotic, said Yehuda Ringel, MD, an associate professor of medicine in the UNC School of Medicine and a co-author of the studies, which are published in the Jan. 6, 2011 issue of the New England Journal of Medicine.

"These results support the idea that intestinal microbiota or gut bacteria may be an underlying cause of IBS, and altering gut bacteria by treatment with rifaximin appears to be an effective way of providing relief to those who suffer from IBS symptoms," Dr. Ringel said.

First author of the studies is Mark Pimentel, MD of Cedars-Sinai Medical Center in Los Angeles. The corresponding author is William P. Forbes, PharmD, of Salix Pharmaceuticals of Morrisville, N.C., which makes the drug and funded the studies.

The two studies, known as TARGET 1 and TARGET 2, were conducted in parallel from June 2008 through June 2009. In the studies, a total of 1,260 patients who had irritable bowel syndrome (IBS) without constipation were enrolled at one of 179 study sites in the U.S. and Canada. All were randomized to receive the study drug in 550 milligram doses, three times daily for two weeks, or placebo. All were then followed for an additional 10 weeks.

During the first four weeks after treatment, 40.7 percent of the patients in the rifaximin group reported adequate relief of global IBS symptoms, compared to 31.7 percent in the placebo group. Similarly, 40.2 percent in the rifaximin group had adequate relief of bloating, compared to 30.3 percent on placebo. Also, significantly more patients taking the study drug reported adequate reductions of abdominal pain and loose or watery stools.

The studies concluded that taking 550 milligram doses of rifaximin three times a day for 14 days provides better relief of IBS symptoms than placebo for up to 10 weeks after completion of therapy.

Dr. Ringel added that rifaximin, which is a semisynthetic antibiotic, has additional advantages including low systemic absorption (more than 99 percent is secreted in the stool), good antibacterial activity, low microbial resistance and a high safety profile. "These studies support the idea that gut bacteria have an important role in maintaining normal intestinal function and emphasize the need for further research on the interaction between the intestinal microbiota and the human host," Dr. Ringel said.

Additional studies currently under way at UNC are aimed at providing a better understanding of the mechanisms by which changes in the composition of the intestinal microbiota can alter intestinal function and lead to gastrointestinal symptoms, Dr. Ringel said.

http://www.eurekalert.org/pub_releases/2011-01/cumc-bef010511.php

Bacteria eyed for possible role in atherosclerosis

Enterobacter hormaechei -- normally associated with pneumonia and sepsis -- found in excised atherosclerotic plaque tissue

Dr. Emil Kozarov and a team of researchers at the Columbia University College of Dental Medicine have identified specific bacteria that may have a key role in vascular pathogenesis, specifically atherosclerosis, or what is commonly referred to as "hardening of the arteries" – the number one cause of death in the United States.

Fully understanding the role of infections in cardiovascular diseases has been challenging because researchers have previously been unable to isolate live bacteria from atherosclerotic tissue. Using tissue specimens from the Department of Surgery and the Herbert Irving Comprehensive Cancer Center at Columbia University, Dr. Kozarov and his team, however, were able to isolate plaques from a 78-year-old male who had previously suffered a heart attack. Their findings are explained in the latest Journal of Atherosclerosis and Thrombosis.

In the paper, researchers describe processing the tissue using cell cultures and genomic analysis to look for the presence of culturable bacteria. In addition, they looked at five pairs of diseased and healthy arterial tissue. The use of cell cultures aided in the isolation of the bacillus Enterobacter hormaechei from the patient's tissue. Implicated in bloodstream infections and other life-threatening conditions, the isolated bacteria were resistant to multiple antibiotics. Surprisingly, using quantitative methods, this microbe was further identified in very high numbers in diseased but not in healthy arterial tissues.

The data suggest that a chronic infection may underlie the process of atherosclerosis, an infection that can be initiated by the systemic dissemination of bacteria though different "gates" in the vascular wall – as in the case of a septic patient, through intestinal infection. The data support Dr. Kozarov's previous studies, where his team identified periodontal bacteria in carotid artery, thus pointing to tissue-destructing periodontal infections as one possible gate to the circulation.

Bacteria can gain access to the circulation through different avenues, and then penetrate the vascular walls where they can create secondary infections that have been shown to lead to atherosclerotic plaque formation, the researchers continued. "In order to test the idea that bacteria are involved in vascular pathogenesis, we must be able not only to detect bacterial DNA, but first of all to isolate the bacterial strains from the vascular wall from the patient," Dr. Kozarov said.

One specific avenue of infection the researchers studied involved bacteria getting access to the circulatory system via internalization in white blood cells (phagocytes) designed to ingest harmful foreign particles. The model that Dr. Kozarov's team was able to demonstrate showed an intermediate step where Enterobacter hormaechei is internalized by the phagocytic cells, but a step wherein bacteria are able to avoid immediate death in phagocytes. Once in circulation, Dr. Kozarov said, bacteria using this "Trojan horse" approach can persist in the organism for extended periods of time while traveling to and colonizing distant sites. This can lead to multitude of problems for the patients and for the clinicians: failure of antibiotic treatment, vascular tissue colonization and initiation of an inflammatory process, or atherosclerosis, which ultimately can lead to heart attack or stroke.

"Our findings warrant further studies of bacterial infections as a contributing factor to cardiovascular disease, and of the concept that 'bacterial persistence' in phagocytic cells likely contributes to systemic dissemination," said Dr. Kozarov, an associate professor of oral biology at the College of Dental Medicine. Dr. Jingyue Ju, co-author and director of the Columbia Center for Genome Technology & Bio-molecular Engineering, also contributed to this research, which was supported in part by a grant from the National Heart, Lung, and Blood Institute of the National Institutes of Health and by the Columbia University Section of Oral and Diagnostic Sciences.

The article appeared in Volume 18 of the Journal of Atherosclerosis and Thrombosis.

http://www.eurekalert.org/pub_releases/2011-01/nsf-wao010511.php

Widespread ancient ocean 'dead zones' challenged early life

Persistent lack of oxygen in Earth's oceans affected animal evolution

The oceans became oxygen-rich as they are today about 600 million years ago, during Earth's Late Ediacaran Period. Before that, most scientists believed until recently, the ancient oceans were relatively oxygen-poor for the preceding four billion years.

Now biogeochemists at the University of California-Riverside (UCR) have found evidence that the oceans went back to being "anoxic," or oxygen-poor, around 499 million years ago, soon after the first appearance of animals on the planet. They remained anoxic for two to four million years. The researchers suggest that such anoxic conditions may have been commonplace over a much broader interval of time.

"This work is important at many levels, from the steady growth of atmospheric oxygen in the last 600 million years, to the potential impact of oxygen level fluctuations on early evolution and diversification of life," said Enriqueta Barrera, program director in the National Science Foundation (NSF)'s Division of Earth Sciences, which funded the research.

The researchers argue that such fluctuations in the oceans' oxygen levels are the most likely explanation for what drove the explosive diversification of life forms and rapid evolutionary turnover that marked the Cambrian Period some 540 to 488 million years ago. They report in this week's issue of the journal Nature that the transition from a generally oxygen-rich ocean during the Cambrian to the fully oxygenated ocean we have today was not a simple turn of the switch, as has been widely accepted until now.

"Our research shows that the ocean fluctuated between oxygenation states 499 million years ago," said paper co-author Timothy Lyons, a UCR biogeochemist and co-author of the paper.

"Such fluctuations played a major, perhaps dominant, role in shaping the early evolution of animals on the planet by driving extinction and clearing the way for new organisms to take their place."

Oxygen is necessary for animal survival, but not for the many bacteria that thrive in and even demand life without oxygen. Understanding how the environment changed over the course of Earth's history can give scientists clues to how life evolved and flourished during the critical, very early stages of animal evolution.

"Life and the environment in which it lives are intimately linked," said Benjamin Gill, the first author of the paper, a biogeochemist at UCR, and currently a postdoctoral researcher at Harvard University.

When the ocean's oxygenation states changed rapidly in Earth's history, some organisms were not able to cope. Oceanic oxygen affects cycles of other biologically important elements such as iron, phosphorus and nitrogen.

"Disruption of these cycles is another way to drive biological crises," Gill said. "A switch to an oxygen-poor state of the ocean can cause major extinction of species."

The researchers are now working to find an explanation for why the oceans became oxygen-poor about 499 million years ago. "We have the 'effect,' but not the 'cause,'" said Gill.

"The oxygen-poor state persisted likely until the enhanced burial of organic matter, originally derived from oxygen-producing photosynthesis, resulted in the accumulation of more oxygen in the atmosphere and ocean

"As a kind of negative feedback, the abundant burial of organic material facilitated by anoxia may have bounced the ocean to a more oxygen-rich state."

Understanding past events in Earth's distant history can help refine our view of changes happening on the planet now, said Gill.

"Today, some sections of the world's oceans are becoming oxygen-poor--the Chesapeake Bay (surrounded by Maryland and Virginia) and the so-called 'dead zone' in the Gulf of Mexico are just two examples," he said.

"We know the Earth went through similar scenarios in the past. Understanding the ancient causes and consequences can provide essential clues to what the future has in store for our oceans."

The team examined the carbon, sulfur and molybdenum contents of rocks they collected from localities in the United States, Sweden, and Australia. Combined, these analyses allowed the scientists to infer the amount of oxygen present in the ocean at the time the limestones and shales were deposited.

By looking at successive rock layers, they were able to compile the biogeochemical history of the ocean.

Lyons and Gill were joined in the research by Seth Young of Indiana University, Bloomington; Lee Kump of Pennsylvania State University; Andrew Knoll of Harvard University; and Matthew Saltzman of Ohio State University.

http://www.eurekalert.org/pub_releases/2011-01/bmj-bdm010511.php

BMJ declares MMR study 'an elaborate fraud' -- autism claims likened to 'Piltdown man' hoax

Editorial: Wakefield's article linking MMR vaccine and autism was fraudulent

Today, the BMJ declares the 1998 Lancet paper that implied a link between the MMR vaccine and autism "an elaborate fraud."

Dr Fiona Godlee, BMJ Editor in Chief says "the MMR scare was based not on bad science but on a deliberate fraud" and that such "clear evidence of falsification of data should now close the door on this damaging vaccine scare." She is struck by a comparison between researcher Andrew Wakefield's fraud and Piltdown man, that great paleontological hoax that led people to believe for 40 years that the missing link between man and ape had been found. She also questions the veracity of Wakefield's other publications and calls for an investigation "to decide whether any others should be retracted."

A series of three articles starting this week reveal the true extent of the scam behind the scare. The series is based on interviews, documents and data, collected during seven years of inquiries by award-winning investigative journalist Brian Deer.

Thanks to the recent publication of the General Medical Council's six million word transcript, the BMJ was able to peer-review and check Deer's findings and confirm extensive falsification in the Lancet paper.

In an editorial, Dr Godlee, together with deputy BMJ editor Jane Smith, and leading paediatrician and associate BMJ editor Harvey Marcovitch, conclude that there is "no doubt" that it was Wakefield who perpetrated this fraud. They say: "A great deal of thought and effort must have gone into drafting the paper to achieve the results he wanted: the discrepancies all led in one direction; misreporting was gross."

