Smokers would rather give up for their pooch's health rather than their own

Pet owners' attitudes and behaviours related to smoking and secondhand smoke: A pilot study

Smokers are more likely to quit smoking for the sake of their pets' health than they are for their own, suggests research published ahead of print in Tobacco Control.

The published evidence shows that second hand tobacco smoke can be as dangerous for pets as it is for the non-smoking partners of smokers. Exposure to it has been associated with lymph gland, nasal, and lung cancers; allergies; eye and skin diseases; as well as respiratory problems in cats and dogs.

But few smokers realise what impact their habit is having on the health of their pets, say the US researchers. They set up an online survey for pet owners resident in south eastern Michigan, quizzing them about their and their partners' smoking behaviours, and what they knew about the effects of second hand smoke on their pets.

In all, almost 3300 people responded, one in five of whom were smokers and more than one in four of whom (27%) lived with at least one smoker. The average number of cigarettes smoked was 13.5 a day, with around half of those smoked in the home.

Nearly one in three of the smokers (28.4%) said that knowing that smoking was bad for their pets' health would spur them to give it up. And almost one in 10 (8.7%) said this would prompt them to ask their partners to quit, while around one in seven (14%) said they would tell their partner to smoke outdoors.

These figures were even higher among non-smokers, more than 16% of whom said they would ask their partner to quit, while around one in four (24%) said they would tell their partner to smoke outdoors.

Around four out of 10 smokers and one in four non-smokers living with smokers said they would be interested in receiving information on the effects of smoking and how to give up.

Public health campaigns targeting smokers would do well to focus on the detrimental impact of second hand tobacco smoke on pets, say the authors. US pet owners are clearly a very devoted bunch, they say, which such campaigns could tap into.

Almost two thirds of US households have a pet, and their combined spending power on pet supplies and over the counter medicines was estimated to be in the region of more than US$ 10 billion last year. And a survey carried out by the American Animal Hospital Association in 2008 showed that more than half of the respondents said that if they were stranded on a desert island, they would prefer the company of their pet to that of another person.

UF study: Rapid burst of flowering plants set stage for other species

Gainesville, Fla. --- A new University of Florida study based on DNA analysis from living flowering plants shows that the ancestors of most modern trees diversified extremely rapidly 90 million years ago, ultimately leading to the formation of forests that supported similar evolutionary bursts in animals and other plants.

This burst of speciation over a 5-million-year span was one of three major radiations of flowering plants, known as angiosperms. The study focuses on diversification in the rosid clade, a group with a common ancestor that now accounts for one-third of the world's flowering plants. The forests that resulted provided the habitat that supported later evolutionary diversifications for amphibians, ants, placental mammals and ferns.

"Shortly after the angiosperm-dominated forests diversified, we see this amazing diversification in other lineages, so they basically set the habitat for all kinds of new things to arise," said Pamela Soltis, study co-author and curator of molecular systematics and evolutionary genetics at UF's Florida Museum of Natural History. "Associated with some of the subsequent radiations is even the diversification of the primates."

The study appearing online in next week's Proceedings of the National Academy of Sciences is the first to show the evolutionary relationships of these plants and provide evidence for their rapid emergence and diversification.

Because the diversification happened so quickly, at least in evolutionary terms, molecular methods were needed to sort out the branches of the rosid clade's phylogenetic tree, a sort of family tree based on genetic relationships. Only after sequencing many thousands of DNA base pairs are genetic researchers able to tease apart the branches and better understand how plant species evolved.

Often, when scientists discuss the rapid radiation of flowering plants, they talk as if there had been one massive burst of early diversification, said Doug Soltis, co-author and chair of UF's botany department.

"I think one thing that becomes very clear from our phylogenetic trees when you look at them closely is that it's not just one big explosion of species within the flowering plants," Doug Soltis said. "There's a series of explosions."

The rosid clade's diversification is one of at least three bursts in the early evolution of flowering plants. More than 300,000 species of angiosperms exist, classified into an estimated 15,000 genera and more than 400 families. Understanding how these plants are related is a large undertaking that could help ecologists better understand which species are more vulnerable to environmental factors such as climate change.

"We really need to know on a finer scale how these species are related and on different parts of the planet how members of the clade are related," Doug Soltis said. "That's where the action is going to be in terms of how this clade responds to climate change. How members of this large clade respond is really going to determine the fate of most of the organisms on the planet."

The study's authors sequenced 25,000 base pairs of DNA and sampled a broad range of 104 species from the rosid clade. Using a phylogenetic tree to date the diversification of lineages requires the use of a molecular clock, which calibrates the degree of change that has occurred over time.

"You can assume that over time DNA sequences accumulate change, and things that are more similar to each other in general would have diverged from each other more recently than things that are more different," Pam Soltis said.

But different genes have different rates of evolution, as do different clades. To compensate, the study used algorithms that accommodate the different rates. Rosid fossils selected by co-author Steven Manchester, the museum's curator of paleobotany, were used to help calibrate that clock by setting minimum ages for member species.

The study's first author is Hengchang Wang, who worked at the Florida Museum as a post-doctoral fellow but is now with The Chinese Academy of Science. Other authors include former post-doctoral fellows Michael J. Moore from Oberlin College and Charles D. Bell from the University of New Orleans. UF botany graduate students Samuel F. Brockington and Maribeth Latvis, former UF undergraduate Roolse Alexandre, and Charles C. Davis of Harvard University also contributed to the study.

Stroke therapy window might be extended past nine hours for some

Oak Brook, Ill. – Some patients who suffer a stroke as a result of a blockage in an artery in the brain may benefit from a clot-busting drug nine or more hours after the onset of symptoms. The findings are published in the online edition of Radiology.

"Stroke is the third leading cause of death in the U.S.," said the study's lead author, William A. Copen, M.D., Director of Advanced Magnetic Resonance Neuroimaging at Massachusetts General Hospital (MGH) in Boston. "Every hour that we can add to the treatment window would allow vastly more stroke patients to be treated with potentially life-saving therapy."

The most common type of stroke is called ischemic stroke. These strokes occur when a blood clot blocks a blood vessel supplying blood to the brain. Some ischemic strokes can be treated with thrombolytic, or clot-busting, therapy using tissue plasminogen activator (t-PA), which helps dissolve the blockage. However, the window of opportunity to safely administer the medication is generally considered to be just three hours. Because few patients get to the hospital to be diagnosed and treated within that time frame, fewer than seven percent of patients receive the drug.

In this retrospective study, researchers analyzed the test results of 109 ischemic stroke patients at MGH. The testing methods included two different MRI scanning techniques: perfusion MRI, which measures blood flow in the brain, and diffusion MRI, which measures the movement of water molecules in tissue.

"Comparing the lesions that we see in these two MR images reveals which areas of the brain are threatened by a lack of blood flow, but could still be salvageable," Dr. Copen said. "A mismatch between the lesions suggests that a patient might still benefit from thrombolytic therapy."

In the study, most patients with blockage in a proximal artery, close to the base of the brain, continued to demonstrate a diffusion-perfusion mismatch between nine and 24 hours after the onset of their strokes.

"Patients who have a mismatch have been successfully treated up to nine hours after stroke onset, which is already much longer than the guidelines allow, Dr. Copen said. "Our findings suggest a need for a clinical trial to measure the effectiveness of thrombolytic therapy more than nine hours after the onset of an ischemic stroke."

"Existence of the Diffusion-Perfusion Mismatch within 24 Hours after Onset of Acute Stroke: Dependence on Proximal Arterial Occlusion." Collaborating with Dr. Copen were Leila Rezai Gharai, M.D., Elizabeth R. Barak, M.D., Lee H. Schwamm, M.D., Ona Wu, Ph.D., Shahmir Kamalian, M.D., R. Gilberto Gonzalez, M.D., Ph.D., and Pamela W. Schaefer, M.D.

Women Who Drink Two-Plus Cans Of Soda Pop Per Day At Nearly Twice The Risk For Early Kidney Disease

Maywood, Ill. -- Women who drink two or more cans of soda pop per day are nearly twice as likely to show early signs of kidney disease, a recent study has found.

However, researchers did not find an elevated risk for men, or for people who drink diet soda, said lead researcher David Shoham of Loyola University Health System.

The study was published in PLoSONE, a peer-reviewed journal of science and medical research published by the Public Library of Science.

Researchers examined data from a representative sample of 9,358 U.S. adults in the National Health and Nutrition Examination Survey. The NHANES survey included urine samples and a questionnaire about dietary habits.

Women who reported drinking two or more sodas in the previous 24 hours were 1.86 times more likely to have albuminuria, a sensitive marker for early kidney damage. Albuminuria is an excess amount of a protein called albumin in the urine. Since healthy kidneys filter out large molecules such as albumin, an excess amount can be a sign of damage to the kidneys.

About 11 percent of the population has albuminuria. Among those who drink two or more cans of soda per day, 17 percent have this early marker of kidney disease, the study found. It's unclear why drinking soda increased the risk only in women, Shoham said. There may be an unknown underlying cause that is linked to both soda consumption and kidney damage, he said. Shoham is an assistant professor in the Department of Preventive Medicine and Epidemiology.

In recent years, diabetes, obesity and kidney disease have been increasing, along with consumption of high fructose corn syrup, the sweetener used in most sodas.

But what's most important is the amount of sugar, not the type, Shoham said. "I don't think there is anything demonic about high fructose corn syrup per se," Shoham said. "People are consuming too much sugar. The problem with high fructose corn syrup is that it contributes to over consumption. It's cheap, it has a long shelf life and it allows you to buy a case of soda for less than $10."

Shoham and colleagues concluded that additional studies are needed to determine whether the elevated risk of kidney disease is due to high fructose corn syrup itself, an overall excess intake of sugar, unmeasured lifestyle factors or other causes.

A recent pilot study by other researchers, reported in the journal Environmental Health, found that nine of 20 commercial samples of high fructose corn syrup from three manufacturers contained detectable levels of mercury. "This adds the intriguing possibility that it is not just the sugar itself in high fructose corn syrup that is harmful, because mercury is harmful to kidneys as well," Shoham said.

About 26 million American adults have chronic kidney disease, according to the National Kidney Foundation. Advanced kidney disease causes such symptoms as fatigue, poor appetite, trouble sleeping and concentrating and swollen feet. Kidney disease can lead to high blood pressure, anemia, nerve damage, weak bones and cardiovascular disease.

The study was published in the Oct. 17 edition of PLoSONE. Shoham's co-authors are Ramon Durazo-Arizu, Holly Kramer, Amy Luke and Richard Cooper of Loyola University Health System, Suma Vupputuri of Kaiser Permanente and Abhijit Kshirsagar of the University of North Carolina.

Asteroid bound for Earth! Warn your grandchildren

* 09 February 2009 **by David Shiga**

AN ASTEROID that had initially been deemed harmless has turned out to have a slim chance of hitting Earth in 160 years. While that might seem a distant threat, there's far less time available to deflect it off course.

Asteroid 1999 RQ36 was discovered a decade ago, but it was not considered particularly worrisome since it has no chance of striking Earth in the next 100 years - the time frame astronomers routinely use to assess potential threats.

Now, new calculations show a 1 in 1400 chance that it will strike Earth between 2169 and 2199, according to Andrea Milani of the University of Pisa in Italy and colleagues (www.arxiv.org/abs/0901.3631).

With an estimated diameter of 560 metres, 1999 RQ36 is more than twice the size of the better-known asteroid Apophis, which has a 1 in 45,000 chance of hitting Earth in 2036 (New Scientist, 12 July 2008, p 12). Both are large enough to unleash devastating tsunamis if they were to smash into the ocean.

Although 1999 RQ36's potential collision is late in the next century, the window of opportunity to deflect it comes much sooner, prior to a series of close approaches to Earth that the asteroid will make between 2060 and 2080.

Asteroid trajectories are bent by Earth's gravity during such near misses, and the amount of bending is highly dependent on how close they get to Earth. A small nudge made ahead of a fly-by will get amplified into a large change in trajectory afterward. In the case of 1999 RQ36, a deflection of less than 1 kilometre would be enough to eliminate any chance of collision in the next century.

But after 2080, the asteroid does not come as close to Earth before the potential impact, so any mission to deflect it would have to nudge the asteroid off course by several tens of kilometres - a much more difficult and expensive proposition.

"That's worth thinking about," says Clark Chapman of the Southwest Research Institute in Boulder, Colorado.

As is often the case, more precise calculations enabled by future observations will most likely rule out a collision. But Milani's team says that routine monitoring of asteroids should be extended to look for potential impacts beyond the 100-year time frame, to identify any other similar cases.

Autism Consortium members publish in PNAS: Mechanism, treatment for Rett syndrome -- top cause autism girls

Clinical trial to test molecule in humans is being planned

Autism Consortium Scientists Publish Study Defining Mechanism and Potential Treatment for Rett Syndrome, Leading Cause of Autism in Girls

- Clinical trial to test molecule in humans is being planned - - Data in PNAS reveals therapeutic that could apply to other forms of autism -

Boston –The Autism Consortium, an innovative research, clinical and family collaboration dedicated to radically accelerating research and enhancing clinical care for autism spectrum disorders (ASDs), announced today that several Consortium members published a paper with significance for clinical trials in autism in the Proceedings of the National Academy of Sciences.

The research led by Autism Consortium members Mriganka Sur, PhD, Newton Professor of Neuroscience at the Picower Institute and Head of the MIT Department of Brain and Cognitive Sciences; and Rudolf Jaenisch, PhD, Founding Member, Whitehead Institute and Professor of Biology at MIT, demonstrates for the first time a mechanism for Rett Syndrome and a therapeutic that could be directly applicable to humans. As a result, a clinical trial in humans is in development.