Yet he has repeatedly denied doing anything wrong at all, they add. "Instead, although now disgraced and stripped of his clinical and academic credentials, he continues to push his views. Meanwhile the damage to public health continues."

"Science is based on trust," concludes Dr Godlee. "Such a breach of trust is deeply shocking. And even though almost certainly rare on this scale, it raises important questions about how this could happen, what could have been done to uncover it earlier, what further inquiry is now needed, and what can be done to prevent something like this happening again."

The BMJ will explore these and other questions over the next two weeks.

http://www.newscientist.com/article/dn19915-stem-cells-hold-key-to-cure-for-baldness.html

Stem cells hold key to cure for baldness

*** 14:47 05 January 2011 by Andy Coghlan**

Patches of hair may be gone in many men, but the stem cells that make hair are still there. This unexpected finding is raising hopes of a cure for baldness.

By comparing bald and hairy patches in scalp samples from 54 men undergoing restoration treatments, George Cotsarelis at the University of Pennsylvania School of Medicine in Philadelphia and colleagues discovered that although both had similar numbers of stem cells, most of those in the bald patches fail to develop to the next stage.

In samples from the same individuals, stem cells that had matured into so-called "progenitor cells" were 10 to 100 times as abundant on hairy patches as on bald ones, suggesting they are the key to hair growth.

If a way can be found to reawaken the stem cells, it could provide a shortcut to new hair for millions of men with male-pattern baldness.

"The fact that [the stem cells] are there at all is pretty exciting, and lowers the bar for treatment," says Cotsarelis. "I'm generally very pessimistic about the prospects for treatment, so we were surprised to find such a large and normal complement of stem cells in the bald patches."

Hair today…

Now the team is investigating why some of the stem cells become dormant while others remain active. "It could be the lack of a stimulus, or too much of an inhibitor on different parts of the scalp," says Cotsarelis. "We're working on that now, and have some leads, but this is a very early step in development of a treatment."

Encouragingly, the team reports in the same paper that mouse progenitor cells were capable of regenerating entire hair follicles. This suggests that the same might be possible in people, if progenitor cells can be made from reawakened stem cells.

In earlier experiments, Cotsarelis also showed that in mice, transplanted follicular stem cells were able to regenerate hair.

One possibility would be to take stem cells from balding men, multiply these into progenitor cells, and then return them to the scalp. Another is to find a chemical signal that reawakens the stem cells, so it could simply be rubbed onto the bald areas of the scalp.

Cotsarelis says that although the finding is in men, it may be also be applicable to women. "Some 30 per cent of women have some degree of female-pattern hair loss by the age of 50," he says.

Journal reference: The Journal of Clinical Investigation, DOI: 10.1172/JCI44478

http://www.bbc.co.uk/news/science-environment-12120430

Wreckage is from 'pristine star'

By Jonathan Amos Science correspondent, BBC News

UK and US scientists have found the remnants of a star that exploded more than 13 billion years ago.

It would most probably have been one of the very first stars to shine in the Universe, they say. All that is left of this pioneer is the gas cloud it threw out into space when it blew itself apart. It was identified when its contents were illuminated by the brilliant light coming from the surroundings of a distant black hole.

The cloud's atoms occur in abundances that are quite unlike that found in the nearby cosmos today and are more what one would expect from stars that were originally made only of hydrogen and helium.

The research required the observations of two of the world's most powerful telescopes - the Keck facility in Hawaii and the Very Large Telescope in Chile.

It has been written up in the Monthly Notices of the Royal Astronomical Society.

'Holy Grail'

The study is said to provide fresh insight on key events in the earliest stages of the Universe, in particular it offers some new details on the endings of the so-called "Dark Ages", the period before the first stars formed.

"It's a period we know very little about, but the Universe at that time was a rather boring place, just filled with hydrogen and helium gas and not much else; there was no light - that's why it's called the Dark Ages," explained Professor Max Pettini at Cambridge's Institute of Astronomy, UK.

"And then somehow from that initial state it changed into this wonderful mix of stars and planets and galaxies that we see around us today."

Working on the Keck and VLT, Pettini's team probed the composition of distant clouds of gas known as "damped Lyman alpha systems". They did this using the light provided by quasars, extremely luminous galaxies whose brilliance is powered by a mighty black hole at their core.

The investigation identified one particular DLA that had a unique chemical signature - one where the ratio of carbon to iron atoms was 35 times greater than what can be measured in the Sun. It enabled the group to infer that the gas was released by a star 25 times more massive than our star and which originally consisted of only hydrogen and helium - exactly the type of star expected to have ended the Dark Ages.

"The first stars have been a bit like the Holy Grail for astronomers," said Professor Pettini, who led the research with PhD student Ryan Cooke.

"We think that they all lived very short and furious lives. They are all dead now, and there is no way for us even with the most powerful telescopes to observe them directly. So, what we have found is the remnants of one of these first stars to form in the Universe, and the elements carbon, oxygen and iron and pristine gas in a mix that has never been seen before."

Next generation

The results of the study, conducted with partners at the California Institute of Technology (Caltech), will feed into the theoretical framework scientists are building to describe this early epoch.

Scientists believe the very first stars to shine in the Universe were hot giants that fundamentally changed the cosmic environment.

Not only did they seed the cosmos with the heavier elements needed to make planets, but their intense ultraviolet radiation also "fried" the neutral gas around them. The consequences of this latter process are evident even today in the diffuse plasma that fills the space between the galaxies.

"This was only a first step; it was like finding some fossil," Professor Pettini told the BBC.

"Now that we have discovered how to find such fossil evidence, we are much better placed for finding other examples of these particular clouds in the distant Universe that hold the special clues; and then progress from there and really breakthrough what has been called the last frontier in observational astronomy."

Finding out any information about this early period, just a few hundred million years after the Big Bang, is a tour de force because it takes existing technology such as the Keck and VLT right to the limits of their capability.

Only with the next generation of observatories - super telescopes with mirrors tens of metres in diameter - can scientists hope to probe more widely into the circumstances that ended the Dark Ages.

The European Southern Observatory Organisation, which runs the VLT, plans to build a telescope with a 42m-wide mirror, and Caltech is involved in a project to build one with a mirror that is 30m in diameter.

http://www.physorg.com/news/2011-01-advance-mri-scans-faster.html

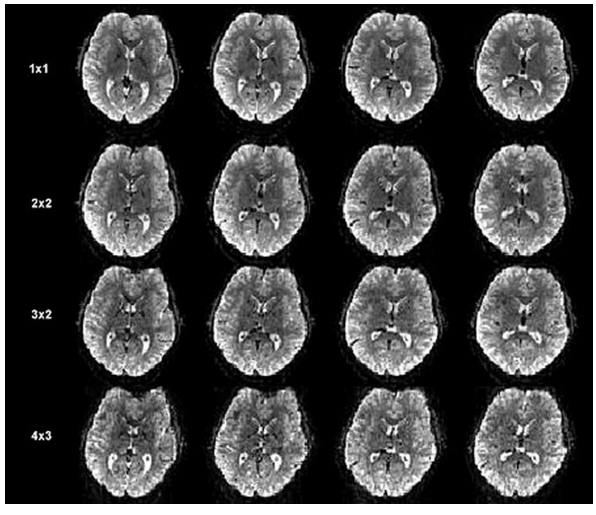
Advance makes MRI scans more than seven times faster

(PhysOrg.com) -- ***An international team of physicists and neuroscientists has reported a breakthrough in magnetic resonance imaging that allows brain scans more than seven times faster than currently possible.***

In a paper that appeared Dec. 20 in the journal PLoS ONE, a University of California, Berkeley, physicist and colleagues from the University of Minnesota and Oxford University in the United Kingdom describe two improvements that allow full three-dimensional brain scans in less than half a second, instead of the typical 2 to 3 seconds.

"When we made the first images, it was unbelievable how fast we were going," said first author David Feinberg, a physicist and adjunct professor in UC Berkeley's Helen Wills Neuroscience Institute and president of the company Advanced MRI Technologies in Sebastopol, Calif. "It was like stepping out of a prop plane into a jet plane. It was that magnitude of difference."

For neuroscience, in particular, fast scans are critical for capturing the dynamic activity in the brain.



fMRI brain scans without the new acceleration techniques (top row) and with increasing numbers of multiplexings and slice accelerations. The bottom row was obtained seven times faster than the top row, although all show similar resolution. Only 4 of the 60 slices of a full, 3-D brain scan are shown. **(David Feinberg/UC Berkeley)**

"When a functional MRI study of the brain is performed, about 30 to 60 images covering the entire 3-D brain are repeated hundreds of times like the frames of a movie but, with fMRI, a 3-D movie," Feinberg said. "By multiplexing the image acquisition for higher speed, a higher frame rate is achieved for more information in a shorter period of time."

"The brain is a moving target, so the more refined you can sample this activity, the better understanding we will have of the real dynamics of what's going on here," added Dr. Marc Raichle, a professor of radiology, neurology, neurobiology, biomedical engineering and psychology at Washington University in St. Louis who has followed Feinberg's work.

In addition to broadly advancing the field of neural-imaging, the discovery will have an immediate impact on the Human Connectome Project, funded last year by the National Institutes of Health (NIH) to map the connections of the human brain through functional MRI (fMRI) and structural MRI scans of 1,200 healthy adults.

"At the time we submitted our grant proposal for the Human Connectome Project, we had aspirations of acquiring better quality data from our study participants, so this discovery is a tremendous step in helping us accomplish the goals of the project," said Dr. David Van Essen, a neurobiologist at Washington University and co-leader of the project. "It's vital that we get the highest quality imaging data possible, so we can infer accurately the brain's circuitry – how connections are established, and how they perform."

Advance speeds up MRI scans

The new technique accelerates diffusion MRI as well. The colored tracks show the direction of nerve fiber bundles, providing a 3-D image of the axonal pathways in the white matter (cortex) of a resting human brain. A normal structural cross sectional image of the brain bisects the diffusion 3-D fibertrack image. The entire 3-D image was scanned in 8.5 minutes instead of 30 minutes. (David Feinberg)

The faster scans are made possible by combining two technical improvements invented in the past decade that separately boosted scanning speeds two to four times over what was already the fastest MRI technique, echo planar imaging (EPI). Physical limitations of each method prevented further speed improvements, "but together their image accelerations are multiplied," Feinberg said. The team can now obtain brain scans substantially faster than the time reductions reported in their paper and many times faster than the capabilities of today's machines.

Probing the brain with radio waves

Magnetic resonance imaging works by using a magnetic field and radio waves to probe the environment of hydrogen atoms in water molecules in the body. Because hydrogen atoms in blood, for example, respond differently than atoms in bone or tissue, computers can reconstruct the body's interior landscape without the use of penetrating X-rays.

Nearly 20 years ago, however, a new type of MRI called functional MRI (fMRI) was developed to highlight areas of the brain using oxygen, and thus presumably engaged in neuronal activity, such as thinking .Using echo planar imaging (EPI), fMRI vividly distinguishes oxygenated blood funneling into working areas of the brain from deoxygenated blood in less active areas.

As with standard MRI, fMRI machines create magnetic fields that vary slightly throughout the brain, providing a different magnetic environment for hydrogen atoms in different areas. The differing magnetic field strengths make the spin of each hydrogen atom precess at different rates, so that when a pulse of radio waves is focused on the head, the atoms respond differently depending on location and on their particular environment. Those that absorb radio energy and then release the energy are detected by magnetic coils surrounding the head, and these signals, or "echoes," are used to produce an image of the brain.