IGF1 Reverses Rett Symptoms in Mice; Clinical Trial Planned

This groundbreaking study demonstrated that by treating mice with a peptide fragment of IGF1, a molecule that is utilized by the brain for neuronal and synaptic development, the symptoms of Rett Syndrome in the mice were largely reversed.

"The next step is to test recombinant human IGF1 which is already available for pediatric use in humans with the hope of treating or reversing Rett Syndrome," said Omar Khwaja, MD, PhD, Director of the Rett Syndrome Program at Children's Hospital Boston and head of the clinical trial team for IGF1. "We are working as quickly possible to develop the protocol, secure funding, and initiate the trial."

"This new study presents promising novel data suggesting that targeting the IGF1 signaling axis may present a useful therapeutic strategy that could ultimately be translated to humans," said Dr. Antony Horton, Chief Scientific Officer at the International Rett Syndrome Foundation. "We are encouraged by this collaboration between scientists and clinicians which is yielding valuable insights into potential new treatments for Rett syndrome."

About Rett Syndrome and the Findings

Rett Syndrome, a neurodevelopmental disorder mainly affecting girls and also the most common basis of autism in girls, is primarily caused by a sporadic mutation in the MECP2 gene on the X chromosome. The MECP2 gene makes a protein, also called MeCP2, believed to play a pivotal role in silencing, i.e. turning off the activity of other genes. The MECP2 mutation causes the regulatory mechanism to fail, which in turn causes other genes to function abnormally.

Rett is a genetic disorder of developmental arrest or failure of brain maturation. This is thought to occur when subsets of neurons and their connections (synapses) are disrupted during a very dynamic phase of brain development. Scientists have been investigating ways to reverse that arrest and therefore, turn brain activity back on.

By crossing into the brain and activating 'IGF1 signaling' – IGF1 binds to its receptor and activates downstream molecules within neurons that make synapses mature. This activity in turn ends the developmental arrest thought to underlie the syndrome.

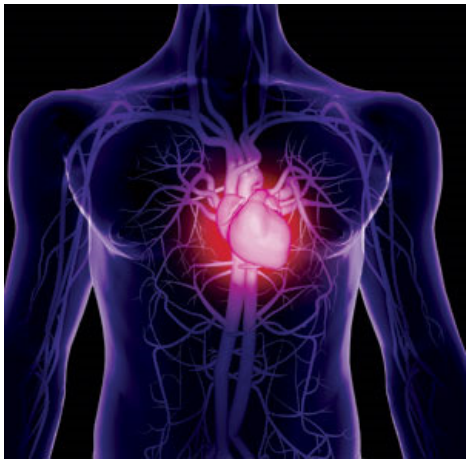
Using mutant mice in which MeCP2 is deleted, Sur and his co-authors demonstrated a major underlying mechanism for the disorder - synapses in the brain remain immature and showed persistent, abnormal plasticity into adulthood.

"Our research is beginning to show that other forms of autism also have, as their basis, this persistent immaturity of synapses," said Sur. "As a result, an even more exciting and promising aspect of this work is the possibility that IGF1 or similar therapeutics could apply not only to autism caused by Rett Syndrome, but also to other causes of autism as well."

In addition to Sur and Jaenisch, the study's authors are postdoctoral fellows Daniela Tropea, Nathan R. Wilson and Cortina McCurry at the Picower Institute; and Emanuela Giacometti, Caroline Beard, Dong Dong Fu and Ruth Flannery at the Whitehead Institute.

Chemical drink breathes life into damaged hearts

* 22:00 09 February 2009 **by Andy Coghlan**



After drinking a chemical dissolved in water, mice with damaged hearts turn from couch potatoes into treadmill tearaways, researchers say. The finding raises hopes that the same substance can invigorate patients weakened from heart attacks by increasing the supply of oxygen to damaged cardiac muscle.

Designed to make haemoglobin release more of its oxygen than normal, the drug, myo-inositol trispyrophosphate (ITPP) boosted exercise levels in the ailing mice by 35% when given dissolved in water. When given by injection into the abdomen, exercise levels rose a massive 60%.

"ITPP doesn't deliver oxygen itself, but makes haemoglobin able to release a larger amount of oxygen to tissues," explains Jean-Marie Lehn of the University of Strasbourg in France.

An anterior view of the blood vessels of the upper body (Image: Medical RF.com / SPL)

Normally, he says, haemoglobin releases only 25% of its oxygen cargo during one circuit of the body. But when ITPP binds to haemoglobin, it releases 35% more than usual, boosting supplies of oxygen to tissues without people having to inhale any extra air.

Sports warning

Further evidence of increased oxygen supply came from blood samples taken from the mice showing a fivefold reduction within just three days of hypoxia-inducible factor, a chemical distress signal produced by oxygen-starved tissue. The results also suggested that the effects from a single dose could last almost a week, so patients wouldn't need to take ITPP every day.

Unlike artificial blood substitutes, which have run into practical and ethical problems during clinical trials, ITPP simply boosts the efficiency with which native blood supplies tissues with oxygen. It is also very similar to a naturally occurring chemical made in the body, myo-inositol.

As a result, Lehn hopes to begin clinical trials "as soon as possible". For athletes tempted to use the substance to enhance performance, he warns: "It could be very easily detected."

'Novel approach'

Lehn says that studies are under way to find out exactly how ITPP interferes with haemoglobin to make it give up more oxygen. He says that the substance is not natural, but is related to myo-inositol, a substance found in rice and cereals.

Peter Weissberg, medical director of the British Heart Foundation, says that inadequate oxygen delivery to tissue causes many symptoms of heart failure. "Around 700,000 people in the UK alone are living with heart failure, and inadequate tissue oxygen delivery is the cause of many of the symptoms - particularly fatigue and poor exercise tolerance," he says.

Such a novel approach is welcome, says Weissberg, given the limited success of existing treatments based on increasing heart output or improving blood circulation.

"They've found a molecule that can boost the amount of oxygen transferred from blood to the muscles," he says. "If a similar effect can be achieved in [humans], it will raise the possibility of a new treatment to improve debilitating heart failure symptoms."

Journal reference: Proceedings of the National Academy of Sciences (DOI: 10.1073/pnas.0812381106)

Canadian scientists read minds with infrared scan

Using optical brain imaging, Bloorview researchers decode preference with 80 percent accuracy

Researchers at Canada's largest children's rehabilitation hospital have developed a technique that uses infrared light brain imaging to decode preference – with the goal of ultimately opening the world of choice to children who can't speak or move.

In a study published this month in The Journal of Neural Engineering, Bloorview scientists demonstrate the ability to decode a person's preference for one of two drinks with 80 per cent accuracy by measuring the intensity of near-infrared light absorbed in brain tissue. http://www.iop.org/EJ/abstract/1741-2552/6/1/016003

"This is the first system that decodes preference naturally from spontaneous thoughts," says Sheena Luu, the University of Toronto PhD student in biomedical engineering who led the study under the supervision of Tom Chau, Canada Research Chair in pediatric rehab engineering.

Most brain-computer interfaces designed to read thoughts require training. For example, in order to indicate yes to a question, the person needs to do an unrelated mental task – such as singing a song in their head.

The nine adults in Luu's study received no training. Prior to the study they rated eight drinks on a scale of one to five.

Wearing a headband fitted with fibre-optics that emit light into the pre-frontal cortex of the brain, they were shown two drinks on a computer monitor, one after the other, and asked to make a mental decision about which they liked more. "When your brain is active, the oxygen in your blood increases and depending on the concentration, it absorbs more or less light," Luu says. "In some people, their brains are more active when they don't like something, and in some people they're more active when they do like something."

After teaching the computer to recognize the unique pattern of brain activity associated with preference for each subject, the researchers accurately predicted which drink the participants liked best 80 per cent of the time.

"Preference is the basis for everyday decisions," Luu says. When children with disabilities can't speak or gesture to control their environment, they may develop a learned helplessness that impedes development.

In future, Luu envisions creating a portable, near-infrared sensor that rests on the forehead and relies on wireless technology, opening up the world of choice to children who can't speak or move.

Her work is part of Tom Chau's body-talk research, which aims to give children who are "locked in" by disability a way to express themselves through subtle body processes like breathing pattern, heart rate and brain activity.

Luu notes that the brain is too complex to ever allow decoding of a person's random thoughts. "However, if we limit the context – limit the question and available answers, as we have with predicting preference – then mind-reading becomes possible."

Clinicians override most electronic medication safety alerts

BOSTON--Computer-based systems that allow clinicians to prescribe drugs electronically are designed to automatically warn of potential medication errors, but a new study reveals clinicians often override the alerts and rely instead on their own judgment.

The study, led by investigators at Dana-Farber Cancer Institute and Beth Israel Deaconess Medical Center (BIDMC), suggests that most clinicians find the current medication alerts more of an annoyance than a valuable tool. The authors conclude that if electronic prescribing is to effectively enhance patient safety, significant improvements are necessary. The study's findings appear in the Feb. 9 issue of the Archives of Internal Medicine.

"Electronic prescribing clearly will improve medication safety, but its full benefit will not be realized without the development and integration of high-quality decision support systems to help clinicians better manage medication safety alerts," says the study's senior author Saul Weingart, MD, PhD, vice president for patient safety at Dana-Farber and an internist at BIDMC.

The researchers reviewed the electronic prescriptions and associated medication safety alerts generated by 2,872 clinicians at community-based outpatient practices in Massachusetts, New Jersey, and Pennsylvania to learn how clinicians responded to the alerts.

The clinicians submitted 3.5 million electronic prescriptions between Jan. 1, and Sept. 30, 2006. Approximately one in 15 prescription orders, or 6.6 percent, produced an alert for a drug interaction or a drug allergy. The vast majority of the 233,537 alerts (98.6 percent) were for a potential interaction with a drug a patient already takes.

Clinicians overrode more than 90 percent of the drug interaction alerts and 77 percent of the drug allergy alerts. Even when a drug interaction alert was rated with high severity, clinicians typically dismissed those for medications commonly used in combination to treat specific diseases. They also were less likely to accept an alert if the patient had previously been treated with the medication.

The high override rate of all alerts, the researchers contend, suggests that the utility of electronic medication alerts is inadequate, adding that for some clinicians, most alerts "may be more of a nuisance than an asset."

"The sheer volume of alerts generated by electronic prescribing systems stands to limit the safety benefits," says Thomas Isaac, MD, MBA, MPH, of BIDMC and Dana-Farber and the paper's first author. "Too many alerts are generated for unlikely events, which could lead to alert fatigue. Better decision support programs will generate more pertinent alerts, making electronic prescribing more effective and safer."

Although the study analyzed orders generated on only one electronic prescribing system, PocketScript, the researchers say their observations are relevant to other systems because the alerts they reviewed were typical and were generated by a commercial database, Cerner Multum, used by other electronic prescribing systems.

Based on these findings, Weingart and his colleagues offer several recommendations to improve medication safety alerts, including reclassifying severity of alerts, especially those that are frequently overridden; providing an option for clinicians to suppress alerts for medications a patient already has received; and customizing the alerts for a clinician's specialty. The research team identified a list of potentially dangerous drug interactions

based on those alerts that most often changed the clinicians' decision to prescribe. This list is available at www.dana-farber.org/electronic-medication-safety.

"We need to find a way to help clinicians to separate the proverbial wheat from the chaff," says Weingart. "Until then, electronic prescribing systems stand to fall far short of their promise to enhance patient safety and to generate greater efficiencies and cost savings."

In addition to Weingart and Isaac, the paper's other authors are Joel Weissman, PhD, Executive Office of Health and Human Services, Commonwealth of Massachusetts; Roger Davis, ScD, BIDMC; Daniel Sands, MD, MPH, BIDMC and Cisco Systems, San Jose, Calif.; Michael Massagli, PhD, PatientsLikeMe, Inc., Cambridge, Mass.; and Adrienne Cyrulik, MPH, Blue Cross Blue Shield of Massachusetts, Boston.

The research was supported by a grant from the Physicians' Foundation for Health Systems Excellence, Boston.

Study: fluid buildup in lungs is part of the damage done by the flu

Columbus, Ohio – In a fight against respiratory infections, the body typically produces a little fluid to help the lungs generate a productive cough. But new research suggests that the influenza virus can tip the balance toward too much fluid in the lungs, interfering with the supply of oxygen to the rest of the body.

An immune response ultimately is needed to eliminate the virus, but this research suggests that it's not the presence of the virus alone that does all the harm to a sick person. Instead, the fluid buildup deep inside the lungs might help kill a person infected with the flu, according to the research, which was conducted in mice.

Ian Davis

"My take is that when people die of these illnesses, they're dying because they can't breathe," said Ian Davis, assistant professor of veterinary biosciences at Ohio State University and senior author of the study. "If the lungs aren't working well, then it doesn't matter whether a week from now you can make an immune response and clear the virus if you can't survive that long because you just can't get oxygen."

Detailing exactly how flu interferes with fluid clearance in the lungs lays the groundwork for a second phase of related studies to test a new therapy – a drug that is already known to regulate the amount of fluid that builds up in infected lungs.

In the event of a flu pandemic, such a drug, which scientists hope could be inhaled by mouth, might be an important supplement to antiviral medications and ventilators, Davis said.

The research is published in a recent issue of the American Journal of Respiratory and Critical Care Medicine.

Davis and colleagues infected mice with high doses of an influenza A virus to compare oxygen levels and fluid clearance in infected mice to those of healthy mice.

Mice that were infected with the highest doses of flu experienced a steady decline in oxygen in their blood and higher levels of fluid in the lungs. Two days after infection, fluid clearance in these mice was only half as effective as fluid clearance from the lungs of uninfected mice.