With EPI, a single pulse of radio waves is used to excite the hydrogen atoms, but the magnetic fields are rapidly reversed several times to elicit about 50 to 100 echoes before the atoms settle down. The multiple echoes provide a high-resolution picture of the brain.

In 2002, Feinberg proposed using a sequence of two radio pulses to obtain two times the information in the same amount of time. Dubbed simultaneous image refocusing (SIR) EPI, it has proved useful in fMRI and for 3-D imaging of neuronal axonal fiber tracks, though the improvement in scanning speed is limited because with a train of more than four times as many echoes, the signal decays and the image resolution drops.

Another acceleration improvement, multiband excitation of several slices using multiple coil detection, was developed in the U.K. at about the same time by David Larkmann for spinal imaging. The technique was recently pioneered for fMRI by Steen Moeller and colleagues at the University of Minnesota. This technique, too, had limitations, primarily because the multiple coils are relatively widely spaced and cannot differentiate very closely spaced images.

In collaboration with Essa Yacoub, senior author on the paper, and Kamil Ugurbil, director of the University of Minnesota's Center for Magnetic Resonance Research and co-leader of the Human Connectome Project, Feinberg combined these techniques to get significantly greater acceleration than either technique alone while maintaining the same image resolution.

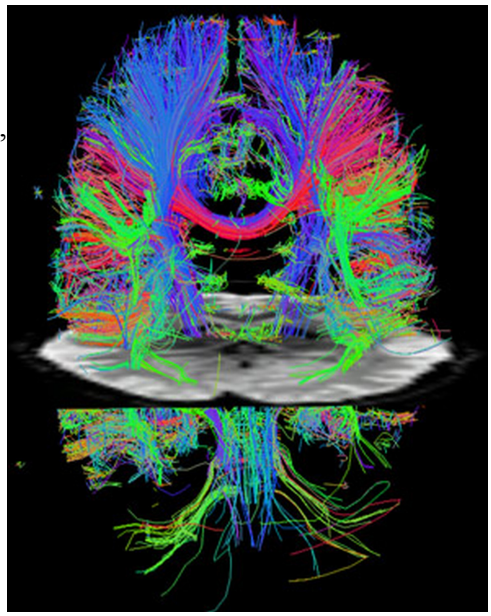
"With the two methods multiplexed, 10, 12 or 16 images the product of their two acceleration factors were read out in one echo train instead of one image," Feinberg said.

fMRI moving closer to speed of EEG

The ability to scan the brain in under 400 milliseconds moves fMRI closer to electroencephalography (EEG) for capturing very rapid sequences of events in the brain.

"Other techniques which capture signals derived from neuronal activity, EEG or MEG, have much higher temporal resolution; hundred microsecond neuronal changes. But MRI has always been very slow, with 2 second temporal resolution," Feinberg said. "Now MRI is getting down to a few hundred milliseconds to scan the entire brain, and we are beginning to see neuronal network dynamics with the high spatial resolution of MRI."

The development will impact general fMRI as well as diffusion imaging of axonal fibers in the brain, both of which are needed to achieve the main goal of the Human Connectome Project. Diffusion imaging reveals the axonal fiber networks that are the main nerve connections between areas of the brain, while fMRI shows which areas of the brain are functionally connected, that is, which areas are active together or sequentially during various activities.



The new technique accelerates diffusion MRI as well. The colored tracks show the direction of nerve fiber bundles, providing a 3-D image of the axonal pathways in the white matter (cortex) of a resting human brain. A normal structural cross sectional image of the brain bisects the diffusion 3-D fibertrack image. The entire 3-D image was scanned in 8.5 minutes instead of 30 minutes. **(David Feinberg)**

"While it simply is not possible to show the billions of synaptic connections in the live human brain, the hope is that understanding patterns of how the normal brain is functionally interacting and structurally connected will lead to insights about diseases that involve miswiring in the brain," Feinberg said.

"We suspect several neurologic and psychiatric disorders, such as autism and schizophrenia, could be brain connectivity disorders, but we don't know what normal connectivity is," Feinberg added. "Although the fMRI and neuronal fiber images do not have the resolution of an electron microscope, the MRI derived Connectome reveals the live human brain and can be combined with genetic and environmental information to identify individual differences in brain circuitry."

Raichle, a collaborator in the NIH Human Connectome project, is one of the pioneers of "resting state" MRI, in which brain scans are taken of patients not involved in any specific task. He believes that the ongoing spontaneous activity discovered during such scans will tell us about how the brain remains flexible and maintains a degree of homeostatis so that "you know who you are."

"Being able to sample this ongoing activity at increasing temporal fidelity and precision becomes really important for understanding how the brain is doing this," Raichle said. "David is superclever at this kind of technical stuff, and I have been cheering him along, saying that the faster we can go, the better we can understand the brain's spontaneous activity." *Provided by University of California - Berkeley*

http://www.eurekalert.org/pub_releases/2011-01/pu-psc010611.php

Princeton scientists construct synthetic proteins that sustain life

In a groundbreaking achievement that could help scientists "build" new biological systems, Princeton University scientists have constructed for the first time artificial proteins that enable the growth of living cells.

The team of researchers created genetic sequences never before seen in nature, and the scientists showed that they can produce substances that sustain life in cells almost as readily as proteins produced by nature's own toolkit.

"What we have here are molecular machines that function quite well within a living organism even though they were designed from scratch and expressed from artificial genes," said Michael Hecht, a professor of chemistry at Princeton, who led the research. "This tells us that the molecular parts kit for life need not be limited to parts -- genes and proteins -- that already exist in nature."

The work, Hecht said, represents a significant advance in synthetic biology, an emerging area of research in which scientists work to design and fabricate biological components and systems that do not already exist in the natural world. One of the field's goals is to develop an entirely artificial genome composed of unique patterns of chemicals.

"Our work suggests," Hecht said, "that the construction of artificial genomes capable of sustaining cell life may be within reach."

Nearly all previous work in synthetic biology has focused on reorganizing parts drawn from natural organisms. In contrast, Hecht said, the results described by the team show that biological functions can be provided by macromolecules that were not borrowed from nature, but designed in the laboratory.

Although scientists have shown previously that proteins can be designed to fold and, in some cases, catalyze reactions, the Princeton team's work represents a new frontier in creating these synthetic proteins.

The research, which Hecht conducted with three former Princeton students and a former postdoctoral fellow, is described in a report published online Jan. 4 in the journal Public Library of Science ONE.

Hecht and the students in his lab study the relationship between biological processes on the molecular scale and processes at work on a larger magnitude.

For example, he is studying how the errant folding of proteins in the brain can lead to Alzheimer's disease, and is involved in a search for compounds to thwart that process. In work that relates to the new paper, Hecht and his students also are interested in learning what processes drive the routine folding of proteins on a basic level -- as proteins need to fold in order to function -- and why certain key sequences have evolved to be central to existence. Proteins are the workhorses of organisms, produced from instructions encoded into cellular DNA.

The identity of any given protein is dictated by a unique sequence of 20 chemicals known as amino acids. If the different amino acids can be viewed as letters of an alphabet, each protein sequence constitutes its own unique "sentence."

And, if a protein is 100 amino acids long (most proteins are even longer), there are an astronomically large number of possibilities of different protein sequences, Hecht said. At the heart of his team's research was to question how there are only about 100,000 different proteins produced in the human body, when there is a potential for so many more. They wondered, are these particular proteins somehow special? Or might others work equally well, even though evolution has not yet had a chance to sample them?

Hecht and his research group set about to create artificial proteins encoded by genetic sequences not seen in nature. They produced about 1 million amino acid sequences that were designed to fold into stable three-dimensional structures.

"What I believe is most intriguing about our work is that the information encoded in these artificial genes is completely novel -- it does not come from, nor is it significantly related to, information encoded by natural genes, and yet the end result is a living, functional microbe," said Michael Fisher, a co-author of the paper who earned his Ph.D. at Princeton in 2010 and is now a postdoctoral fellow at the University of California-Berkeley. "It is perhaps analogous to taking a sentence, coming up with brand new words, testing if any of our new words can take the place of any of the original words in the sentence, and finding that in some cases, the sentence retains virtually the same meaning while incorporating brand new words."

Once the team had created this new library of artificial proteins, they inserted those proteins into various mutant strains of bacteria in which certain natural genes previously had been deleted. The deleted natural genes are required for survival under a given set of conditions, including a limited food supply.

Under these harsh conditions, the mutant strains of bacteria died -- unless they acquired a life-sustaining novel protein from Hecht's collection. This was significant because formation of a bacterial colony under these selective conditions could occur only if a protein in the collection had the capacity to sustain the growth of living cells.

In a series of experiments exploring the role of differing proteins, the scientists showed that several different strains of bacteria that should have died were rescued by novel proteins designed in the laboratory. "These artificial proteins bear no relation to any known biological sequences, yet they sustained life," Hecht said.

Added Kara McKinley, also a co-author and a 2010 Princeton graduate who is now a Ph.D. student at the Massachusetts Institute of Technology: "This is an exciting result, because it shows that unnatural proteins can sustain a natural system, and that such proteins can be found at relatively high frequency in a library designed only for structure."

In addition to Hecht, Fisher and McKinley, other authors on the paper include Luke Bradley, a former postdoctoral fellow in Hecht's lab who is now an assistant professor at the University of Kentucky, and Sara Viola, a 2008 Princeton graduate who is now a medical student at Columbia University.

The research was funded by the National Science Foundation.

Title: "De Novo Designed Proteins from a Library of Artificial Sequences Function in Escherichia Coli and Enable Cell Growth" Journal: Public Library of Science ONE

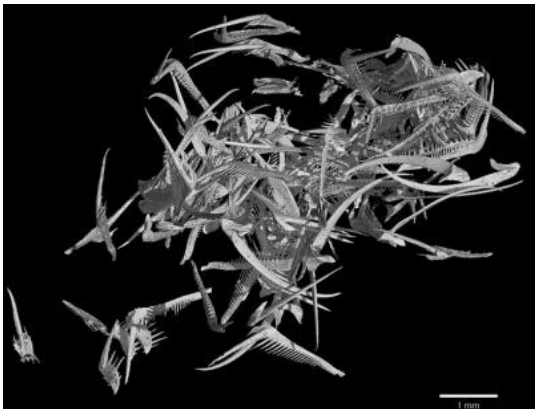
http://www.eurekalert.org/pub_releases/2011-01/esrf-ado122810.php

Ammonites' last meal: New light on past marine food chains

Scientists have discovered direct evidence of the diet of one of the most important group of ammonites, distant relatives of squids, octopuses and cuttlefishes.

The discovery may bring a new insight on why they became extinct 65.5 million years ago, at the end of the Cretaceous.

Ammonites are among the world's most well known fossils but until now, there has been no experimental evidence of their place in the food chain. Using synchrotron X-rays, a Franco-American team of scientists led by Isabelle Kruta has discovered exceptionally preserved mouth organs of ammonites, along with the remains of a meal that show that these ammonites dined on plankton. Plankton was largely destroyed in the wake of the same asteroid impact that led to the demise of the dinosaurs and other species. After losing their source of food, ammonites and many other marine groups could not survive this cataclysmic event. The findings are published this week in Science.



This is a 3-D reconstruction of the radula (a tongue-like anatomical structure used by molluscs for feeding) of one of the Baculites fossils (AMNH 6653). **Credit: I. Kruta/MNHN**

Ammonites are extinct relatives of the squid and octopus. The Nautilus, a present-day marine invertebrate, is similar in appearance to many ammonites but is a more distant relative. Ammonites appeared about 400 million years ago (the Early Devonian) and experienced a population explosion in the early Jurassic. In fact, ammonites became such an abundant and diverse part of the marine fauna that they are used by paleontologists as classic "index" fossils to determine the relative ages of marine Mesozoic rocks in which they are found.

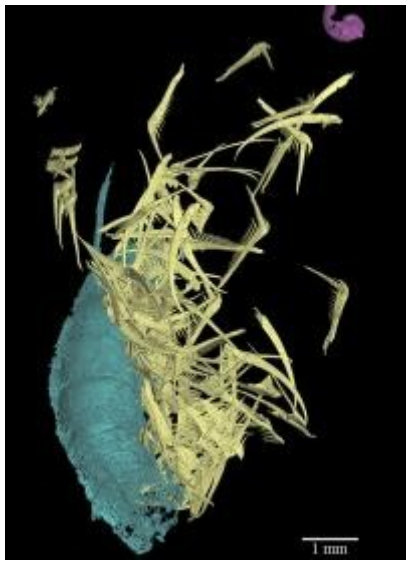
The team of researchers, led by Isabelle Kruta (MNHN, CNRS, UPMC), used the ESRF to perform X-ray scans of exceptional quality of Baculites fossils found on AMNH expeditions to the Great Plains in the United States. Results suggests that the large group of ammonites to which Baculites belongs, had jaws and radula (a kind of tongue covered with teeth) adapted for eating small prey floating in the water.

The study used synchrotron X-ray microtomography to check the presence, and then digitally reconstruct the mouths of three fossils found in South Dakota. The three-dimensional reconstructions are of such high quality that the jaws and teeth are revealed in their complete form. In addition, one specimen has a tiny snail and three tiny crustaceans in its mouth, one of them having been cut into two parts. Because these planktonic fossils are not found anywhere else on the specimen, the team thinks that the specimen died while eating its last meal rather than being scavenged by these organisms after death.

"I was astonished when I saw the teeth for the first time, and when I found the tiny plankton in the mouth," Isabelle Kruta (MNHN). "For the first time we were able to observe the delicacy of these exceptionally well preserved structures and use high quality details to obtain information on the ecology of these enigmatic animals."

"When you take into consideration the large lower jaws of ammonites in combination with this new information about their teeth, you realize that these animals must have been feeding in a different way from modern carrion-eating Nautilus," says Neil Landman (AMNH). "Ammonites have a surprisingly large lower jaw with slender teeth, but the effect is opposite to that of the wolf threatening to eat Little Red Riding Hood. Here, the bigger mouth facilitates feeding on smaller prey."

"X-ray synchrotron microtomography is currently the most sensitive technique for non-destructive investigations of internal structures of fossils. It started ten years ago with primate teeth, but is now widely applied in paleontology," says Paul Tafforeau (ESRF). " We made a first test on one of the Ammonite specimens after a test with a conventional scanner failed, and the results were so impressive that we scanned all the other available samples, discovering nearly each time a radula and for one of them, many other structures."



This is a 3-D reconstruction of the radula (tongue-like anatomical structure of mollusks for feeding) of a Baculites fossil. Teeth are depicted in yellow and the fragments of the fossil's last meal, caught between the jaws, in blue (for a crustacean) and pink (for a snail), respectively. **Credit: I. Kruta/MNHN**

Ammonite jaws lie just inside the body chamber. The research team's new scans of Baculites, a straight ammonite found world-wide, confirms older research that ammonites had multiple cusps on their radula teeth. The radula can now be seen in exquisite detail: the tallest cusp is 2 mm high, tooth shape varies from saber to comb-like, and teeth are very slender. The jaw is typical of this group of ammonites (the aptychophorans) in that the lower jaw is larger than the upper jaw and consists of two halves separated along a midline.

Until recently, the role of ammonites in the marine food web was unknown, although some clues were provided by Landman and colleagues on the shape of the jaw, as well as a 1992 paper by Russian scientists that reconstructed some of the internal structures by slicing fossils..

"The plankton in the Baculites jaws is the first direct evidence of how these uncoiled ammonites fed. This helps in understanding their evolutionary success in the Cretaceous." says Fabrizio Cecca (UPMC).

"Our research suggests several things. First, the radiation of aptychophoran ammonites might be associated with the radiation of plankton during the Early Jurassic," say Landman. "In addition, plankton were severely hit at the Cretaceous-Tertiary boundary, and the loss of their food source probably contributed to the extinction of ammonites. This research has implications for understanding carbon cycling during this time".

Isabelle Rouget (UPMC) agrees, adding that "we now realize that ammonites occupied a different niche in the food chain than we previously thought."

This research was supported by the Centre National de la Recherche Scientifique (CNRS, France), the Museum National d ´Histoire Naturelle (MNHN, Paris, France) , the Université Pierre et Marie Curie (UPMC, Paris, France), the American Museum of Natural History (AMNH, New York, USA) and the European Synchrotron Radiation Facility (ESRF, Grenoble, France).

http://www.eurekalert.org/pub_releases/2011-01/uoc--ns010511.php

Neural stem cells maintain high levels of reactive oxygen species, UCLA study finds

Findings may have implications for repairing brain injury, studying diseases such as autism and brain cancer

For years, the majority of research on reactive oxygen species (ROS) – ions or very small molecules that include free radicals – has focused on how they damage cell structure and their potential link to stroke, cardiovascular disease and other illnesses.

However, researchers at the Eli and Edythe Broad Center of Regenerative Medicine and Stem Cell Research have shown for the first time that neural stem cells, the cells that give rise to neurons, maintain high levels of ROS to help regulate normal self-renewal and differentiation.

The findings, published in the Jan. 7, 2011 issue of the journal Cell Stem Cell, may have significant implications for brain repair and abnormal brain development.

"Everyone thinks of ROS as things that kill cells, and they do," said Dr. Harley Kornblum, a professor at the Intellectual and Developmental Disabilities Research Center in the Semel Institute of Neuroscience and Behavior and senior author of the study. "Stem cells generally have been thought to maintain low levels of ROS to protect against damage, so our findings were surprising and we hope to be able to exploit this to promote neural repair and explore diseases such as autism and brain cancer."

The study also found that the neural stem cells were highly responsive to ROS stimulation, which increased their growth and differentiation. Conversely, diminishing cellular levels of ROS in the neural stem cells interfered with normal cell function in mice and in human and mouse cell lines.

"It wasn't just that neural stem cells maintained high ROS levels" said Janel Le Belle, an assistant researcher in Kornblum's lab and lead author of the study. "Changes in cellular ROS can affect how the stem cells function. This study could lead to an understanding of how elevated ROS due to environmental factors might play a role in brain overgrowth, such as occurs in some cases of autism."

The body has a system to make ROS when it needs it. Some cells, such as immune cells, surround bacteria or viruses and use ROS to kill the invading microbes. Outside influences such as stress and environmental factors such as exposure to radiation can increase ROS levels in cells.

Although ROS is produced by all cells in a passive manner as a by-product of normal cell metabolism, some cells also produce ROS in a directed manner using ROS-producing enzymes like NADPH oxidase (NOX). NOX-generated ROS can act as second messengers in tightly controlled signal transduction pathways for many growth and trophic factors. However, too much ROS damages and ultimately kills cells, so finding the correct balance is vital, Kornblum said. And in fact, when the neural stem cells in the study were given too much ROS, they did die.

Kornblum and his team also found that the ROS-mediated stem cell self-renewal and differentiation of these cells into neurons depended on a cell signaling pathway called PI3K/Akt, which is known to be involved in cellular functions such as cell growth, proliferation, differentiation, motility and survival. NOX-generated ROS

affect PI3K/Akt signaling by causing the inactivation of the PTEN protein, an important tumor suppressor and negative regulator of the pathway, by oxidizing a cysteine residue in the protein, which inactivates its function.

Kornblum and his collaborators at UCLA, including Dr. Hong Wu, a professor of molecular and medical pharmacology and a co-author of the study, have been studying the PI3K pathway for years. The pathway is activated in some diseases, for example a subset of autism cases and in tuberous sclerosis, a rare, multi-system genetic disease that causes non-malignant tumors to grow in the brain and in other vital organs. The pathway also can be activated in certain cancers.

"One of our hypotheses is that in these disease states, for instance in autism, that in those with a genetic predisposition to PI3K activation, exposure to a stressor that increases ROS levels can exacerbate the predisposition, perhaps promoting the disease," Kornblum said.

In brain tumors, if the pathway gets activated in cells already susceptible to becoming cancerous, it may promote the proliferation of brain tumor cells or the propagation of brain tumors. Blocking the pathway, Kornblum said, may be one way to interrupt the malignant process.

Going forward, Kornblum and his team will seek to determine whether brain cancer cells use elevated ROS and the PI3K pathway to promote their own growth. Le Belle said they will also test whether elevated ROS during brain development can contribute to brain overgrowth in Autism. Additionally, the team will test to see if they can exploit the ROS-activated pathway to promote brain repair, for example, increasing the production of new neurons to replace damaged or dead neurons.

The five-year study was funded by the National Institutes of Mental Health, Cure Autism Now, Autism Speaks, the UCLA CART, the Dr. Miriam and Sheldon G. Adelson Medical Research Foundation and the Koh Charitable Foundation.

http://www.eurekalert.org/pub_releases/2011-01/wios-wis010611.php

Weizmann Institute scientists discover: A chemical signal in human tears

Emotional crying is a universal, uniquely human behavior. When we cry, we clearly send all sorts of emotional signals. In a paper published online today in Science Express, scientists at the Weizmann Institute have demonstrated that some of these signals are chemically encoded in the tears themselves.

Specifically, they found that merely sniffing a woman's tears – even when the crying woman is not present -- reduces sexual arousal in men.

Humans, like most animals, expel various compounds in body fluids that give off subtle messages to other members of the species. A number of studies in recent years, for instance, have found that substances in human sweat can carry a surprising range of emotional and other signals to those who smell them.

But tears are odorless. In fact, in a first experiment led by Shani Gelstein, Yaara Yeshurun and their colleagues in the lab of Prof. Noam Sobel in the Weizmann Institute's Neurobiology Department, the researchers first obtained emotional tears from female volunteers watching sad movies in a secluded room and then tested whether men could discriminate the smell of these tears from that of saline. The men could not.

In a second experiment, male volunteers sniffed either tears or a control saline solution, and then had these applied under their nostrils on a pad while they made various judgments regarding images of women's faces on a computer screen. The next day, the test was repeated -- the men who were previously exposed to tears getting saline and vice versa. The tests were double blinded, meaning neither the men nor the researchers performing the trials knew what was on the pads. The researchers found that sniffing tears did not influence the men's estimates of sadness or empathy expressed in the faces. To their surprise, however, sniffing tears negatively affected the sex appeal attributed to the faces.