Lung infections have been known to cause problems with what is called alveolar fluid clearance, but the effects of the flu on the lungs had not been tested before, Davis said. The alveoli are air spaces deep inside the lungs where oxygen enters the blood in exchange for carbon dioxide to be exhaled.

The scientists used an unusual method to observe the fluid clearance. After being infected, the mice were anesthetized and put on ventilators. The researchers then placed fluid containing protein into one lung of each mouse and tested the fluid 30 minutes later. The amount of protein left in the remaining fluid allowed the investigators to determine whether the infected lung was clearing fluid adequately.

Davis said the study showed that when the flu virus infects cells in the lung, those cells release small molecules, or nucleotides, that are part of the energy-producing and replication machinery of the cell. Those nucleotides then bind to receptors of other cells in a series of events that ultimately shut down the transport of sodium from airways to the blood. All of these interactions take place in the epithelium, the lining of the airways in the lungs.

Under normal conditions, sodium is absorbed from the lungs into the blood and carried away to be excreted by the kidneys. But in infected lungs, when the sodium channels are damaged, more and more fluid builds up in air spaces instead of being pumped across the airway lining and into the blood.

"A little bit of fluid gives us a way to flush the nasties out of the lung," Davis said. "If you get a bacterial infection and bacteria are all over the lungs and cell surfaces, you release these nucleotides and generate fluid so you can cough up the bacteria and wash them out. The downside is as you build up fluid in the lung, you can't oxygenate as well.

"If the bacteria or virus wins, you get more and more fluid and you get sicker and sicker. But if you can wash it away, then you've won and things go back to normal."

The potential therapy that Davis is investigating was originally used in earlier research to block the generation of these nucleotides to prove the fluid clearance mechanism behind a different viral lung disease.

Now that his newer work has shown that influenza A has the same effect on the lungs, Davis is planning to test the therapy's effects against flu infection.

The first studies will involve delivering the drug in aerosol form to mice. Davis also plans to culture human epithelial cells to see whether these cells in humans have the same role as do mouse cells in the fluid clearance mechanism.

Davis noted that the current health-care system can help manage most serious cases of the flu, even if some of the sicker patients require ventilation to help them clear fluid and get oxygen to the blood.

"But if we get into a pandemic situation and there are 200 people with low levels of oxygen in their blood and five ventilators, 195 people are out of luck. We hope to develop something that those people can take by aerosol that might reduce the need for that kind of ventilation," he said.

This work was supported by the National Institutes of Health and Ohio State's Department of Veterinary Biosciences. Coauthors of the study are Kendra Wolk, Zachary Traylor and Erin Yu of veterinary biosciences at Ohio State; Eduardo Lazarowski of the Department of Medicine at the University of North Carolina, Chapel Hill; Russell Durbin of the Research Institute at Nationwide Children's Hospital; and Nancy Jewell and Joan Durbin of the Research Institute at Nationwide Children's Hospital and Ohio State's Department of Pediatrics.

Mayo Clinic research shows that improving brain processing speed helps memory

Rochester, Minn. -- Mayo Clinic researchers found that healthy, older adults who participated in a computer-based training program to improve the speed and accuracy of brain processing showed twice the improvement in certain aspects of memory, compared to a control group.

"What's unique in this study is that brain-processing activities seemed to help aspects of memory that were not directly exercised by the program -- a new finding in memory research," says Glenn Smith, Ph.D., Mayo Clinic neuropsychologist and lead researcher on the study.

The research, a controlled, multisite, double-blind study, will be published in the April issue of the Journal of the American Geriatrics Society. A copy is available online Feb. 9, 2009.

For an hour a day, five days a week for eight weeks, study participants worked on computer-based activities in their homes. The participants, from Minnesota and California, were age 65 or older. No one had a diagnosis of cognitive impairment, such as early Alzheimer's disease.

The control group, with 245 adults, watched educational videos on art, history and literature topics. They completed quizzes on the content.

The experimental therapy group, with 242 adults, completed six auditory exercises designed to help the brain improve the speed and accuracy of processing. For example, participants were asked to distinguish between high- and low-pitched sounds. To start, the sounds were slow and distinct. Gradually, the speed increased and separation disappeared.

"The sounds go faster and faster, until it ends up sounding almost like a click," says Dr. Smith. The difficulty increases only as participants master each step with 85 percent accuracy. Other exercises, such as matching or distinguishing between similar-sounding words, for example, pop and pot, also were part of the skill building.

The commercially available program was developed by Posit Science, San Francisco company that financed the research. Mayo Clinic researchers do not have financial ties to this business.

At the end of eight weeks, researchers used a standardized tool to measure participants' memory changes. Called the Repeatable Battery for the Assessment of Neuropsychological Status, it includes tasks such as repeating words or numbers after hearing them once.

"We found that the improvement in these skills was significantly greater in the experimental group -- about double," says Dr. Smith.

Participants in the experimental group self-reported memory improvement, too, indicating the change was noticeable in day-to-day tasks.

While the study results are statistically significant, Dr. Smith says it is important to understand the extent of the memory boost. Collectively, the experimental group's memory function increased about 4 percent over the baseline measured at the study's onset. The control group's overall memory gain was about 2 percent.

But, Dr. Smith says, because participants were in generally good health, the results don't offer insights on preventing Alzheimer's or other forms of dementia.

Results indicate that aging adults may be able to make better-informed decisions about ways to improve memory. "Brain processing speed slows as we age," says Dr. Smith. "The study indicates that choosing a memory-enhancing approach that focuses on improving brain processing speed and accuracy, rather than memory retention, may be helpful."

There's no harm in trying other approaches -- mnemonics, workshops or even doing crosswords or playing piano, he says, but there's little evidence these methods sustain benefits in memory.

Other researchers involved in this study include: Patricia Housen, Ph.D., and Elizabeth Zelinski, Ph.D., both with Leonard Davis School of Gerontology, University of Southern California, Los Angeles; Kristine Yaffe, M.D., University of California, San Francisco; Ronald Ruff, Ph.D., Stanford University, Stanford, Calif.; Robert Kennison, Ph.D., California State University, Los Angeles; and Henry Mahncke, Ph.D., Posit Science Corporation, San Francisco. His affiliation with this company is noted in the journal article.

Did increased gene duplication set the stage for human evolution?

Roughly 10 million years ago, a major genetic change occurred in a common ancestor of gorillas, chimpanzees, and humans. Segments of DNA in its genome began to form duplicate copies at a greater rate than in the past, creating an instability that persists in the genome of modern humans and contributes to diseases like autism and schizophrenia. But that gene duplication also may be responsible for a genetic flexibility that has resulted in some uniquely human characteristics.

"Because of the architecture of the human genome, genetic material is constantly being added and deleted in certain regions," says Howard Hughes Medical Institute investigator and University of Washington geneticist Evan Eichler, who led the project that uncovered the new findings. "These are really like volcanoes in the genome, blowing out pieces of DNA." The research was published in the February 12, 2009, issue of Nature.

Eichler and his colleagues focused on the genomes of four different species: macaques, orangutans, chimpanzees, and humans. All are descended from a single ancestral species that lived about 25 million years ago. The line leading to macaques broke off first, so that macaques are the most distantly related to humans in evolutionary terms. Orangutans, chimpanzees, and humans share a common ancestor that lived 12-16 million years ago. Chimps and humans are descended from a common ancestral species that lived about 6 million years ago.

By comparing the DNA sequences of the four species, Eichler and his colleagues identified gene duplications in the lineages leading to these species since they shared a common ancestor. They also were able to estimate when a duplication occurred from the number of species sharing that duplication. For example, a duplication observed in orangutan, chimpanzees, and humans but not in macaques must have occurred sometime after 25 million years ago but before the orangutan lineage branched off.

Eichler's research team found an especially high rate of duplications in the ancestral species leading to chimps and humans, even though other mutational processes, such as changes in single DNA letters, were slowing down during this period. "There's a big burst of activity that happens where genomes are suddenly rearranged and changed," he says. Surprisingly, the rate of duplications slowed down again after the lineages leading to humans and to chimpanzees diverged. "You might like to think that humans are special because we have more duplications than did earlier species," he says, "but that's not the case."

These duplications have created regions of our genomes that are especially prone to large-scale reorganizations. "That architecture predisposes to recurrent deletions and duplications that are associated with autism and schizophrenia and with a whole host of other diseases," says Eichler.

Yet these regions also exhibit signs of being under positive selection, meaning that some of the rearrangements must have conferred advantages on the individuals who inherited them. Eichler thinks that uncharacterized genes or regulatory signals in the duplicated regions must have created some sort of reproductive edge. "I believe that the negative selection of these duplications is being outweighed by the selective advantage of having these newly minted genes, but that's still unproven," he said.

An important task for future studies is to identify the genes in these regions and analyze their functions, according to Eichler. "Geneticists have to figure out the genes in these regions and how variation leads to different aspects of the human condition such as disease. Then, they can pass that information on to neuroscientists and physiologist and biochemists who can work out what these proteins are and what they do," he says. "There is the possibility that these genes might be important for language or for aspects of cognition, though much more work has to be done before we'll be able to say that for sure."

Pubic hair provides evolutionary home for gorilla lice

There are two species of lice that infest humans: pubic lice, Pthirus pubis, and human head and body lice, Pediculus humanus. A new article in BioMed Central's open access Journal of Biology suggests one explanation for the separation of the two species.

In the article, Robert Weiss from University College London describes how he was struck by inspiration while pondering the question of why lice would separate into two groups when our ancestors are quite uniformly hairy, "I was having difficulty envisioning a clear separation of habitats between the groin and other parts of

our ancient common ancestor. My 'eureka moment' came, appropriately enough, in the shower: although naked apes have pubic hair, surely our hairy cousins don't?"
Pthirus pubis, popularly known as crabs, evolved from the structurally similar gorilla louse, Pthirus gorillae. Interestingly however, while genetic analysis carried out by David Reed at the University of Florida indicates that this split occurred around 3.3 million years ago, humans are believed to have diverged from gorillas much earlier - at least 7 million years ago - suggesting that early humans somehow caught pubic lice from their gorilla cousins. Happily, this may not be as sordid as it sounds. According to Weiss, "Before one conjures up a King Kong scenario, it should be noted that predators can pick up parasites from their prey. The close contact involved in human ancestors butchering gorillas could have enabled Pthirus to jump hosts, rather as bushmeat slaughter practices allowed HIV to invade humans from chimpanzees in modern times."
So, while head lice may be viewed as a 'family heirloom', inherited down the generations as humans have evolved, pubic lice may well be a recent and slightly unwelcome gift from the more hirsute branch of our evolutionary family.

Notes to Editors Article available at journal website: http://jbiol.com/content/8/2/20

1. Apes, lice and prehistory Robin A Weiss Journal of Biology 2009, 8:20 doi:10.1186/jbiol114

Y chromosome and surname study challenges infidelity 'myth'

Our surnames and genetic information are often strongly connected, according to a study funded by the Wellcome Trust. The research, published this week in the journal Molecular Biology and Evolution, may help genealogists create more accurate family trees even when records are missing. It also suggests that the often quoted "one in ten" figure for children born through infidelity is unlikely to be true.

Dr Turi King and Professor Mark Jobling from the University of Leicester examined the Y chromosomes of over 1,600 unrelated men with forty surnames (including variations in spelling). Sons inherit both the Y chromosome and – generally – the surname from their fathers, unlike daughters, who do not carry this sex-specific chromosome and usually change their surname through marriage.

Hereditary surnames were introduced to Britain by the Normans at the time of the conquest. The practice of using hereditary surnames filtered down from Norman noble families to all classes of society so that by the fourteenth century people in many classes had surnames and by the sixteenth century it was rare not to have one.

Dr King and Professor Jobling found that men with rare surnames – such as Grewcock, Wadsworth, Ketley and Ravenscroft – tended to share Y chromosomes that were very similar, suggesting a common ancestor within the past 700 years. However, men with common surnames, such as Smith, were no more likely to have such a common ancestor than men chosen at random from the general population.

"Surnames such as Smith come from a person's trade and would have been adopted many times by unrelated people," explains Dr King. "Less common names, such as Swindlehurst, were more geographically-specific and possibly adopted by only one or two men, so we would expect people with these surnames to be more closely related."

One of the most familiar of the rarer names in the study was Attenborough. A random sample of Attenboroughs – including derivations such as Attenborrow – found that almost nine out of ten of these men share the same Y chromosome type.

"Attenboroughs essentially form one big family of distant relatives," says Dr King. "The Y chromosome type was the same even across spelling variants, which confirms that the spellings of names were formalised only relatively recently."

Dr King believes that these findings will help genealogists in their efforts to populate their family trees, particularly when parish records and other documents are incomplete. A genetic test of two people with a common surname would show whether they share a paternal ancestor.

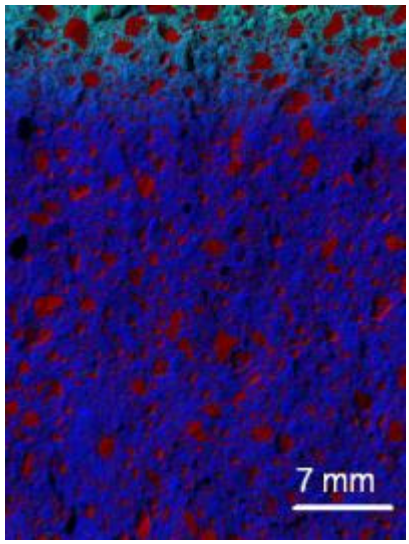
The researchers also looked at whether the Y chromosome-surname link could provide information about historical rates of children born illegitimately. People with a rare surname are very likely to be related as the surname is likely to have been adopted by only one or two men initially, so anyone now sharing this surname but with a different Y chromosome to the majority is likely to have an ancestor born illegitimately.