To further explore the finding, male volunteers watched emotional movies after similarly sniffing tears or saline. Throughout the movies, participants were asked to provide self-ratings of mood as they were being monitored for such physiological measures of arousal as skin temperature, heart rate, etc. Self-ratings showed that the subjects' emotional responses to sad movies were no more negative when exposed to women's tears, and the men "smelling" tears showed no more empathy. They did, however, rate their sexual arousal a bit lower. The physiological measures, however, told a clearer story. These revealed a pronounced tear-induced drop in physiological measures of arousal, including a significant dip in testosterone – a hormone related to sexual arousal.

Finally, in a fourth trial, Sobel and his team repeated the previous experiment within an fMRI machine that allowed them to measure brain activity. The scans revealed a significant reduction in activity levels in brain areas associated with sexual arousal after the subjects had sniffed tears.

Sobel: "This study raises many interesting questions. What is the chemical involved? Do different kinds of emotional situations send different tear-encoded signals? Are women's tears different from, say, men's tears?

Children's tears? This study reinforces the idea that human chemical signals – even ones we're not conscious of – affect the behavior of others."

Human emotional crying was especially puzzling to Charles Darwin, who identified functional antecedents to most emotional displays -- for example, the tightening of the mouth in disgust, which he thought originated as a response to tasting spoiled food. But the original purpose of emotional tears eluded him. The current study has offered an answer to this riddle: Tears may serve as a chemosignal. Sobel points out that some rodent tears are known to contain such chemical signals. "The uniquely human behavior of emotional tearing may not be so uniquely human after all," he says.

The work was authored by Shani Gelstein, Yaara Yeshurun, Liron Rozenkrantz, Sagit Shushan, Idan Frumin, Yehudah Roth and Noam Sobel, was conducted in collaboration with the Edith Wolfson Medical Center, Holon.
Prof. Noam Sobel's research is supported by the James S. McDonnell Foundation 21st Century Science Scholar in Understanding Human Cognition Program; the Minerva Foundation; the European Research Council; and Regina Wachter, NY.

http://www.eurekalert.org/pub_releases/2011-01/uof-uso010611.php

UF study of lice DNA shows humans first wore clothes 170,000 years ago

GAINESVILLE, Fla. — *A new University of Florida study following the evolution of lice shows modern humans started wearing clothes about 170,000 years ago, a technology which enabled them to successfully migrate out of Africa.*

Principal investigator David Reed, associate curator of mammals at the Florida Museum of Natural History on the UF campus, studies lice in modern humans to better understand human evolution and migration patterns. His latest five-year study used DNA sequencing to calculate when clothing lice first began to diverge genetically from human head lice. Funded by the National Science Foundation, the study is available online and appears in this month's print edition of Molecular Biology and Evolution.

"We wanted to find another method for pinpointing when humans might have first started wearing clothing," Reed said. "Because they are so well adapted to clothing, we know that body lice or clothing lice almost certainly didn't exist until clothing came about in humans."

The data shows modern humans started wearing clothes about 70,000 years before migrating into colder climates and higher latitudes, which began about 100,000 years ago. This date would be virtually impossible to determine using archaeological data because early clothing would not survive in archaeological sites.

The study also shows humans started wearing clothes well after they lost body hair, which genetic skin-coloration research pinpoints at about 1 million years ago, meaning humans spent a considerable amount of time without body hair and without clothing, Reed said.

"It's interesting to think humans were able to survive in Africa for hundreds of thousands of years without clothing and without body hair, and that it wasn't until they had clothing that modern humans were then moving out of Africa into other parts of the world," Reed said.

Lice are studied because unlike most other parasites, they are stranded on lineages of hosts over long periods of evolutionary time. The relationship allows scientists to learn about evolutionary changes in the host based on changes in the parasite.

Applying unique data sets from lice to human evolution has only developed within the last 20 years, and provides information that could be used in medicine, evolutionary biology, ecology or any number of fields, Reed said. "It gives the opportunity to study host-switching and invading new hosts — behaviors seen in emerging infectious diseases that affect humans," Reed said.

A study of clothing lice in 2003 led by Mark Stoneking, a geneticist at the Max Planck Institute in Leipzig, Germany, estimated humans first began wearing clothes about 107,000 years ago. But the UF research includes new data and calculation methods better suited for the question.

"The new result from this lice study is an unexpectedly early date for clothing, much older than the earliest solid archaeological evidence, but it makes sense," said Ian Gilligan, lecturer in the School of Archaeology and Anthropology at The Australian National University. "It means modern humans probably started wearing clothes on a regular basis to keep warm when they were first exposed to Ice Age conditions."

The last Ice Age occurred about 120,000 years ago, but the study's date suggests humans started wearing clothes in the preceding Ice Age 180,000 years ago, according to temperature estimates from ice core studies, Gilligan said. Modern humans first appeared about 200,000 years ago.

Because archaic hominins did not leave descendants of clothing lice for sampling, the study does not explore the possibility archaic hominins outside of Africa were clothed in some fashion 800,000 years ago. But while archaic humans were able to survive for many generations outside Africa, only modern humans persisted there until the present.

"The things that may have made us much more successful in that endeavor hundreds of thousands of years later were technologies like the controlled use of fire, the ability to use clothing, new hunting strategies and new stone tools," Reed said.

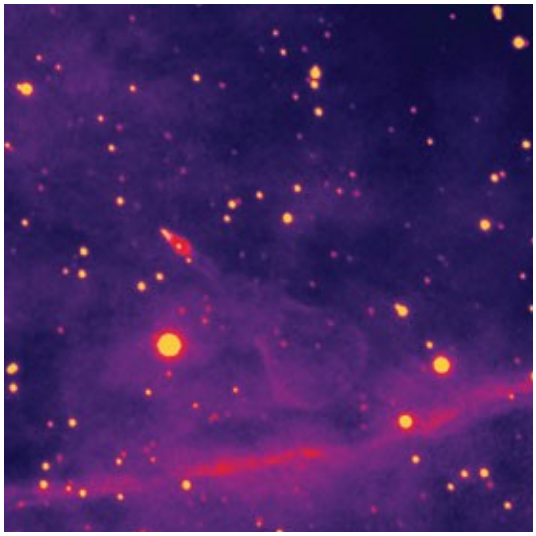
Study co-authors were Melissa Toups of Indiana University and Andrew Kitchen of The Pennsylvania State University, both previously with UF. Co-author Jessica Light of Texas A&M University was formerly a post-doctoral fellow at the Florida Museum. The researchers completed the project with the help of Reed's NSF Faculty Early Career Development Award, which is granted to researchers who exemplify the teacher-researcher role.

http://www.scientificamerican.com/article.cfm?id=a-whole-lot-of-nothing

New Subatomic Particle Could Help Explain the Mystery of Dark Matter

A flurry of evidence reveals that "sterile neutrinos" are not only real but common, and could be the stuff of dark matter

By Michael Moyer | Thursday, January 6, 2011 | 26



Neutrinos are the most famously shy of particles, zipping through just about everything—your body, Earth, detectors specifically designed to catch them—with nary a peep. But compared with their heretofore hypothetical cousin the sterile neutrino, ordinary neutrinos are veritable firecrackers. Sterile neutrinos don't even interact with ordinary matter via the weak force, the ephemeral hook that connects neutrinos to the everyday world. Recently, however, new experiments have revealed tantalizing evidence that sterile neutrinos are not only real but common. Some of them could even be the stuff of the mysterious dark matter astronomers have puzzled over for decades.

Physicists aren't quite ready to make such dramatic pronouncements, but the results "will be extremely important—if they turn out to be correct," says Alexander Kusenko of the University of California, Los Angeles.

HIDDEN CLUE: Pulsars, including one inside this "guitar nebula," provide evidence of sterile neutrinos. **Image: Courtesy of Shami Chatterjee and James M. Cordes Cornell University**

How did scientists go about looking for particles that are virtually undetectable? Kusenko and Michael Loewenstein of the NASA Goddard Space Flight Center reasoned that if sterile neutrinos really are dark matter, they would occasionally decay into ordinary matter, producing a lighter neutrino and an x-ray photon, and it would make sense to search for these x-rays wherever dark matter is found. Using the Chandra x-ray telescope, they observed a nearby dwarf galaxy thought to be rich in dark matter and found an intriguing bump of x-rays at just the right wavelength.

Another piece of evidence comes from supernovae. If sterile neutrinos really do exist, supernovae would shoot them out in a tight stream along magnetic field lines, and the recoil from this blast would kick the pulsars out through the cosmos. It turns out astronomers observe precisely that: pulsars whizzing through the universe at speeds of thousands of kilometers a second.

Astronomers don't have to rely on the skies for evidence of sterile neutrinos, though. Scientists at Fermi National Accelerator Laboratory recently verified a 16-year-old experiment that sought the first evidence of these particles. The Fermilab scientists fired ordinary neutrinos through Earth at a detector half a kilometer away. They found that in flight, many of these neutrinos changed their identities in just the way they should if sterile neutrinos do in fact exist.

The next step is to confirm the results. Loewenstein and Kusenko recently repeated their experiment on another space-based x-ray telescope, the XMM-Newton, and Fermilab scientists are also setting up another run. The shyest elementary particles may not be able to evade their seekers for long.

http://www.bbc.co.uk/news/uk-scotland-edinburgh-east-fife-12129090

Jet lag found to hinder malaria parasite

The parasite which causes malaria is poor at spreading the disease if it is jet lagged, research has suggested.

Edinburgh University experts gave the parasites "jet lag" by inserting them into mice whose body clocks were different to their own 24-hour cycle. Some of the mice were awake in the daytime and others active at night. They found malaria was only half as effective at causing infection and spreading disease in mice which had a different routine to the parasite. The study gives scientists a greater understanding of when malaria parasites are at their most harmful and when they are vulnerable.

'Clever tricks'

The scientists said the findings may also be a useful aid in developing treatments to tackle the disease.

Dr Sarah Reece, of Edinburgh University's school of biological sciences, who led the research, said: "For this study, we effectively gave the parasites jet lag.

"Our findings suggest that parasites have developed some clever tricks to get their timing right and cause an infection. "This is rare evidence that organisms whose body clock is in sync with their environment have a better chance of survival. "The more we know about how malaria parasites work, the better equipped we will be to tackle them effectively."

Internal body clocks, found in most living things, are determined by patterns of daylight and darkness and govern a range of functions such as sleep cycles, blood pressure, and physical strength.

Malaria, which is spread by the bite of the mosquito, kills one million people each year in sub-Saharan Africa. It affects hundreds of millions more.

The research was carried out at the universities of Edinburgh and Oxford. The study was funded by the Wellcome Trust and the Royal Society and published in Proceedings of the Royal Society B.

http://www.scientificamerican.com/blog/post.cfm?id=can-sitting-too-much-kill-you-2011-01-06

Can sitting too much kill you?