"People often quote a figure of one in ten for the number of people born illegitimately," says Professor Jobling. "Our study shows that this is likely to be an exaggeration. The real figure is more likely to be less that one in twenty-five."

The study follows on from previous research from the two researchers into the link between surnames and the Y chromosome. A previous study showed that it may be possible to apply the research to forensic science, extrapolating from a DNA sample to identify likely surnames of suspects.

Viscosity-enhancing nanomaterials may double service life of concrete

Engineers at the National Institute of Standards and Technology (NIST) are patenting a method that is expected to double the service life of concrete. The key, according to a new paper*, is a nano-sized additive that slows down penetration of chloride and sulfate ions from road salt, sea water and soils into the concrete. A reduction in ion transport translates to reductions in both maintenance costs and the catastrophic failure of concrete structures. The new technology could save billions of dollars and many lives.



Concrete has been around since the Romans, and it is time for a makeover. The nation's infrastructure uses concrete for millions of miles of roadways and 600,000 bridges, many of which are in disrepair. In 2007, 25 percent of U.S. bridges were rated as structurally deficient or functionally obsolete, according to the Federal Highway Administration. Damaged infrastructure also directly affects large numbers of Americans' own budgets. The American Society of Civil Engineers estimates that Americans spend $54 billion each year to repair damages caused by poor road conditions.

Infiltrating chloride and sulfate ions cause internal structural damage over time that leads to cracks and weakens the concrete.

The barely visible blue-green area at the top of this X-ray image of concrete with the NIST nanoadditive shows that very few chloride ions (in green) penetrate into the concrete. **NIST**

Past attempts to improve the lifetime of concrete have focused on producing denser, less porous concretes, but unfortunately these formulations have a greater tendency to crack. NIST engineers took a different approach, setting out to double the material's lifetime with a project called viscosity enhancers reducing diffusion in concrete technology (VERDICT). Rather than change the size and density of the pores in concrete, they reasoned, it would be better to change the viscosity of the solution in the concrete at the microscale to reduce the speed at which chlorides and sulfates enter the concrete. "Swimming through a pool of honey takes longer than making it through a pool of water," engineer Dale Bentz says.

They were inspired by additives the food processing industry uses to thicken food and even tested out a popular additive called xanthum gum that thickens salad dressings and sauces and gives ice cream its texture.

Studying a variety of additives, engineers determined that the size of the additive's molecule was critical to serving as a diffusion barrier. Larger molecules such as cellulose ether and xanthum gum increased viscosity, but did not cut diffusion rates. Smaller molecules - less than 100 nanometers - slowed ion diffusion. Bentz explains, "When additive molecules are large but present in a low concentration, it is easy for the chloride ions to go around them, but when you have a higher concentration of smaller molecules increasing the solution viscosity, it is more effective in impeding diffusion of the ions."

The NIST researchers have demonstrated that the additives can be blended directly into the concrete with current chemical admixtures, but that even better performance is achieved when the additives are mixed into the concrete by saturating absorbant, lightweight sand. Research continues on other materials as engineers seek to improve this finding by reducing the concentration and cost of the additive necessary to double the concrete's service life.

A non-provisional patent application was filed in September, and the technology is now available for licensing from the U.S. government; the NIST Office of Technology Partnerships can be contacted for further details (Contact: Terry Lynch, terry.lynch@nist.gov, (301) 975-2691).

** D.P. Bentz, M.A. Peltz, K.A. Snyder and J.M. Davis. VERDICT: Viscosity Enhancers Reducing Diffusion in Concrete Technology. Concrete International. 31 (1), 31-36, January 2009.*

Penn Study Shows Why Sleep is Needed to Form Memories

First-of-its-kind study shows how brain connections strengthen during sleep

Philadelphia – If you ever argued with your mother when she told you to get some sleep after studying for an exam instead of pulling an all-nighter, you owe her an apology, because it turns out she's right. And now, scientists are beginning to understand why.

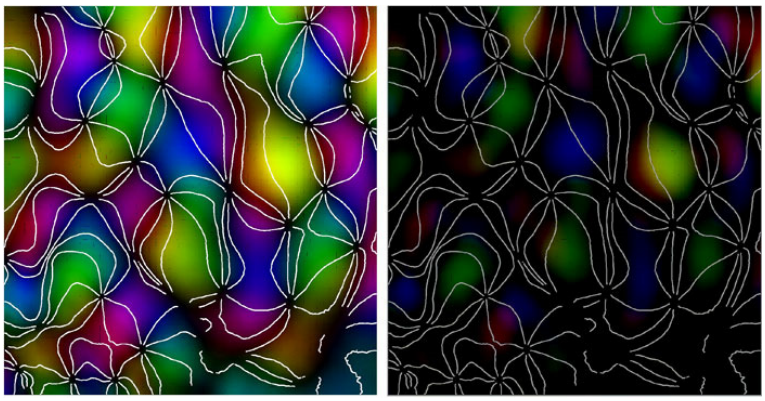
In research published this week in Neuron, Marcos Frank, PhD, Assistant Professor of Neuroscience, at the University of Pennsylvania School of Medicine, postdoctoral researcher Sara Aton, PhD, and colleagues describe for the first time how cellular changes in the sleeping brain promote the formation of memories.

"This is the first real direct insight into how the brain, on a cellular level, changes the strength of its connections during sleep," Frank says.

The findings, says Frank, reveal that the brain during sleep is fundamentally different from the brain during wakefulness.

"We find that the biochemical changes are simply not happening in the neurons of animals that are awake," Frank says. "And when the animal goes to sleep it's like you've thrown a switch, and all of a sudden, everything is turned on that's necessary for making synaptic changes that form the basis of memory formation. It's very striking."

The team used an experimental model of cortical plasticity – the rearrangement of neural connections in response to life experiences. "That's fundamentally what we think the machinery of memory is, the actual making and breaking of connections between neurons," Frank explains



The world as the brain sees it. Optical 'polar' maps of the visual cortex are generated by measuring micro-changes in blood oxygenation as the left eye (left panel) or right eye is stimulated by bars of light of different orientations (0-180 degrees). The cortical response to each stimulus is pseudo-colored to represent the orientation that best activates visual cortical neurons. If vision is blocked in an eye (the right eye in this example) during a critical period of development, neurons no longer respond to input from the deprived eye pathway (indicated by a loss of color in the right panel) and begin to respond preferentially to the non-deprived eye pathway. These changes are accompanied by alterations in synaptic connections in single neurons. This process, known as ocular dominance plasticity, is enhanced by sleep via activation of NMDA receptors and intracellular kinase activity. Through these mechanisms, sleep strengthens synaptic connections in the non-deprived eye pathway. **Marcos Frank, PhD University of Pennsylvania**

In this case, the experience Frank and his team used was visual stimulation. Animals that were young enough to still be establishing neural networks in response to visual cues were deprived of stimulation through one eye by covering that eye with a patch. The team then compared the electrophysiological and molecular changes that resulted with control animals whose eyes were not covered. Some animals were studied immediately following the visual block, while others were allowed to sleep first.

From earlier work, Frank's team already knew that sleep induced a stronger reorganization of the visual cortex in animals that had an eye patch versus those that were not allowed to sleep. Now they know why.

A molecular explanation is emerging. The key cellular player in this process is a molecule called N-methyl D-aspartate receptor (NMDAR), which acts like a combination listening post and gate-keeper. It both receives extracellular signals in the form of glutamate and regulates the flow of calcium ions into cells.

Essentially, once the brain is triggered to reorganize its neural networks in wakefulness (by visual deprivation, for instance), intra- and intercellular communication pathways engage, setting a series of enzymes into action within the reorganizing neurons during sleep.

To start the process, NMDAR is primed to open its ion channel after the neuron has been excited. The ion channel then opens when glutamate binds to the receptor, allowing calcium into the cell. In turn, calcium, an intracellular signaling molecule, turns other downstream enzymes on and off.

Some neural connections are strengthened as a result of this process, and the result is a reorganized visual cortex. And, this only happens during sleep.

"To our amazement, we found that these enzymes never really turned on until the animal had a chance to sleep," Frank explains, "As soon as the animal had a chance to sleep, we saw all the machinery of memory start to engage." Equally important was the demonstration that inhibition of these enzymes in the sleeping brain completely prevented the normal reorganization of the cortex.

Frank stresses that this study did not examine recalling memories. For example, these animals were not being asked to remember the location of their food bowl. "It's a mechanism that we think underlies the formation of memory." And not only memory; the same mechanism could play a role in all neurological plasticity processes.

As a result, this study could pave the way to understanding, on a molecular level, why humans need sleep, and why they are so affected by the lack of it. It could also conceivably lead to novel therapeutics that could compensate for the lack of sleep, by mimicking the molecular events that occur during sleep.

Finally, the study could lead to a deeper understanding of human memory. Though how and even where humans store long-lasting memories remains a mystery, Frank says, "we do know that changes in cortical connections is at the heart of the mystery. By understanding that in animal models, it will bring us close to understanding how it works in humans."

The research was funded by the National Institutes of Health, the National Sleep Foundation, and L'Oreal USA, and also involved researchers at the Penn's Center for Sleep and Respiratory Neurobiology, and the School of Life Sciences, Jawaharlal Nehru University, New Delhi, India.

Researchers crack the code of the common cold

Study also sheds light on the suspected cause of asthma and acute asthma attacks

Scientists have begun to solve some of the mysteries of the common cold by putting together the pieces of the genetic codes for all the known strains of the human rhinovirus. Researchers at the University of Maryland School of Medicine in Baltimore and colleagues at the University of Wisconsin-Madison have completed the genomic sequences of the viruses and assembled them into a "family tree," which shows how the viruses are related, with their commonalities and differences. The study will be released on the online version of the journal Science (Science Express) at 2 p.m. EST on February 12.

The researchers say this work provides a powerful tool that may lead to the development of the first effective treatments against the common cold.

"There has been no success in developing effective drugs to cure the common cold, which we believe is due to incomplete information about the genetic composition of all these strains," says the study's senior author, Stephen B. Liggett, M.D., professor of medicine and physiology at the University of Maryland School of Medicine and director of its Cardiopulmonary Genomics Program.

"We generally think of colds as a nuisance, but they can be debilitating in the very young and in older individuals, and can trigger asthma attacks at any age. Also, recent studies indicate that early rhinovirus infection in children can program their immune system to develop asthma by adolescence," says Dr. Liggett, who is a pulmonologist and molecular geneticist.

Major discoveries of the study

The researchers found that human rhinoviruses are organized into about 15 small groups that come from distant ancestors. The discovery of these multiple groups explains why a "one drug fits all" approach for anti-viral agents does not work. But, says Dr. Liggett, "Perhaps several anti-viral drugs could be developed, targeted to specific genetic regions of certain groups. The choice of which drug to prescribe would be based on the genetic characteristics of a patient's rhinovirus infection."

Dr. Liggett adds that while anti-viral drugs seem to be the most likely to succeed, "the data gathered from these full genome sequences gives us an opportunity to reconsider vaccines as a possibility, particularly as we gather multiple-patient samples and sequence the entire genomes, to see how frequently they mutate during a cold season. That work is underway now."

The researchers also found that the human rhinovirus skips a step when it makes its protein product, a shortcut that probably speeds up its ability to make a person feel sick soon after infection. "This is a new insight," says co-investigator Claire M. Fraser-Liggett, Ph.D., director of the Institute for Genome Sciences and professor of medicine and microbiology at the University of Maryland School of Medicine. "We would not have had any sort of intuition about this had it not been revealed through genome analysis. Information that comes from this discovery might present a completely different approach in terms of therapy."

The analysis shows that some human rhinoviruses result from the exchange of genetic material between two separate strains of the virus that infect the same person. Such a swap, known as recombination, was previously not thought possible in human rhinovirus. During cold season, when many different strains of rhinovirus may be causing infections, recombination could rapidly produce new strains.

Multiple mutations (as many as 800) were evident in virus samples taken recently from patients with colds, compared to older rhinovirus reference strains. Some viruses mutate by making slight changes in certain proteins to avoid being destroyed by antibodies from a person's immune system. "Mutations were found in every area of the genome," says Dr. Liggett.

The study's lead author, Ann C. Palmenberg, Ph.D., professor of biochemistry and chair of the Institute for Molecular Virology at the University of Wisconsin-Madison, notes, "As we begin to accumulate additional samples from a large number of patients, it is likely that hotspots for mutation or recombination will become apparent, and other regions resistant to mutational change may emerge. This will provide clues as to how flexible the virus is as it responds to the human environment, important hints if you are designing new therapeutics."

Study background

Human rhinovirus infection is responsible for half of all asthma attacks and is a factor in bronchitis, sinusitis, middle ear infections and pneumonia. The coughs, sneezes and sniffles of colds impose a major health care burden in the United States - including visits to health care providers, cost of over-the-counter drugs for

symptom relief, often-inappropriate antibiotic prescriptions and missed work days - with direct and indirect costs of about $60 billion annually.

Prior to the start of this project, the genomes of only a few dozen rhinoviruses had been sequenced from what was considered the reference library, a frozen collection of 99 different rhinovirus strains taken from patients over a span of more than two decades. During this team's work, several other groups began to report the full genomes of some of these viruses, as well as some odd rhinovirus-like strains from relatively sick patients.

"It was clear to us that the spectrum of rhinoviruses out there was probably much greater than we realized. Further, we needed to develop a framework from which we could begin to figure out ways to combat these viruses and use their genetic signatures to predict how a specific virus would affect a patient," says Dr. Fraser-Liggett.

The current study adds 80 new full genome sequences to the rhinovirus library and 10 more acquired recently from people with colds. Each sequence was modeled and compared to each other. Dr. Liggett says, "Now we can put together many pieces of the human rhinovirus puzzle to help us answer some fundamental questions: how these rhinoviruses might mutate as they spread from one person to another; which rhinoviruses are more associated with asthma exacerbations and why rhinovirus exposure in infancy may cause asthma later in life. With all this information at hand, we see strong potential for the development of the long-sought cure for the common cold, using modern genomic and molecular techniques."

"With recent improvements in technology, including next-generation DNA sequencing tools, it has become easier to generate whole genome sequence information," says Dr. Fraser-Liggett. "There is no reason any longer to focus on a very limited part of the rhinovirus molecule to learn what it's doing, what the predominant strain is in a population, or to try to infer what the evolution of the entire molecule might be. Instead, by studying the complete genome sequence, we can answer multiple questions in parallel."

Researchers from the J. Craig Venter Institute also contributed to this study.
The University of Maryland School of Medicine funded this project.
Palmenberg AC, Spiro D, Kuzmickas R, Wang S, Djikeng A, Rathe JA, Fraser-Liggett CM, Liggett SB. "Sequencing and Analyses of All Known Human Rhinovirus Genomes Reveals Structure and Evolution." Science. Published online ahead of print, February 12, 2009.

Astronomers Unveiling Life's Cosmic Origins

Processes that laid the foundation for life on Earth -- star and planet formation and the production of complex organic molecules in interstellar space -- are yielding their secrets to astronomers armed with powerful new research tools, and even better tools soon will be available. Astronomers described three important developments at a symposium on the "Cosmic Cradle of Life" at the annual meeting of the American Association for the Advancement of Science in Chicago, IL.

In one development, a team of astrochemists released a major new resource for seeking complex interstellar molecules that are the precursors to life. The chemical data released by Anthony Remijan of the National Radio Astronomy Observatory (NRAO) and his university colleagues is part of the Prebiotic Interstellar Molecule Survey, or PRIMOS, a project studying a star-forming region near the center of our Milky Way Galaxy.

PRIMOS is an effort of the National Science Foundation's Center for Chemistry of the Universe, started at the University of Virginia (UVa) in October 2008, and led by UVa Professor Brooks H. Pate. The data, produced by the NSF's Robert C. Byrd Green Bank Telescope (GBT) in West Virginia, came from more than 45 individual observations totalling more than nine Gigabytes of data and over 1.4 million individual frequency channels.

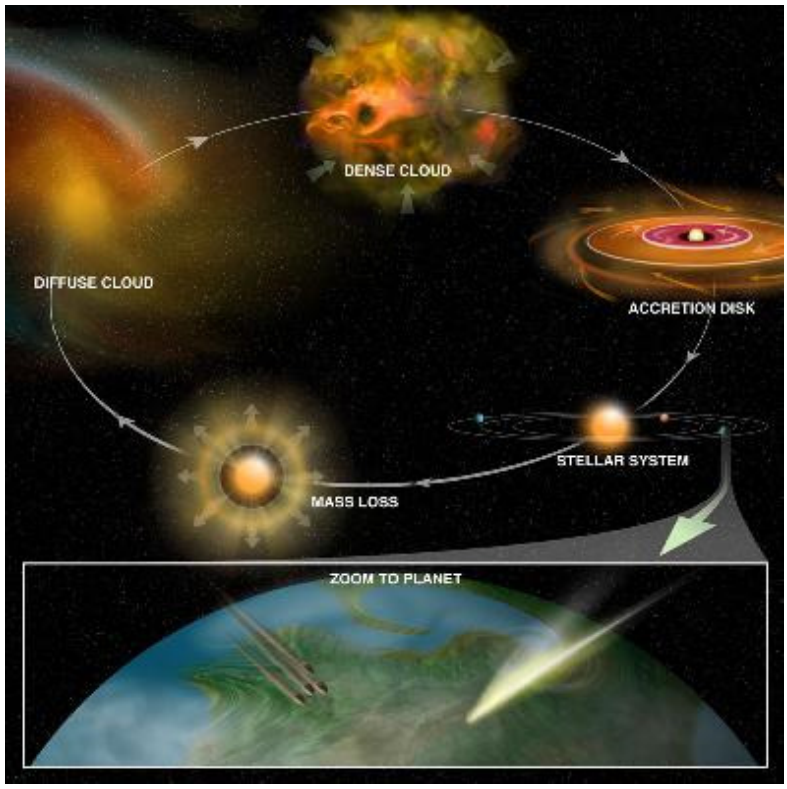
Scientists can search the GBT data for specific radio frequencies, called spectral lines -- telltale "fingerprints" -- naturally emitted by molecules in interstellar space. "We've identified more than 720 spectral lines in this collection, and about 240 of those are from unknown molecules," Remijan said. He added, "We're making available to all scientists the best collection of data below 50 GHz ever produced for the study of interstellar chemistry," Remijan said.

Astronomers have already identified more than 150 molecules in interstellar space in the past 40 years, including complex organic compounds such as sugars and alcohols. "This is a major change in how we search for molecules in space," Remijan explained. "Before, people decided beforehand which molecules they were looking for, then searched in a very narrow band of radio frequencies emitted by those molecules. In this GBT survey, we've observed a wide range of frequencies, collected the data and immediately made it publicly available. Scientists anywhere can 'mine' this resource to find new molecules," he said.

Another key development, presented by Crystal Brogan of the NRAO, showed that highly-detailed images of "protoclusters" of massive young stars reveal a complex mix of stars in different stages of formation, complicated gas motions, and numerous chemical clues to the physical conditions in such stellar nurseries. "We saw a much more complex picture than we had expected and now have new questions to answer," she said.

Using the Smithsonian Astrophysical Observatory's Submillimeter Array (SMA) in Hawaii, Brogan and her colleagues studied a nebula 5,500 light-years from Earth in the constellation Scorpius where stars significantly more massive than our Sun are forming. "It's essential to understand what's going on in systems like this because most stars, Sun-like stars included, form in clusters," Brogan said.

"The most massive stars in the cluster have a tremendous impact on the formation and environment of the rest of the cluster, including the less-massive stars and their planets," Brogan said, adding that "if we want to understand how solar systems that could support life form and evolve, we need to know how these giant stars affect their environment."



The Cosmic Chemistry Cycle Bill Saxton, NRAO/AUI/NSF

Also, Brogan said, the massive young stars are surrounded by "hot cores" that include copious organic material that later may be spewed into interstellar space by stellar winds and other processes. This can help "seed" star-forming regions with some of the chemicals found by the GBT and other telescopes.

Narrowing in on the problem of how planets form around young stars, David Wilner of the Harvard-Smithsonian Center for Astrophysics presented observations with the SMA that revealed new details of solar systems in the earliest stages of their formation. Wilner and his colleagues studied nine dusty disks surrounding young stars in a region in the constellation Ophiuchus.

"These are the most detailed images of such disks made at these wavelengths," Wilner said. The images show the distribution of material on the same size scale as our own Solar System, and indicate that these disks are capable of producing planetary systems. Two of the disks show large central cavities where young planets may already have swept out the material from their neighborhoods.

"Before, we knew that such disks have enough material to form solar systems. These new images tell us that material is in the right places to form solar systems. We're getting a tantalizing peek at the very earliest stages of planet formation," said Sean Andrews, a Hubble Fellow at the CfA.

All three areas of study are poised for major advances with the impending arrival of powerful new radio-telescope facilities such as the Atacama Large Millimeter/submillimeter Array (ALMA) and the Expanded Very Large Array (EVLA), and new capabilities for the GBT.

Studies of protoplanetary disks and young solar systems will benefit greatly from the groundbreaking new capabilities of ALMA, Wilner said. "While we've been able to study a few of these objects so far, ALMA will be able to give us highly-detailed images of many more that we can't study today," he said. Wilner added that ALMA also will likely provide new information on the chemicals in those still-forming planetary systems.

The complex motions and chemistry of Brogan's protoclusters of young, massive stars, also will become much clearer with ALMA. "Both the detail of the images and the ability to find molecular spectral lines will improve by a factor of at least 25 with ALMA," she said. In addition, the increased power of the EVLA will give astronomers a far better look into the inner regions of the disks around young stars -- regions obscured to telescopes operating at shorter wavelengths.

"We know that complex chemicals exist in interstellar space before stars and planets form. With the new research tools coming in the next few years, we're on the verge of learning how the chemistry of the interstellar clouds, the young stars and their environments, and the disks from which planets are formed is all linked together to provide the chemical basis for life on those planets," Remijan explained.

Astrophysicist Neil deGrasse Tyson of the American Museum of Natural History noted, "Like no other science, astrophysics cross-pollinates the expertise of chemists, biologists, geologists and physicists, all to discover the past, present, and future of the cosmos -- and our humble place within it."

The National Radio Astronomy Observatory is a facility of the National Science Foundation, operated under cooperative agreement by Associated Universities, Inc.

Psychoactive compound activates mysterious receptor

MADISON — A hallucinogenic compound found in a plant indigenous to South America and used in shamanic rituals regulates a mysterious protein that is abundant throughout the body, University of Wisconsin-Madison researchers have discovered.

The finding, reported in the Feb. 13 issue of Science, may ultimately have implications for treating drug abuse and/or depression. Many more experiments will be needed, the researchers say.

Scientists have been searching for years for naturally occurring compounds that trigger activity in the protein, the sigma-1 receptor. In addition, a unique receptor for the hallucinogen, called dimethyltryptamine (DMT), has never been identified.

The UW-Madison researchers made the unusual pairing by doing their initial work the "old-fashioned," yet still effective, way. They diagrammed the chemical structure of several drugs that bind to the sigma-1 receptor, reduced them to their simplest forms and then searched for possible natural molecules with the same features. Biochemical, physiological and behavioral experiments proved that DMT does, in fact, activate the sigma-1 receptor.

"We have no idea at present if or how the sigma-1 receptor may be connected to hallucinogenic activity," says senior author Arnold Ruoho, chair of pharmacology at the UW-Madison School of Medicine and Public Health. "But we believe that the National Institute on Drug Abuse (NIDA) may be interested in biological mechanisms underlying psychoactive and addictive drug action."

In addition to being a component of psychoactive snuffs and sacramental teas used in native religious practices in Latin America, DMT is known to be present in some mammalian tissues, and it has also been identified in mammalian blood and spinal fluid. Elevated levels of DMT and a related molecule have been found in the urine of schizophrenics.

Ruoho speculates that the hallucinogen's involvement may mean that the sigma-1 receptor is connected in some fashion to psychoactive behavior. When his team injected DMT into mice known to have the receptor, the animals became hyperactive; mice in which the receptor had been genetically removed did not.

"Hyperactive behavior is often associated with drug use or psychiatric problems," says Ruoho. "It's possible that new, highly selective drugs could be developed to inhibit the receptor and prevent this behavior."

The study revealed an additional neurologic link by confirming that the sigma-1 receptor and some compounds that bind to it inhibit ion channels, which are important for nerve activity. Work by many researchers — including some from UW-Madison — initially showed this relationship in earlier studies.

Some studies have also linked the receptor to the action of antidepressant drugs, and National Institutes of Health (NIH) scientists recently found that it appears to serve as a "chaperon," helping proteins to fold properly.

The Wisconsin researchers found that DMT is derived from the naturally occurring amino acid tryptophan and is structurally related to the neurotransmitter serotonin. This finding, Ruoho says, illustrates the mantra often used in the biological processing of natural molecules: Nothing goes to waste.

"Our findings support the idea that biochemical alterations of molecules such as tryptophan can produce simple compounds such as DMT that may target other regulatory pathways served by sigma-1 receptors," he says.

DMT may also reflect the presence of an even larger family of natural compounds that arise from other structurally related amino acids that may further regulate the receptor, Ruoho adds.

"It may well be that these different, naturally derived chemical forms regulate the sigma-1 receptor in tissue and organ-specific ways," he says.

Publication of flu vaccines studies in prestigious journals are determined by the sponsor

Research: Relation of study quality, concordance, take home message, funding, and impact in studies of influenza vaccines: Systematic review, BMJ Online

Industry-sponsored studies on influenza vaccines are published in journals with higher rankings (impact factors) and are cited more than studies with other sponsors, but this is not because they are bigger or better, finds a study published on bmj.com today.

Tom Jefferson and colleagues at the Cochrane Vaccine Field in Italy identified and assessed 274 studies on influenza vaccines and analysed their methodological quality, prestige of the host journals (impact factor) and citation rates in the scientific literature.

They found no relationship between study quality, publication in prestige journals or their subsequent citation in other articles. They also found that influenza vaccine studies are of poor quality and those with conclusions in favour of the vaccines are of significantly lower methodological quality.

The single most important determinant of where the studies were published or how much they were cited was sponsorship. Those partially or wholly funded by industry had higher visibility.

The researchers also found no relationship between journal impact factor and the quality of the influenza vaccine studies it publishes, suggesting that the impact factor is not the robust quality indicator that publishers suggest and confirming some of the widely expressed doubts on its appropriateness as a means of rewarding researchers with promotions and funds.