By Travis Saunders | Jan 6, 2011 10:39 AM | 11

Does it matter whether you spend 16 hours sitting on your butt, versus standing or walking at a leisurely pace? Fortunately or unfortunately, new evidence suggests that it does matter, and in a big way

We all know that physical activity is important for good health—regardless of your age, gender or body weight, living an active lifestyle can improve your quality of life and dramatically reduce your risk of death and disease. But even if you are meeting current physical activity guidelines by exercising for one hour per day (something few Americans manage on a consistent basis), that leaves 15 to 16 hours per day when you are *not* being active. Does it matter how you spend those hours, which account for more than 90% of your day? For example, does it matter whether you spend those 16 hours sitting on your butt, versus standing or walking at a leisurely pace? Fortunately or unfortunately, new evidence suggests that it *does* matter, and in a big way.

What is sedentary behavior?

Before we go any further, it's important that we define the term "sedentary behavior". Sedentary behavior is typically defined as any behavior with an exceedingly low energy expenditure (defined as <1.5 metabolic equivalents). In general, this means that almost any time you are sitting (e.g. working on a computer, watching TV, driving) or lying down, you are engaging in sedentary behavior. There are a few notable exceptions when you can be sitting or lying down but still expend high energy expenditure (e.g. riding a stationary bike), but in general if you are sitting down, you are being sedentary.

The above definition may seem rather intuitive, but this is not the way that the term sedentary has been used by exercise science researchers for the past 50 years. Up until very recently, referring to someone as sedentary meant simply that they were not meeting current guidelines for physical activity. In simple terms, if you were exercising for 60+ minutes/day, you were considered physically active. If you were exercising 10 minutes/day, you were sedentary. Case closed. But as we will discuss below, *sedentary time is closely associated with health risk regardless of how much physical activity you perform on a daily basis*. Further, it is entirely possible to meet current physical activity guidelines while still being incredibly sedentary. Thus, to quote researcher Marc Hamilton, **sitting too much is not the same as exercising too little**. (if you take only one thing from this post, let it be that quote from Dr Hamilton). Which is why it is so important that when we use the term "sedentary", we are all on the same page about what that means.

Now that we know what sedentary behavior is, let's look at its relationship with health risk.

Epidemiological Evidence

In 2009 Dr Peter Katzmarzyk and colleagues at the Pennington Biomedical Research Center published an influential longitudinal paper examining the links between time spent sitting and mortality in a sample of more than 17,000 Canadians (available here). Not surprisingly, they report that time spent sitting was associated with increased risk of all-cause and cardiovascular disease mortality (there was no association between sitting and deaths due to cancer). But what is fascinating is that the relationship between sitting time and mortality was independent of physical activity levels. In fact, **individuals who sat the most were roughly 50% more likely to die during the follow-up period than individuals who sat the least, even after controlling for age, smoking, and physical activity levels**. Further analyses suggested that the relationship between sitting time and mortality was also independent of body weight. This suggests that all things being equal (body weight, physical activity levels, smoking, alcohol intake, age, and sex) the person who sits more is at a higher risk of death than the person who sits less.

The above findings linking excessive sitting with poor health are far from isolated. For example, a similar longitudinal study from Australia reports that each hour of daily television viewing (a proxy of sedentary time) is associated with an 11% increase in the risk of all-cause mortality regardless of age, sex, waist circumference, and physical activity level. And as my colleagues and I summarize in a recent review paper (PDF), numerous epidemiological studies have linked sedentary behavior with obesity, cardiometabolic risk, and even some cancers.

New evidence also suggests that in addition to the quantity of sedentary time, the *quality* of sedentary time may also have an important health impact. For example, Genevieve Healy and colleagues examined this issue in participants of the Australian Diabetes, Obesity and Lifestyle (AusDiab) Study. A total of 168 men and women aged 30-87 years wore an accelerometer (an objective measure of bodily movement) during all waking hours for 7 consecutive days, which allowed the researchers to quantify the amount of time that participants spent being sedentary, as well as how frequently they interrupted these sedentary activities (e.g. standing, walking to the washroom, etc).

What did they find?

The greater the number of breaks taken from sedentary behavior, the lower the waist circumference, body mass index, as well as blood lipids and glucose tolerance. This was true even if the total amount of sedentary time and physical activity time were equal between individuals—the one who took breaks more frequently during their time at the office or while watching television was less obese and had better metabolic health. Importantly, the breaks taken by the individuals in this study were of a brief duration (<5 min) and a low intensity (such as walking to the washroom, or simply standing).

Taken together, the epidemiological evidence strongly suggests that prolonged sitting is an important health risk factor. But what explains these relationships? Let's now look at the multiple mechanisms linking sedentary time with increased health risk.

Mechanisms

Reduced Energy Expenditure

Quite obviously (and by definition), when you are sedentary, you are not being physically active. And so one common assumption is that people who sit more are at increased health risk simply because they are getting less physical activity. However, somewhat surprisingly, sitting time and physical activity do not appear to be related for most people. For example a paper from the European Youth Heart Study published in PLoS Medicine reports no association between physical activity and TV watching in a sample of nearly 2000 children and teenagers, and other reports suggest that there is little evidence that sedentary behavior displaces moderate or vigorous physical activity. So while it makes intuitive sense that being sedentary reduces energy expenditure, it is likely through the reduction of very light intensity physical activity (e.g. standing, walking at a slow pace), rather than by reducing the volume of what we typically think of as exercise. This may also help explain why the relationship between sedentary behavior and health risk are often independent of moderate or vigorous physical activity.

Increased Food Intake

In addition to reducing our energy expenditure, sedentary behaviors may also promote excess food intake. For example, a recently published study in the *American Journal of Public Health* suggests that the amount of commercial television (e.g. television with advertisements) that children watch before the age of 6 is associated with increased body weight 5 years down the road, even after adjustment for other important variables including physical activity, socio-economic status and mother's BMI. In contrast, watching non-commercial television (DVD's or TV programs without commercials) showed no association with body weight. Similarly, it has also been reported that each hour of daily television watching in children is associated with an increased consumption of 167 calories per day (PDF), mainly through increased consumption of high calorie, low nutrient foods (e.g. the foods most commonly advertised on television). Much of this is likely just a learned behavior—watching TV exposes us to food ads promoting unhealthy fare, which is likely to have a disproportionate influence on younger viewers. Just as importantly, people may just really enjoy munching on food while relaxing on the couch. Either way, excess sitting (and TV watching in particular) seems to put us in situations where we choose to eat more than we would otherwise.

Physiological Adaptations

I don't think the mechanisms described above—that sitting too much may lead to reduced energy expenditure and increased food intake—will come as much of a surprise. But what I find truly fascinating is that **sedentary behavior also results in rapid and dramatic changes in skeletal muscle**. For example, in rat models, it has been shown that just 1 day of complete rest results in dramatic reductions in muscle triglyceride uptake, as well as reductions in HDL cholesterol (the good cholesterol). And in healthy human subjects, just 5

days of bed rest has been shown to result in increased plasma triglycerides and LDL cholesterol, as well as increased insulin resistance—all very bad things. And these weren't small changes—triglyceride levels increased by 35%, and insulin resistance by 50%!

These negative changes are likely related to reductions in the activity of lipoprotein lipase, an enzyme which allows muscle to uptake fat, thereby reducing the amount of fat circulating in the blood (it also strongly influences cholesterol levels—the details can be found here). Animal research has shown that lipoprotein lipase activity is reduced dramatically after just six hours of sedentary behavior—not unlike a typical day at work or school for many individuals. Sedentary behavior may also reduce glucose transporter protein content in the muscle, making it more difficult for glucose to be taken into the muscle and resulting in higher blood sugar levels. What is most interesting to me personally is that **these physiological changes in skeletal muscle have little or nothing to do with the accumulation of body fat, and occur under extremely rapid time-frames**. This means that both lean and obese individuals, and even those with otherwise active lifestyles, are at increased health risk when they spend excessive amounts of time sitting down.

Should we be concerned about the health impact of sedentary behavior?

Yes.

Western society is built around sitting. **We sit at work, we sit at school, we sit at home, and we sit in our cars as we commute back and forth**. In fact, a recent survey reports that the average American accumulates more than 8 hours of sedentary behavior every day—roughly half of their waking hours. The situation in children is, unfortunately, no different. There is evidence that children in both Canada and the USA (PDF) accumulate more than 6 hours of screen-time (time spent in front of the TV, computer, or other screen-based device) on a daily basis. Keep in mind that screen-time is almost exclusively sedentary (active video games excluded), and that all these hours of sedentary behavior are in addition to the hours and hours (and hours) that kids spend sitting at school. In fact, a recent study reports that roughly 70% of class time, including physical education class, is completely sedentary (while slightly better than class time, children were also sedentary for the majority of lunch and recess).

In short, given the consistent links between sedentary behavior and both death and disease, and the ubiquity of sedentary behavior in our society, we should be very concerned about the health impact of sedentary behavior.

What is the take-home message?

There is a rapidly accumulating body of evidence which suggests that prolonged sitting is very bad for our health, even for lean and otherwise physically active individuals. The good news? Animal research suggests that simply walking at a leisurely pace may be enough to rapidly undo the metabolic damage associated with prolonged sitting, a finding which is supported by epidemiological work in humans. So, while there are a *lot* of questions that remain unanswered (e.g. Is there a "safe" amount of daily sedentary time?), the evidence seems clear that we should strive to limit the amount of time we spend sitting. And when we do have to sit for extended periods of time (which, let's face it, is pretty much every single day for many of us) we should take short breaks whenever possible.

Finally, if you take only one thing from this post, let it be this - sitting too much is not the same as exercising too little.

http://www.bbc.co.uk/news/world-europe-12128729

Czech doctors go for broke

By Rob Cameron BBC News, Prague

Dozens of Czech hospitals could find themselves struggling to provide basic care on 1 March after almost 4,000 doctors - one quarter of the total number working in the country's hospitals - tendered their resignations in protest at low wages.

The resignations are being co-ordinated by the doctors' union (Lok), which says successive Czech governments have done nothing to improve doctors' salaries since the fall of communism.

Peter Papp, 31, is an oncologist working at a hospital in Usti nad Labem, an industrial city about an hour's drive north of Prague. He spent six years at medical school followed by three years on the cancer wards of two district hospitals. With maximum overtime, Dr Papp's gross salary is $1,165 (£750, 880 euros) per month, well below the national average.

After tax, health and social insurance payments, he takes home around $900 dollars, less than a car mechanic or waiter. With rent in the Usti area at around $350 per month, he is left with slightly more than $500 to feed, clothe, transport and entertain himself. After devoting the last nine years of his life to medicine, Dr Papp has had enough.

"I'm not willing to work for the salary of a McDonald's employee," he told me, pointing out that he had made more money teaching English to pay his way through medical school.

Applications in the post

Dr Papp is one of 3,837 Czech doctors who have submitted formal letters of resignation to their hospital managers. On 1 March, he will be unemployed. "I have a little amount of money to sustain me for a month or two," he said. "I'm already sending applications to several companies that send doctors to Ireland and the United Kingdom. I'm also thinking about Australia or the United States.

"I wasn't trained to treat particular nations. A sick person is a sick person anywhere in the world."

The mass resignations have been orchestrated by the Lok union under a slick PR campaign called "Thank You, We're Leaving".

A battered ambulance toured hospitals in December advising doctors on how to quit their jobs. There was a grim message for Czech citizens written on the side: "Our exodus - your exitus".

"We didn't start the brain drain," said Dr Martin Engel, a radiologist at Prague's sprawling Vinohrady Hospital and the chairman of the Lok.