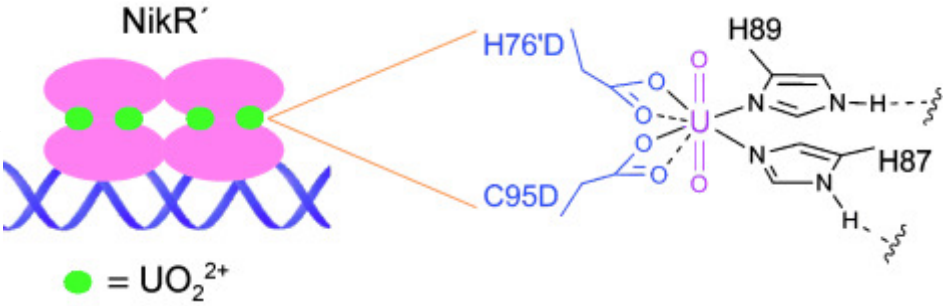
Dr Jefferson concludes: "The study shows that one of the levers for accessing prestige journals is the financial size of your sponsor. Pharmaceutical sponsors order many reprints of studies supporting their products, often with in-house translations into many languages. They will also purchase publicity space on the journal. Many publishers openly advertise these services on their website. It is time journals made a full disclosure of their sources of funding."

A Pocketful of Uranium

Construction of a selective uranium-binding protein

The use of uranium as a nuclear fuel and in weapons increases the risk that people may come into contact with it, and the storage of radioactive uranium waste poses an additional environmental risk. However, radioactivity is not the only problem related to contact with uranium; the toxicity of this metal is generally more dangerous to human health. Researchers are still looking for simple, effective methods for the sensitive detection and effective treatment of uranium poisoning. Researchers led by Chuan He at the University of Chicago and Argonne National Laboratory (USA) have now developed a protein that binds to uranium selectively and tightly. As reported in the journal Angewandte Chemie, it is based on a bacterial nickel-binding protein.

In oxygen-containing, aqueous environments, uranium normally exists in the form of the uranyl cation (UO22+), a linear molecule made of one uranium atom and two terminal oxygen atoms. The uranyl ion also likes to form coordination complexes. It prefers to surround itself with up to six ligands arranged in a plane around the ion's "equator". The research team thus chose to develop a protein that offers the uranyl ion a binding cavity in which it is surrounded by the protein's side-groups in the manner it prefers.



As a template, the scientists used the protein NikR (nickel-responsive repressor) from E. coli, a regulator that reacts to nickel ions. When NikR is loaded with nickel ions, it binds to a special DNA sequence. This represses transcription of the neighboring genes, which code for proteins involved in nickel uptake. If no nickel is present in the bacteria, NikR does not bind to the DNA.

The nickel ion is located in a binding cavity in which it is surrounded by a square-planar arrangement of binding groups. By using several mutation steps, the researchers generated a new protein that can bind uranium instead of nickel. Only three amino acids had to be changed. In the specially designed cavity, the uranyl group has six binding partners that surround it equatorially. In addition, there are spaces for the two terminal oxygen atoms of uranyl.

This NikR mutant only binds to DNA in the presence of uranyl, not in the presence of nickel or other metal ions. This confirms its selectivity for uranyl and may make it useful for the detection of uranyl and nuclear waste bioremediation. It also represents the first step towards developing potential protein- or peptide-based agents for treatment of uranium poisoning.

Chewing gum helps treat hyperphosphatemia in kidney disease patients

Treatment could reduce patients' risk of developing cardiovascular disease

Chewing gum made with a phosphate-binding ingredient can help treat high phosphate levels in dialysis patients with chronic kidney disease (CKD), according to a study appearing in the March 2009 issue of the Journal of the American Society Nephrology (JASN). The results suggest that this simple measure could maintain proper phosphate levels and help prevent cardiovascular disease in these patients.

Hyperphosphatemia (high levels of phosphate in the blood) commonly occurs in CKD patients on dialysis. Even when patients take medications to reduce phosphate acquired through their diet, about half of them cannot reduce phosphate to recommended levels.

Because patients with hyperphosphatemia also have high levels of phosphate in their saliva, researchers tested whether there might be a benefit to binding salivary phosphate during periods of fasting, in addition to using phosphate binders with meals. Vincenzo Savica, MD, of the University of Messina, and Lorenzo A. Calò MD, PhD, of the University of Padova, Italy and their colleagues recruited 13 dialysis patients with high blood phosphate levels to chew 20 mg of phosphate-binding chewing gum twice daily for two weeks between meals, in addition to their prescribed phosphate-binding regimen.

Dr. Savica and Dr. Calò's team found that salivary phosphate and blood phosphate levels significantly decreased during the first week of chewing, and by the end of two weeks, salivary phosphate decreased 55% and blood phosphate decreased 31% from levels measured at the start of the study. Salivary phosphate returned to its original level by day 15 after discontinuing the chewing gum, whereas blood phosphate took 30 days to return to its original value.

While these observations are preliminary and require confirmation in a randomized, double blind, placebo controlled study with more participants, the findings indicate that this chewing regimen might help control phosphate levels in patients with CKD. "Adding salivary phosphate binding to traditional phosphate binders could be a useful approach for improving treatment of hyperphosphatemia in hemodialysis patients," the authors concluded.

The study authors declare CM&D Pharma Limited, UK as a financial interest, for their supply of the experimental chewing gum.

The article, entitled "Salivary Phosphate-Binding Chewing Gum Reduces Hyperphosphatemia in Dialysis Patients," is currently online and will appear in the March 2009 print issue of JASN, doi 10.1681/ASN.2008020130.

More power from bumps in the road

MIT students develop energy-harvesting shock absorbers

David Chandler, MIT News Office February 9, 2009

A team of MIT undergraduate students has invented a shock absorber that harnesses energy from small bumps in the road, generating electricity while it smoothes the ride more effectively than conventional shocks. The students hope to initially find customers among companies that operate large fleets of heavy vehicles. They have already drawn interest from the U.S. military and several truck manufacturers.

Senior Shakeel Avadhany and his teammates say they can produce up to a 10 percent improvement in overall vehicle fuel efficiency by using the regenerative shock absorbers. The company that produces Humvees for the army, and is currently working on development of the next-generation version of the all-purpose vehicle, is interested enough to have loaned them a vehicle for testing purposes.

GenShock prototype **Photo courtesy / Zack Anderson**

The project came about because "we wanted to figure out where energy is being wasted in a vehicle," senior Zack Anderson explains. Some hybrid cars already do a good job of recovering the energy from braking, so the team looked elsewhere, and quickly homed in on the suspension.

They began by renting a variety of different car models, outfitting the suspension with sensors to determine the energy potential, and driving around with a laptop computer recording the sensor data. Their tests showed "a significant amount of energy" was being wasted in conventional suspension systems, Anderson says, "especially for heavy vehicles."

Once they realized the possibilities, the students set about building a prototype system to harness the wasted power. Their prototype shock absorbers use a hydraulic system that forces fluid through a turbine attached to a generator. The system is controlled by an active electronic system that optimizes the damping, providing a smoother ride than conventional shocks while generating electricity to recharge the batteries or operate electrical equipment.

In their testing so far, the students found that in a 6-shock heavy truck, each shock absorber could generate up to an average of 1 kW on a standard road -- enough power to completely displace the large alternator load in heavy trucks and military vehicles, and in some cases even run accessory devices such as hybrid trailer refrigeration units.

They filed for a patent last year and formed a company, called Levant Power Corp., to develop and commercialize the product. They are currently doing a series of tests with their converted Humvee to optimize the system's efficiency. They hope their technology will help give an edge to the military vehicle company in securing the expected $40 billion contract for the new army vehicle called the Joint Light Tactical Vehicle, or JLTV.

"They see it as something that's going to be a differentiator" in the quest for that lucrative contract, says Avadhany. He adds, "it is a completely new paradigm of damping." "This is a disruptive technology," Anderson says. "It's a game-changer." "Simply put -- we want this technology on every heavy-truck, military vehicle and consumer hybrid on the road," Avadhany says.

The team has received help from MIT's Venture Mentoring Service, and has been advised by Yet-Ming Chiang, the Kyocera Professor of Ceramics in the Department of Materials Science and Engineering and founder of A123 Systems, a supplier of high-power lithium-ion batteries.

Not only would improved fuel efficiency be a big plus for the army by requiring less stockpiling and transportation of fuel into the war zone, but the better ride produced by the actively controlled shock absorbers make for safer handling, the students say. "If it's a smoother ride, you can go over the terrain faster," says Anderson.

The new shocks also have a fail-safe feature: If the electronics fail for any reason, the system simply acts like a regular shock absorber.

The group, which also includes senior Zachary Jackowski and alumni Paul Abel '08, Ryan Bavetta '07 and Vladimir Tarasov '08, plans to have a final, fine-tuned version of the device ready this summer. Then they will start talking to potential big customers. For example, they have calculated that a company such as Wal-Mart could save $13 million a year in fuel costs by converting its fleet of trucks.

A version of this article appeared in MIT Tech Talk on February 11, 2009 (download PDF).

Ancient virus gave wasps power over caterpillar DNA

* 12:50 13 February 2009 **by Ewen Callaway**

A parasitic wasp lays its eggs into a caterpillar, at the same time delivering a hybrid virus **(Image: Alex Wild/myrmecos.net)**

A historical viral infection gave some insects genes that allow them to parasitise their caterpillar hosts, a new study finds.

Many species of wasps lay their eggs inside caterpillars. To make this possible, the wasps' have a secret weapon in the form of a dose of virus-like particles that are injected along with the eggs. Not only do these disable the caterpillars' immune system to stop it attacking the eggs, they also cause paralysis and keep the host from pupating - turning the caterpillar into an eternally youthful larder and nursery for the wasp grubs.

A closer look at these particles reveals that, although they look like viruses, they contain genetic material from the wasp, which is transcribed into the caterpillars' DNA - causing production of the very toxins that bring about their downfall.

Ancient infection

Essentially, the caterpillars produce their own poison, says Jean-Michel Drezen, a molecular biologist at the National Center for Scientific Research (CNRS) in Tours, France, who led the study.

Now Drezen's team says it has identified traces of a roughly 100-million-year-old viral infection that gave rise to the unique wasp-virus hybrids.

Scientists first identified the particles in the 1960s, yet didn't know what to make of them. They resemble other viruses under the gaze of an electron microscope, but their genomes don't match those of any other known virus.

Some scientists suggested that the wasps independently evolved the ability to produce the virus-mimicking particles, says James Whitfield, an entomologist at the University of Illinois in Urbana-Champaign, who had a hunch that true viruses were involved.

Wasp hijackers

"The spectre arose that the wasps were doing really clever genetic engineering that looks just like a virus but is really a wasp invention," he says.

The new study casts doubt on this possibility, though. Drezen's team identified up to 22 viral genes expressed in wasp ovaries, yet not housed in the virus particles. The sequences matched those of an obscure family of insect pathogens, called nudiviruses.

These genes produce proteins that form the coat of nudiviruses, as well as package DNA into virus particles. Viruses usually stuff their own DNA into these capsid shells, along with the occasional host stowaway. The wasps, it seems, have hijacked the entire system to shove its genes into the virus particles.

Drezen is still piecing together this evolutionary puzzle, but he thinks parasitic wasps were infected with a nudivirus roughly 100 million years ago.

'Lottery win'

Gradually, the wasp and virus coevolved to a mutually beneficial relationship: viruses save the wasps the trouble of producing their own toxins, while the viruses live on in the wasp genome. Viruses "won the lottery big time if you're thinking about their genes," says Whitfield.

The sinister nature of this practice won't surprise anyone familiar with parasitic wasps. Some turn their victims into mindless zombies, left only barely alive until the wasp eggs hatch. Yet another species keeps fly larvae alive long enough so it can survive the winter, thanks to an antifreeze compound made by the flies.

Charles Darwin even used one family of parasitic wasps as evidence for natural selection, writing to a colleague: "I cannot persuade myself that a beneficent and omnipotent God would have designedly created the Ichneumonidae with the express intention of their feeding within the living bodies of caterpillars."

Journal reference: Science (DOI: 10.1126/science.1166788)

Unrelated and mismatched cord blood transplantation can still help children with deadly conditions

DURHAM, N.C. -- An unrelated cord blood transplant, even from a mismatched donor, can be effective in treating children with a host of life-threatening diseases and disorders including cancer, sickle cell anemia, and other genetic diseases, according to researchers in the Duke Pediatric Blood and Marrow Transplantation Program. Unrelated cord blood may be easier to obtain than adult bone marrow, allowing for the treatment of more patients.

"We have done a terrific job in this country of increasing the number of volunteer donors listed in the National Marrow Donor Program registry over the past several years," said Vinod Prasad, M.D., a pediatric oncologist at Duke. "But the fact remains that for many patients, finding a matched donor can be difficult. Ethnic and racial minorities have the hardest time finding a fully matched donor."

The researchers will present their findings in an oral presentation at the American Society of Bone Marrow Transplantation in Tampa, Florida, on Friday, February 13, 2009. The study was partially funded by the National Institutes of Health.

"Our study found that using cord blood can be effective, without increased complications, and can provide more matches for patients, including ethnic minorities," said Prasad, who was the lead investigator. "Based on the findings of our study, we believe that unrelated cord blood transplant should be considered as an option for many of our young patients in need of a transplant."

Bone marrow transplantation has proven to be an effective treatment for thousands of children in the United States each year diagnosed with diseases such as leukemia and sickle cell disease, and inherited metabolic disorders such as Hurler's syndrome. Patients without a suitable match within their families can turn to the bone marrow registry, which currently lists more than seven million donors. Despite that number, however, many patients, particularly ethnic and racial minorities, are unable to find a completely matched donor.

The vast majority of these patients will find a partially matched donor within the public cord blood banks despite a smaller inventory of donor units, Prasad said.

"In order to match a donor to a recipient, doctors compare HLA typing, a test usually performed on a blood sample. In every individual, HLA typing includes the specific genetic make-up at three locations -- within those locations, you are looking at one set from the mother and one from the father, so it ends up to be six-point comparison," Prasad said. "In this analysis of children whose donor units were matched at four of six points, the transplant was successful in many patients, with low incidence of complications. Results were similar to those seen in patients receiving closer matched transplants. Thus the use of the 4/6 matched donors improved access to transplant for patients, especially those of ethnic and racial minorities."

The researchers studied data taken from 314 patients treated at Duke between 1993 and 2007. The patients ranged in age from six months to 21 years and suffered from both malignant and non-malignant conditions.

"We found that transplantation using 4/6 matched cord blood was effective and also carried a low probability of graft versus host disease, a complication caused by the attacking immune cells from the transplanted blood or marrow that perceive the recipient as 'foreign,' in the same way a healthy body's immune system might fight off a virus," Prasad said. "The incidence of other complications was low as well, and the data suggest that using 4/6 matched cord blood could improve access for all patients."

Duke has the largest cord blood transplantation program in the country, and the first unrelated cord blood transplant was performed by Duke physician Joanne Kurtzberg in 1993, on a patient with leukemia.

Other researchers involved in this study include Premjit Gill, Suhag Parikh, Paul Szabolcs, Timothy Driscoll, Kristin Page, Susan Wood, Deborah Semmel, Paul Martin, and Kurtzberg of Duke; and Adam Mendizabal and Shelly Carter, statisticians at the EMMES Corporation.

Cotton candy makes sweet blood vessel copies

* 15:13 13 February 2009 **by Colin Barras**

Cotton candy could provide a simple way to feed synthetic muscles or organs with essential gases and nutrients, say US medics. The tangled mass of sticky threads can be used as a template to grow artificial vascular networks inside engineered tissue. Using tissue engineering to grow replacement body parts in the lab, it has proved possible to fix damaged knees, windpipes, and even - in rabbits - penises.

But progress towards making more complex organs has been held back by the difficulty of mimicking the complex vascular networks that pervade real tissue. Without nets of capillaries that can be connected to the body's circulatory system, complex tissue can't receive the nutrients it needs, or dispose of waste effectively.

Sweet idea

Now Jason Spector of NewYork-Presbyterian Hospital and Leon Bellan of Cornell University have come up with a simple solution. It started when they noticed that cotton candy - also called candy floss - has a tangled structure not unlike a capillary network.

Bellan and colleagues placed some candy in a non-stick mould and poured over a polymer-resin mix that set hard after a day. They then dissolved away the sugar using water and alcohol to leave a solid cube shot through with a network of channels.

Using a scanning electron microscope showed that the channels were similar in dimensions to real networks of capillaries, at 1 to 100 micrometers wide with a few hundred micrometers between channels.

See a video of the cotton candy network (5 MB, .mpg format).

Flesh and blood

To show that blood could flow easily through the material, the researchers pumped rat blood with fluorescent labelling through the network.

See a video of fluorescent blood cells flowing through the artificial network (29 MB, .mpg format).

The researchers are now working on creating casts using a biodegradable resin mixed with cells of a particular tissue, and coating the cast's channels with blood vessel cells. As the cells grow, the biodegradable resin should gradually disappear to leave an artificial tissue sample with its own blood vessel network.

When such tissues are implanted into the body, the surgeon could plumb it into the body's own blood system, ensuring even large implants remain healthy. That paves the way for three-dimensional synthetic tissues, and ultimately could be a step towards artificial organs, Bellan told New Scientist.

Journal reference: Soft Matter (DOI: 10.1039/b819905a)

Why Chemical Warfare Is Ancient History

By Ishaan Tharoor Friday, Feb. 13, 2009

The prospect of chemical and biological warfare in this age of anthrax scares and WMD can feel — like the threat of nuclear Armageddon before it — like a uniquely modern terror. But a British archaeologist's recent find offers a reminder that chemical weapons are nothing new — in fact, they are nearly 2,000 years old. Simon James, a researcher at the University of Leicester in the U.K., claims to have found the first physical evidence of chemical weaponry, dating from a battle fought in A.D. 256 at an ancient Roman fortress. James concluded that 20 Roman soldiers unearthed beneath the town's ramparts did not die of war wounds, as previous archaeologists had assumed, but from poison gas.

The findings, announced in January at a meeting of the Archaeological Institute of America in Philadelphia, have caused a stir in archaeological circles, bringing to light proof of deeds usually encountered just in classical texts. Conducting a CSI-style cold-case forensic analysis of the site, James pieced together clues from records of earlier excavations at the Roman city of Dura-Europos, whose ruins are in modern Syria. An army of Persians had sacked the city and abandoned it, deporting its captive population deep into Persian territory. Dura-Europos became a ghost town, engulfed in sand until joint French-American teams dug it up in the 1930s.

During the final siege of the city, the attackers burrowed beneath the walls in order to breach the Roman defenses; the Romans heard this and started digging a countermine to fend off the assault. But the Persians, James told TIME, "prepared a nasty surprise," pumping lethal fumes from a brazier burning sulfur crystals and bitumen, a tarlike substance, with bellows into the Roman tunnels. The brazier was only doused, James suggests, "when the screaming stopped." Afterward, the Persians stacked the Roman corpses in a wall to prevent any reprisal, then lit the scene on fire.

War in antiquity rarely matched the heroism of its myths - it was ugly, nasty and desperate. To stave off a Roman siege in A.D. 189, the defenders of the Greek city of Ambracia built a complex flamethrower that

coughed out smoking chicken feathers. At Themiscrya, another stubborn Greek outpost, Romans tunneling beneath the city contended with not only a charge of wild beasts but also a barrage of hives swarming with bees - a rather direct approach to biological warfare.

The Romans themselves had few qualms about incorporating chemical warfare into their tactics. Roman armies routinely poisoned the wells of cities they were besieging, particularly when campaigning in western Asia. According to the historian Plutarch, the Roman general Sertorius in 80 B.C. had his troops pile mounds of gypsum powder by the hillside hideaways of Spanish rebels. When kicked up by a strong northerly wind, the dust became a severe irritant, smoking the insurgents out of their caves. The use of such special agents "was very tempting," says Adrienne Mayor, a classical folklorist and author of Greek Fire, Poison Arrows & Scorpion Bombs: Biological & Chemical Warfare in the Ancient World, "especially when you don't consider the enemy fully human."

Unconventional methods were used by both antiquity's weak and strong. In 332 B.C., the citizens of the doomed port of Tyre catapulted basins of burning sand at Alexander the Great's advancing army. Falling from the sky, the sand, says Mayor, "would have had the same ghastly effect as white phosphorus," the chemical agent allegedly used during Israel's recent bombardment of Gaza, not far to the south of ancient Tyre. A Chinese ruler in A.D. 178 put down a peasant revolt by encircling the rebels with chariots heaped with limestone powder. Accompanied by a cacophonous troupe of drummers, the charioteers pumped the powder into a primitive tear gas even more corrosive and lethal than its modern equivalent. The peasants didn't stand a chance.

Still, in the absence of the Geneva Conventions, ancient peoples did maintain "some sense of what it was to cross the line," says Mayor. Across cultures, it was customary to deplore trickery and extol the virtues of the noble warrior. The Brahmanic Laws of Manu, a code of Hindu principles first articulated in the fifth century B.C., forbade the use of arrows tipped with fire or poison. Written in India a century later, Kautilya's Arthashastra, one of the world's earliest treatises on war and realpolitik, advocates surprise night raids and offers recipes for plague-generating toxins, but it also urges princes to exercise restraint and win the hearts and minds of their foes. The Roman military historian Florus denounced a commander for sabotaging an enemy's water supply, saying the act "violated the laws of heaven and the practice of our forefathers."

Even in antiquity, many feared the lurking consequences of unleashing what we now call chemical weapons - indeed, the ancient Greek tale of Pandora's box offers a continuing metaphor for their use. And its moral proved true in the collapsed tunnels of Dura-Europos: among the Roman bodies, James spied one corpse set aside from the rest, which wore differing armor and carried a jade-hilted sword. This was a fallen Persian soldier, James concludes, also asphyxiated by the gas. The warrior who released the poison very likely succumbed to it.

Periodontitis and myocardial infarction: A shared genetic predisposition

This release is available in German.

A mutual epidemiological relationship between aggressive periodontitis and myocardial infarction has already been shown in the past. Scientists at the universities of Kiel, Dresden, Amsterdam and Bonn have now presented the first evidence of a shared genetic variant on chromosome 9, which maps to a genetic region that codes for the "antisense RNA" Anril, as reported in the latest edition of the specialist journal PLoS Genetics.

The first author, Dr Arne Schaefer from the Institute for Clinical Molecular Biology at Kiel University, sees clear similarities in the genetic predisposition: "We have examined the aggressive form of periodontitis, the most extreme form of periodontitis which is characterized by a very early age of onset. The genetic variation associated with this clinical picture is identical to that of patients who suffer from cardiovascular disease and have already had a myocardial infarction."

Because it has to be assumed that there is a causal connection between periodontitis and myocardial infarction, periodontitis should be taken seriously by dentists and diagnosed and treated at an early stage. "Aggressive periodontitis has shown itself to be associated not only with the same risk factors such as smoking, but it shares, at least in parts, the same genetic predisposition with an illness that is the leading cause of death worldwide.," warned Schaefer. Knowledge of the risk of heart attacks could also induce patients with periodontitis to keep the risk factors in check and take preventive measures.

Besides Arne Schaefer, Gesa Richter, who is doing a doctorate on the subject, is also part of Professor Stefan Schreiber's working group from the Institute for Clinical Molecular Biology at Schleswig Holstein University Hospital (UK S-H), Kiel Campus. As cardiologist, Dr Nour Eddine El Mokhtari from the Kiel Heart Centre is an important partner in the group. Dental expertise came from Dr Birte Größner-Schreiber from the Hospital for Conservative Dentistry and Periodontology at the UK S-H, Dr Barbara Noack, Technische Universität Dresden, as well as Professor Søren Jepsen from Bonn University and Professor Bruno Loos, Free University Amsterdam.

Cosmologist Paul Davies explores notion of 'alien' life on Earth

CHICAGO – Astrobiologists have often pondered "life as we do not know it" in the context of extraterrestrial life, says Paul Davies, an internationally acclaimed theoretical physicist and cosmologist at Arizona State University. "But," he asks, "has there been a blind spot to the possibility of 'alien' life on Earth?"

Davies will challenge the orthodox view that there is only one form of life in a lecture titled "Shadow Life: Life As We Don't Yet Know It" on Feb. 15 at the annual meeting of the American Association for the Advancement of Science. His presentation is part of the symposium "Weird Life."

"Life as we know it appears to have had a single common ancestor, yet, could life on Earth have started many times? Might it exist on Earth today in extreme environments and remain undetected because our techniques are customized to the biochemistry of known life?" asks Davies, who also is the director of the BEYOND Center for Fundamental Concepts in Science at Arizona State University in the College of Liberal Arts and Sciences.

In the lecture, Davies will present, challenge and extend some of the conclusions from a July 2007 report by the National Research Council. That report looked at whether the search for life should include "weird life" – described by the Council as "life with an alternative biochemistry to that of life on Earth."

"If a biochemically weird microorganism should be discovered, its status as evidence for a second genesis, as opposed to a new branch on our own tree of life, will depend on how fundamentally it differs from known life," wrote Davies in the Nov. 19, 2007, issue of Scientific American.

Davies and other pioneers who speculate that life on Earth may have started many times are wondering "why we have overlooked this idea for so long?"

The concept of a shadow biosphere, according to Davies, "is still just a theory. If someone discovers shadow life or weird life it will be the biggest sensation in biology since Darwin. We are simply saying, 'Why not let's take a look for it?' It doesn't cost much (compared to looking for weird life on Mars, say), and, it might be right under our noses."

Davies, whose research is steeped in the branches of physics that deal with quantum gravity – an attempt to reconcile theories of the very large and the very small – is a prolific author (27 books, both popular and specialty works) and is a provocative speaker (he delivered the 1995 Templeton Prize address after receiving the prestigious award for initiating "a new dialogue between science and religion that is having worldwide repercussions").

Among his books are: "How to Build a Time Machine," "The Origin of Life," "The Big Questions," "The Last Three Minutes," "The Mind of God," "The Cosmic Blueprint" and his most recent book "The Goldilocks Enigma: Why is the universe just right for life?" published in the United States under the title "Cosmic Jackpot."

He is putting the finishing touches on "The Eerie Silence," to be published in 2010 to coincide with the 50th anniversary of the SETI Institute. According to Davies, the book is "a comprehensive fresh look at the entire SETI enterprise."

Complex clues in a kiss

By James Morgan Science reporter, BBC News, Chicago

When you share a kiss with your lover on Valentine's Day, you may be revealing a lot more than you realise.

Locking lips not only stimulates our senses, it also gives us subtle clues about our suitability as mates, US scientists have found. A man's saliva has a "cocktail of chemicals" hinting at his fertility and evolutionary fitness, they said at a conference in Chicago. That may be why the first kiss is often the last - "the kiss of death".

Primitive instinct

"Kissing is a powerful adaptive mechanism - otherwise we wouldn't see it all over the world. Over 90% of human societies practice kissing," said anthropologist Helen Fisher, of Rutgers University in New Jersey, at the annual conference of the American Association for the Advancement of Science (AAAS) in Chicago.

"Chimpanzees and bonobos kiss. Foxes lick each other's faces. Birds tap their bills together and elephants put their trunks in one another's mouths. "So why do we do it? I think it is a tool for mate assessment. When you kiss, you can touch, see, feel, taste somebody. A huge part of our brain lights up.