After 30 years of experience and numerous specialist qualifications, Dr Engel earns $2,230 per month, including overtime and before tax. "Doctors have been leaving this country for years," he told me, his face betraying a mixture of anger and resignation. "What we're trying to do with this campaign is stop it."

An ambulance with the slogan 'Our exodus - your exitus' Doctors have given a warning that their exodus could have grim repercussions

As we recorded the interview, a colleague entered the tiny room shared by the two men and changed out of his threadbare white uniform. "We want real change and we want it now," he explained.

"We're not waiting for 'reforms'. We want money. Then we can talk about reforms."

Health Minister Leos Heger says there is simply no room in the budget to meet the doctors' demands. He has asked them to wait for the impact of the government's planned healthcare reforms.

Mr Heger remains unmoved by the protests, telling the Czech media this week that even if all 3,837 did leave their jobs, basic care would remain unaffected. That is an optimistic prediction. In one region, the Vysocina, 80% of all hospital doctors have handed in their resignations. If they do quit, some hospitals may be forced to close. And there is the rub - if they do actually quit.

'Unqualified'

The health ministry told the BBC it was unlikely so many Czech doctors could find work abroad.

Most, said a spokesman, had neither the language skills nor the special pan-European qualifications needed to work elsewhere. There have already been reports of doctors rescinding their letters of resignation.

The doctors' union says those reports are exaggerated and points out that most of its members do not want to leave the Czech Republic, merely its state-run health system. Many, says Martin Engel, can easily find work in privately run clinics.

So it is a game of high stakes between physicians and politicians, and the outcome is uncertain.

If the doctors are bluffing and the government stands firm, Czech healthcare - and Mr Heger - will survive. If they are not, many patients will be in for a nasty shock on 1 March.

http://www.eurekalert.org/pub_releases/2011-01/uoth-ssl010711.php

Scientists shed light on what causes brain cell death in Parkinson's patients

New findings could lead to new treatments for the 95 percent of Parkinson's cases with no known cause

SAN ANTONIO — Just 5 percent of Parkinson's disease cases can be explained by genetic mutation, while the rest have no known cause. But a new discovery by researchers at The University of Texas Health Science Center San Antonio may begin to explain why the vast majority of Parkinson's patients develop the progressive neurodegenerative disease.

This week in the Journal of Neuroscience, the researchers demystified a process that leads to the death of brain cells – or neurons – in Parkinson's patients. When researchers blocked the process, the neurons survived.

The findings could lead to an effective treatment to slow the progression of Parkinson's disease, rather than simply address symptoms that include tremors, slowed movement, muscle stiffness and impaired balance. Further studies could lead to a diagnostic test that could screen for Parkinson's years before symptoms develop, said Syed Z. Imam, Ph.D., adjunct assistant professor at the UT Health Science Center.

Parkinson's disease, which usually is not diagnosed until age 60 or later, affects an estimated half-million people in the United States.

Dr. Imam joined the U.S. Food and Drug Administration (FDA) after the research was conducted. Co-authors are from the Health Science Center's Barshop Institute for Longevity and Aging Studies; the South Texas Veterans Health Care System; and the Hertie Institute for Clinical Brain Research in Tübingen, Germany.

The mechanism

After analyzing cells and post-mortem brain tissue from animals and humans, researchers noted that oxidative stress – a known culprit in neuron death – activated a protein called tyrosine kinase c-Abl in the nigra-striatum area of the brain. Neurons in this part of the brain are particularly vulnerable to Parkinson's injury.

Activation of this protein led to changes in another protein called parkin, which is known to be mutated in hereditary Parkinson's. The altered parkin lacked the capacity to break down other proteins, leading to harmful clumps of unprocessed protein in the neuron. The scientists believe this accumulation leads to progressive neuron death, resulting in Parkinson's symptoms that worsen over time.

Implications

"When we blocked tyrosine kinase c-Abl activation, parkin function was preserved and neurons were spared," Dr. Imam said. "We believe these studies provide sound rationale for moving forward with a preclinical trial of tyrosine kinase c-Abl inhibitors, with the goal of developing a potent therapeutic drug for slowing the progression of Parkinson's."

If preclinical trials in animal models of Parkinson's disease yield positive results, the next step would be clinical trials in human patients, Dr. Imam said.

Tyrosine kinase c-Abl inhibitors are approved by the FDA for treating myeloid leukemia and gastrointestinal tumors. This could speed approval of the drug for Parkinson's and its translation from bench research to clinical practice.

"The race is on to understand the mechanism of the 95 percent of Parkinson's cases with no known cause, and our finding certainly is a building block," Dr. Imam said. "We have found a specific signaling mechanism that is only turned on by oxidative stress and is selective only to Parkinson's-affected neurons of the nigra-striatum, which is the area that sends signals for balance to the cerebellum."

Acknowledgements *Co-authors from the UT Health Science Center San Antonio are Senlin Li, M.D., senior author; Qing Zhou, Ph.D.; Anthony J. Valente, Ph.D.; Mona C. Bains, Ph.D.; Robert A. Clark, M.D.; and James L. Roberts, Ph.D., whose primary appointment is now at Trinity University in San Antonio. Co-authors represent the School of Medicine and the Graduate School of Biomedical Sciences as well as the Barshop Institute.*

The National Institutes of Health, Michael J. Fox Foundation, American Parkinson's Disease Association, Parkinson's Disease Foundation, San Antonio Area Foundation and Health Science Center Presidential Research Enhancement Fund supported the research.

http://www.eurekalert.org/pub_releases/2011-01/hu-apb010711.php

A pesky bacterial slime reveals its survival secrets

Surprising discovery about biofilm may provide a new direction in antimicrobial research and bio-inspired liquid-repellent surfaces for use in healthcare, agriculture and industry

Cambridge, Mass., – By rethinking what happens on the surface of things, engineers at Harvard University have discovered that Bacillus subtilis biofilm colonies exhibit an unmatched ability to repel a wide range of liquids—and even vapors. Centimeters across yet only hundreds of microns thick, such slimy bacterial coatings cling to the surfaces of everything from pipes to teeth and are notoriously resistant to antimicrobial agents. The researchers now suspect they know the secret to a biofilm's resiliency.

Published in the January 5th early edition of the Proceedings of the National Academy of Sciences (PNAS), the study holds promise for both creating bio-inspired non-wetting materials and developing better ways to eliminate harmful biofilms that can clog pipes, contaminate food production and water supply systems, and lead to infections.

"By looking at biofilms from a materials perspective rather than a cellular or biochemical one, we discovered that they have a remarkable ability to resist wetting to an extent never seen before in nature," says lead author Alex Epstein, a graduate student at the Harvard School of Engineering and Applied Sciences (SEAS). "In fact the biofilm literally resisted our initial efforts to study it."

The finding came about serendipitously, as the original intention of the researchers was to study the structure of the biofilm. To image the interior of the biofilm, the team had to soak it with liquids such as ethanol and acetone, which normally spread and seep easily into a surface.

"But to our surprise, it was impossible. The liquids kept beading up on the surface and wouldn't infiltrate the colonies," says Epstein, who is a member of the laboratory of Joanna Aizenberg, Amy Smith Berylson Professor of Materials Science at SEAS; Susan S. and Kenneth L. Wallach Professor at the Radcliffe Institute; and a core member of the Wyss Institute for Biologically Inspired Engineering at Harvard.

As the Aizenberg lab studies materials and wetting, the engineers immediately recognized the significance of what they were observing. It turns out that biofilm has an unprecedented liquid-repellent surface, thereby revealing a critical clue to what may be responsible for its broad antimicrobial resistance.

Nature offers numerous examples of water-resistant surfaces, such as the lotus leaf, a longstanding inspiration for creating synthetic materials. Until now, however, no model natural systems have been found for broadly repellent materials.

While such surfaces can be manufactured, the top-down process is costly, labor intensive, and reliant on toxic chemicals and brittle structures. A biofilm, however, is living proof that only the simplest and most natural of components are required—namely, a resilient meshwork made from proteins and polysaccharides assembled into a multi-scale, hierarchical structure.

At the same time, the finding offers a completely new perspective on how biofilms are immune to so many different types of biocides. Even the most sophisticated biochemical strategy will be ineffective if a biocide cannot enter the slime to reach the bacteria. In short, the antimicrobial activity of alcohols and other solvents becomes compromised by the strongly non-wetting behavior at clinically relevant concentrations.

The team expects that their newfound knowledge will help alert researchers to the need to consider this requirement when designing ways to destroy harmful biofilms.

"Their notorious resistance to a broad range of biocide chemistries has remained a mysterious and pressing problem despite two decades of biofilm research," says Aizenberg, a pioneer in the field of biomimicry. "By looking at it as a macroscopic problem, we found an explanation that was just slightly out of view: antimicrobials can be ineffective simply by being a non-wetting liquid that cannot penetrate into the biofilm and access subsurface cells."

Aizenberg and her colleagues speculate that such strong liquid repellence may have evolved in response to the bacteria's natural soil environment where water can leach heavy metals and other toxins.

Moreover, the property may underlie the recent success of the use of biofilm as an eco-friendly form of biocontrol for agriculture, protecting plant roots from water-borne pathogens.

Looking ahead, the Harvard team plans to investigate precisely how the biochemical components of biofilms give rise to their exceptional resistance and to test the properties of other bacterial species.

"The applications are exciting, but we are equally thrilled that our findings have revealed a previously undocumented phenomenon about biofilms," says Aizenberg. "The research should be an inspiring reminder that we have only scratched the surface of how things really work."

Just as with biofilm, she adds, "It has been a challenge to get deep into the core of the problem."

Epstein and Aizenberg's co-authors included Boaz Pokroy, a former postdoctoral fellow in Aizenberg's group and now a faculty member at Technion (the Israel Institute of Technology), and Agnese Seminara, a postdoctoral fellow at SEAS and participant in the Kavli Institute for Bionanoscience and Technology at Harvard University.

The research was funded by the BASF Advanced Research Initiative at Harvard University.

http://www.physorg.com/news/2011-01-road-bike-parkinson-diagnosis.html

On the road: Bike riding helps Parkinson's diagnosis

Neurologists examining a patient with early symptoms of Parkinson's disease should ask that individual to ride a bike, according to unusual research by Dutch doctors.

Distinguishing between patients with Parkinson's and those with a disorder known as atypical parkinsonism is important as the two conditions have different causes and treatments.

They share a set of shared symptoms, including limb tremor, slowed movement, muscle rigidity and unsteadiness -- and sometimes even advanced medical technology is unable to make an early distinction.

But, say researchers at the Parkinson Centre in Nijmegen, an effective, non-disruptive, low-cost diagnostic tool is this: can the patient ride a bike?

It may seem counter-intuitive, but people with "regular" Parkinson's often have a remarkable ability to ride a bike, as they have few problems with sideways balance and with the short, rhythmic movements needed to pedal, the doctors said in a letter published on Friday by The Lancet. This task, though, is often too demanding for people with atypical parkinsonism, the umbrella term for half a dozen syndromes such as progressive supranuclear palsy, multiple system atrophy and corticobasal degeneration.

Parkinson's disease comes from the death of cells in a key part of the brain called the substantia nigra that produce a neurotransmitter chemical called dopamine. The standard treatment is a drug called levodopa, which the brain converts into dopamine. But the treatment does not work effectively, or may not even work at all, in atypical parkinsonism.

The Nijmegen doctors tested their theory on 111 patients with Parkinson-like symptoms and who were able to ride a bike at the start of the study. Afterwards, 45 of the patients went on to be confirmed as having Parkinson's and 64 as having atypical parkinsonism.

During the 30 months that they were under study, two of the 45 later found to have Parkinson's stopped riding a bike, but the tally was 34 out of the 64 who were eventually diagnosed with the "atypical" disease.

"We suggest that the loss of the ability to cycle after disease onset might serve as a new red flag, signalling the presence of atypical parkinsonism," said the letter. *(c) 2011 AFP*

http://www.newscientist.com/article/mg20927942.700-stem-cells-from-urine-could-help-fix-your-plumbing.html

Stem cells from urine could help fix your plumbing

*** 07 January 2011 by Andy Coghlan**

IT MAY be a bodily waste product, but urine contains cells with huge potential for repairing our internal plumbing.

Yuanyuan Zhang and his colleagues at the Wake Forest University School of Medicine in Winston-Salem, North Carolina, have made urethra-like tissue by growing stem cells extracted from the urine of four healthy volunteers on scaffolds made from pig gut tissue.

To do this, the team first converted stem cells extracted from urine into urothelial cells and smooth muscle cells - vital cell lines for making ureters, which empty fluid from the kidneys into the bladder, and urethras, which conduct it from the bladder out of the body.

Zhang then chemically stripped all pig cells from layers of pig gut tissue, leaving just the underlying inert collagen scaffold. He coated this scaffold with the two types of cell. Two weeks later, the deposited cells had formed layers on the scaffolds resembling urethras and ureters.

In another experiment, the same structures developed after the seeded scaffolds had been implanted in mice lacking an immune system, proving that the cells can survive and grow in live animals (Biomaterials, DOI: 10.1016/j.biomaterials.2010.10.006).

Zhang plans further experiments in larger animals and eventually in humans. He and his colleagues hope to emulate the clinical success seen two years ago when researchers replaced a woman's damaged windpipe by growing her stem cells on a section of donated windpipe that had been stripped of the donor's cells.

There seem to be ample stem cells in urine to make these structures. A single colony of converted cells can coat a scaffold up to 10 cubic centimetres in volume, and just 200 millilitres of urine contains enough stem cells to form 15 colonies, say the team.

http://www.technologyreview.com/computing/27037/page1/?a=f

Researchers beat automatic locking and ignition systems.

By Erica Naone

Car thieves of the future might be able to get into a car and drive away without forced entry and without needing a physical key, according to new research that will be presented at the Network and Distributed System Security Symposium next month in San Diego, California.

The researchers successfully attacked eight car manufacturers' passive keyless entry and start systems—wireless key fobs that open a car's doors and start the engine by proximity alone.

Srdjan Capkun, an assistant professor of computer science in the system security group at ETH Zurich in Switzerland, who led the work, says he was inspired to investigate the security of keyless entry and start systems after buying a car that had one. Capkun and Aurélien Francillon and Boris Danev, both researchers in the same institution, examined 10 car models from the eight manufacturers. They were able to access all 10 and drive them away by intercepting and relaying signals from the cars to their wireless keys. While they could relay the signals from the key back to the car as well, usually they did not need to because the key transmits its signals up to around 100 meters. The attack works no matter what cryptography and protocols the key and car use to communicate with each other.

Normally, when a wireless key is within a few meters of the right car, it detects a low-powered signal that causes it to issue a command that opens the car enable the ignition. The researchers used a pair of antennas to transmit these signals from the car to the key when the key was farther away, tricking the car into opening without the ordinary authorization. One antenna needs to be very close to the car, and one needs to be within eight meters of the key.

The researchers came up with two versions of the attack. In one, they ran a cable from near the car to near the key and used it to transmit the signals. They conducted the other wirelessly. Francillon says that the materials for the wired attack cost about $50, and those for the wireless attack cost between $100 and $1,000, depending on the electronic components used.

The researchers tested a few scenarios. An attacker could watch a parking lot and have an accomplice watch as car owners as entered a nearby store. The accomplice would only need to be within eight meters of the targeted owner's key fob, making it easy to avoid arousing suspicion. In another scenario, a car owner might leave a car key on a table near a window. An antenna placed outside the house was able to communicate with the key, allowing the researchers then to start the car parked out front and drive away.

A car won't open or start if the signal from its key takes too long to arrive, so the researchers devised a way to speed communication between their antennas. Most relay attacks require the signals to be converted from analog to digital and back, which takes time. The researchers were able to keep the signals in analog format, which reduced their delay from microseconds to nanoseconds and made their attack more difficult to detect.

The researchers suggest things that car owners and manufacturers can do to protect themselves. Car owners can shield their keys when they're not in use, to prevent attackers from communicating with them. Alternatively, manufacturers could add a button to fobs that would allow owners to deactivate and reactivate them. Capkun worries, however, that these types of solutions detract from the convenience that makes passive keyless entry systems worthwhile.

Ultimately, he says, manufacturers will need to add secure technology that allows the car to confirm that the key is in fact nearby. "I don't see a way around it," Capkun says. His group is actively working on protocols that would accomplish this.

David Wagner, a professor of computer science at the University of California at Berkeley who has studied the cryptographic systems used in keyless entry systems, says the research "should help car manufacturers improve auto security systems in the future."

Wagner doesn't think the research ought to make car owners anxious. "There are probably easier ways to steal cars," he says. But, he adds, a "nasty aspect of high-tech car theft" is that "it doesn't leave any sign of forced entry," so if a thief did use this method to steal a car, he says, it might be hard for police and insurance companies to get sufficient evidence of what happened. Wagner believes that manufacturers, police, and insurance companies all need to prepare for this eventuality.

"Automobiles are a key example of a system that is pervasively computerized," so they need to be thoroughly examined to ensure they are secure, says Tadayoshi Kohno, an assistant professor of computer science at the University of Washington. Kohno helped form the Center for Automotive Embedded Systems Security, which is dedicated to identifying and solving security problems with car security systems before they cause problems in the real world.

http://www.eurekalert.org/pub_releases/2011-01/w-gik010611.php

Ginger is key ingredient in recipe for conserving stag beetles

The humble ginger root could be the key to conserving the UK's largest and most spectacular terrestrial beetle – the stag beetle.

Ecologists from Royal Holloway, University of London and the University of York have developed a series of new methods to monitor stag beetle numbers – including ginger lures to trap adult beetles and tiny microphones to detect sounds made by the larvae in their underground nests. Conservation efforts have been hampered until now because ecologists lacked a reliable way of monitoring stag beetle numbers.

The new research, published in the Royal Entomological Society's journal Insect Conservation and Diversity, found that a combination of ginger-baited aerial traps to catch adult stag beetles, plus tiny microphones to record the underground larvae's sounds and samplers to detect the chemicals they emit, give an accurate picture of the species' abundance.

According to Dr Deborah Harvey, one of the study's authors: "Our new methods offer genuine promise for monitoring the population of this elusive and rare insect, one that we think is declining across much of its European range. We need to know where the stag beetle lives – and in what numbers – to be able to conserve it effectively."

Harvey and her colleagues discovered ginger was irresistible to adult stag beetles only after testing the attractiveness of many other fruit and vegetables – including banana, strawberry, tomato and cherry – as well as wine and beer. Ginger works because it contains large amounts of alpha copaene, a chemical known to attract other insects that live in dead and decaying wood.

By using ginger, and designing the trap using heavy duty plastic, Harvey was able to produce a very cost-effective trap, which is vital because most insect monitoring in the UK is done by a small army of dedicated but unfunded amateur recorders.

Using other methods of trapping insects, such as light traps or traps baited with food, do not work with adult stag beetles because they are not reliably attracted to light and the species does not eat during the adult phase of its life cycle.

As well as finding a method of monitoring adult numbers, Harvey also needed a way to detect larvae, which live underground. Hand searching is likely to destroy their habitats, so instead the team used tiny microphones to pick up the sounds – known as stridulation – the larvae make, together with so-called diffusive samplers to detect a chemical (longifolene) they emit.

Harvey says: "Sampling subterranean insects without destroying the larval habitat is notoriously difficult. These diffusive samplers are widely used to monitor environmental pollution, but this is the first time they have been used for insect detection. Because longifolene can be produced by plants, we used it together with sound recording to come up with a more accurate method of finding stag beetle larvae."

The team found that stridulation patterns produced by stag beetle larvae are very different from other species likely to live nearby, such as the rose chafer (Cetonia aurata) and the lesser stag beetle (Dorcus parallelipipedus). "Stridulation is likely to be a form of communication between larvae; it increases if larvae are handled or placed in solitary confinement," Harvey says.

These are the first ever sound recordings of lesser stag beetle and rose chafer larvae. The latter sound like squeaky shoes.

The new methods could help conserve other rare species. According to Harvey: "Acoustic detection of insects as a sampling method is very underused, but we believe it could have great potential in detecting larvae in the field."

The study was funded by the British Ecological Society, the Forestry Commission, the People's Trust for Endangered Species and the Suffolk Naturalists' Society.

Alan Gange, Deborah Harvey, Colin Hawes, Paul Finch, David Chesmore and Ian Farr. Development of non-invasive monitoring methods for larvae and adults of the stag beetle, Lucanus cervus, Insect Conservation and Diversity, doi: 10.1111/j.1752-4598.2009.00072.x, is published online on Monday 10 January 2011 (http://dx.doi.org/10.1111/j.1752-4598.2009.00072.x) .

http://www.bbc.co.uk/news/health-12135536

Stroke recovery boosted by a course of Prozac

Giving stroke patients Prozac soon after the event could help their recovery from paralysis, a study has found.

Researchers discovered more improvement in movement and greater independence after three months in patients taking the antidepressant (also known as fluoxetine), compared to placebo.

The Lancet Neurology study was based on research on 118 patients in France.

UK stroke experts said the findings were "promising".

This was the largest study of selective serotonin re-uptake inhibitors (SSRIs) and stroke recovery to date.

Tests on stroke patients 90 days after being given the drug found that patients taking fluoxetine had gained significantly more function in their upper and lower limbs than patients who were not given the drug.

Patients in the fluoxetine group were also more likely to be coping independently.

All patients in the study had moderate to severe motor disabilities following their stroke.

'Dual benefit?'

The study noted that the side-effects from the antidepressant were generally mild and infrequent, although this group did notice more instances of nausea and diarrhoea.

The authors, led by Professor François Chollet, said: "The positive effect of the drug on motor function of recovering patients suggests that the... action of SSRIs provides a new pathway that should be explored further in the treatment of acute ischaemic stroke."

Every year in the UK 150,000 people have a stroke and a third of these will be left with a disability such as paralysis down one side of their body.

Dr Sharlin Ahmed, research liaison officer at the UK Stroke Association, said: "We are continually searching for new treatments which can improve the outcomes for stroke survivors and the results of this research look promising.

"Antidepressants, such as fluoxetine, can be used to treat stroke patients with depression which is a common side effect of stroke, so it's very interesting to see that this already licensed drug could have a dual benefit.

"However, further research needs to be undertaken before the use of this antidepressant can be accepted as an effective treatment for improving movement following a stroke."