"This is a real assessment tool - and can be highly positive or highly negative. In one study, 66% of women and 59% of men had experienced a first kiss which killed the relationship. It was the kiss of death."

Chemical bond

As well as acting as a "screening" mechanism for potential mates, Dr Fisher believes kissing evolved to stimulate what she has described as the three key brain systems for mating and reproduction.

The first of these is sex drive.

"Male saliva has testosterone in it. And men as a group seem to like wet kisses, with an open mouth and more tongue action. "So it may be that, unconsciously, they are attempting to transfer testosterone - to trigger the sex drive in women and push them into being more sexually receptive."

Men also have a poor sense of smell, she said, so by open mouth kissing "they might be trying to pick up traces of a woman's oestrogen cycle, to figure out the degree of her fertility."

The second mechanism is romantic love.

"Kissing is novel, at least at the beginning of a relationship, and novelty stimulates dopamine - which is associated with romantic love," said Dr Fisher.

Finally, kissing promotes what she referred to as "attachment" or "pair bonding". It helps us to stay together "at least long enough to have children," she said.

To study the chemistry which underlies kissing and pair bonding, neuroscientist Dr Wendy Hill, of Lafayette College, recruited a group of college students. The young lovers - 15 couples in all - were then split into two groups. Some were asked to smooch for 15 minutes, to the soundtrack of relaxing music. The others sat holding hands and talking.

Romantic setting?

"Afterwards, we measured the changes in their levels of cortisol - a stress hormone - in their saliva. "Levels had declined for everyone in the kissing group. And the longer the relationship, the lower the cortisol."

Dr Hill also took blood samples from the couples to measure levels of oxytocin - a messenger molecule associated with trust and sexual intimacy. After 15 minutes of kissing, the males saw a significant increase in the "pair bonding" chemical. But in the females, a decrease in oxytocin was observed.

"This was very surprising," Dr Hill admitted. "We are exploring the possibility that the setting - a college health centre - was just not very romantic. "After all, this is a place where students go when they are ill. That may have had an effect on the females."

Dr Fisher is now running the study again "in a more romantic setting. "We have a secluded room with a couch, flowers, candles, and a light jazz CD playing."

Interestingly, the females on birth control pills had significantly higher oxytocin levels, even before kissing began. But with so few couples taking part in the study, which has yet to be published, it was not clear if there was any direct link between the two.

Climate change likely to be more devastating than experts predicted, warns top IPCC scientist

Without decisive action, global warming in the 21st century is likely to accelerate at a much faster pace and cause more environmental damage than predicted, according to a leading member of the Nobel Prize-winning Intergovernmental Panel on Climate Change.

IPCC scientist Chris Field of Stanford University and the Carnegie Institution for Science points to recent studies showing that, in a business-as-usual world, higher temperatures could ignite tropical forests and melt the Arctic tundra, releasing billions of tons of greenhouse gas that could raise global temperatures even more - a vicious cycle that could spiral out of control by the end of the century.

"There is a real risk that human-caused climate change will accelerate the release of carbon dioxide from forest and tundra ecosystems, which have been storing a lot of carbon for thousands of years," said Field, a professor of biology and of environmental Earth system science at Stanford, and a senior fellow at Stanford's Woods Institute for the Environment. "We don't want to cross a critical threshold where this massive release of carbon starts to run on autopilot."

Field will present his findings Saturday, Feb. 14, at the annual meeting of the American Association for the Advancement of Science (AAAS) in Chicago during a symposium titled, "What Is New and Surprising Since the IPCC Fourth Assessment?"

Nobel Prize

Established by the United Nations in 1988, the IPCC brings together hundreds of experts from around the world to assess the science and policy implications of climate change. In 2007, the IPCC and Al Gore were awarded the Nobel Peace Prize. Field was among 25 IPCC scientists who attended the award ceremony in Oslo, Norway.

Since 1990, the IPCC has published four comprehensive assessment reports on human-induced climate change. Field was a coordinating lead author of the fourth assessment, Climate Change 2007, which concluded that the Earth's temperature is likely to increase 2 to 11.5 degrees Fahrenheit (1.1 to 6.4 degrees Celsius) by 2100, depending on how many tons of greenhouse gases are released into the atmosphere in coming decades.

But recent climate studies suggest that the fourth assessment report underestimated the potential severity of global warming over the next 100 years. "We now have data showing that from 2000 to 2007, greenhouse gas

emissions increased far more rapidly than we expected, primarily because developing countries, like China and India, saw a huge upsurge in electric power generation, almost all of it based on coal," Field said.

This trend is likely to continue, he added, if more developing countries turn to coal and other carbon-intensive fuels to meet their energy needs. "If we're going to continue re-carbonizing the energy system, we're going to have big CO2 emissions in the future," he said. "As a result, the impacts of climate change will probably be more serious and diverse than those described in the fourth assessment."

IPCC assessment reports are organized into three working groups. In September 2008, Field was elected co-chair of Working Group 2, which is charged with assessing the impacts of climate change on social, economic and natural systems. One of his major responsibilities is to oversee the writing and editing of the "Working Group 2 Report" for the IPCC fifth assessment, which will be published in 2014.

"In the fourth assessment, we looked at a very conservative range of climate outcomes," Field said. "The fifth assessment should include futures with a lot more warming."

Forest-carbon feedback

Of particular concern is the impact of global warming on the tropics. "Tropical forests are essentially inflammable," Field said. "You couldn't get a fire to burn there if you tried. But if they dry out just a little bit, the result can be very large and destructive wildfires."

According to several recent climate models, loss of tropical forests to wildfires, deforestation and other causes could increase atmospheric CO2 concentrations from 10 to 100 parts per million by the end of the century. This would be a significant increase, given that the total concentration of CO2 in the atmosphere is currently about 380 parts per million, the highest in 650,000 years.

"It is increasingly clear that as you produce a warmer world, lots of forested areas that had been acting as carbon sinks could be converted to carbon sources," Field said. "Essentially we could see a forest-carbon feedback that acts like a foot on the accelerator pedal for atmospheric CO2. We don't exactly know how strong the feedback could be, but it's pretty clear that the warmer it gets, the more likely it is that degradation of tropical forests will increase the atmospheric CO2."

The ocean is another vital reservoir for carbon storage. Recent studies show that global warming has altered wind patterns in the Southern Ocean, which in turn has reduced the ocean's capacity to soak up excess atmospheric CO2. "As the Earth warms, it generates faster winds over the oceans surrounding Antarctica," Field explained. "These winds essentially blow the surface water out of the way, allowing water with higher concentrations of CO2 to rise to the surface. This higher-CO2 water is closer to CO2-saturated, so it takes up less carbon dioxide from the atmosphere."

Tundra thawing

Climate scientists also worry that permafrost in the Arctic tundra will thaw, releasing enormous amounts of CO2 and methane gas into the atmosphere. According to Field, the most critical, short-term concern is the release of CO2 from decaying organic matter that has been frozen for millennia. "The new estimate of the total amount of carbon that's frozen in permafrost soils is on the order of 1,000 billion tons," he said. "By comparison, the total amount of CO2 that's been released in fossil fuel combustion since the beginning of the Industrial Revolution is around 350 billion tons. So the amount of carbon that's stored in these frozen soils is truly vast."

Much of the carbon is locked up in frozen plants that were buried under very cold conditions and have remained in deep freeze for 25,000 to 50,000 years, he added. "We know that the Arctic is warming faster than anyplace else," he said. "And there is clear evidence that these frozen plants are very susceptible to decomposition when the tundra thaws. So melting of permafrost is poised to be an even stronger foot on the accelerator pedal of atmospheric CO2, with every increment of warming causing an increment of permafrost-melting that shoots an increment of CO2 into the atmosphere, which in turn increases warming.

"There's a vicious-cycle component to both the tundra-thawing and the tropical forest feedbacks, but the IPCC fourth assessment didn't consider either of them in detail. That's basically because they weren't well understood at the time."

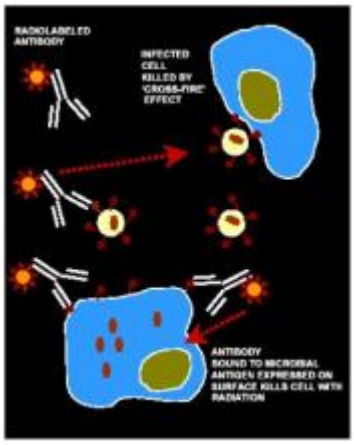
For the fifth assessment report, Field said that he and his IPCC colleagues will have access to new research that will allow them to do a better job of assessing the full range of possible climate outcomes. "What have we learned since the fourth assessment? We now know that, without effective action, climate change is going to be larger and more difficult to deal with than we thought. If you look at the set of things that we can do as a society, taking aggressive action on climate seems like one that has the best possibility of a win-win. It can stimulate the economy, allow us to address critical environmental problems, and insure that we leave a sustainable world for our children and grandchildren. Somehow we have to find a way to kick the process into high gear. We really have very little time."

Radioimmunotherapy: Promising treatment for HIV infection and viral cancers

Novel therapy presented in featured lecture at AAAS Annual Meeting

BRONX, NY - Scientists at Albert Einstein College of Medicine of Yeshiva University have piggybacked antibodies onto radioactive payloads to deliver doses of radiation that selectively target and destroy microbial and HIV-infected cells. The experimental treatment — called radioimmunotherapy, or RIT — holds promise for treating various infectious diseases, including HIV and cancers caused by viruses. The research was presented today at the annual meeting of the American Association for the Advancement of Science (AAAS), the world's largest general scientific society and the publishers of the journal Science.

The talk, part of the AAAS Topical Lecture Series, was delivered by Ekaterina Dadachova, Ph.D., a leading RIT researcher at Einstein. Dr. Dadachova is the Olnick Faculty Scholar in Cancer Research, as well as an associate professor of nuclear medicine and of microbiology & immunology at the College of Medicine.



Radiolabeled antibodies kill HIV infected human cells through binding to viral antigens on these cells. **Albert Einstein College of Medicine**

RIT, which is currently used in cancer treatment, capitalizes on the fact that each type of antibody is programmed to seek out just one type of antigen in the body. Thus, by attaching radioactive material to a particular antibody, radiation can be targeted at specific cells that express the corresponding antigen, minimizing collateral damage to other tissues. This level of specificity is not possible with existing forms of radiation therapy.

RIT was originally developed as a therapy for cancer treatment and has been the most successful so far in treatment of non-Hodgkin lymphoma, a cancer that originates in cells of the immune system. Over the last few years, in collaboration with Dr. Arturo Casadevall, Chair and Forchheimer Professor of Microbiology & Immunology at Einstein, Dr. Dadachova has adapted the technique for fighting fungal, bacterial, and viral infections. Drs. Dadachova and Casadevall performed these studies in conjunction with scientists at Einstein and NYU, and at the European Commission Joint Research Centre; the latter of which supplied some of the important radionuclides for arming the antibodies.

Since viruses are quite different from cancer cells, devising radioimmunotherapy for HIV posed significant challenges. Viruses are tiny bits of DNA or RNA wrapped in a thin protein coat. Simple, tough, and resilient, viruses easily shrug off radiation directed at them and can readily repair any damage that might occur. Complicating matters, HIV can hide in immune cells keeping the virus beyond the reach of antibodies.

"Our approach is not to target the virus particles themselves, but rather lymphocytes that harbor the virus," says Dr. Dadachova. "Fortunately, lymphocytes are among the most radiosensitive cells in the body."

The RIT devised by Einstein researchers consists of an antibody for glycoprotein 41 (gp41) and a radioactive isotope called Bismuth-213, bound together with a special molecule known as a ligand. The gp41 antibody was selected because its corresponding gp41 antigen is reliably expressed on the surface of cells infected with HIV. In addition, unlike other HIV-related glycoproteins, gp41 antigen usually is not shed into the bloodstream, which would lead many of radioactive-labeled antibodies to miss their target. Bismuth-213 was chosen because of several characteristics, including a half-life, or decay rate, of 46 minutes. Such a short half-life rate allows just enough time for the treatment to be administered and for the radioactive antibodies to do their job. After four hours, Bismuth-213 radioactivity falls to negligible levels.

Drs. Dadachova and Casadevall and their colleagues have demonstrated that the treatment can effectively eliminate HIV-infected human cells in both laboratory and animal studies, the latter involving two different models of mice with HIV. The team is now conducting pre-clinical testing of the therapy's efficacy and safety in preparation for a Phase I clinical trial in HIV-infected patients.

RIT also has potential as a therapy for cancers that are preceded by viral infections, such as cervical cancer (certain forms of which are associated with human papilloma virus) and hepatocellular carcinoma (associated with hepatitis B virus). Such cancers account for almost a quarter of all cancers. "Many virus-associated cancer cells continue to express viral antigens," Dr. Dadachova explains. "As these antigens are not found anywhere else in the body, RIT of viral cancers promises exquisite specificity of treatment and very low toxicity to the patient."

Relevant papers by Dr. Dadachova:

Dadachova E, Patel MC, Toussi S, Apostolidis C, Morgenstern A, Brechbiel MW, Gorny MK, Zolla-Pazner S, Casadevall A, Goldstein H.. (2006) Targeted killing of virally infected cells by radiolabeled antibodies to viral proteins. PLoS Med 3(11): e427.

X-G. Wang, E. Revskaya, R A. Bryan, H.D. Strickler, R. D. Burk, A. Casadevall, E. Dadachova (2007) Treating cancer as an infectious disease - viral antigens as novel targets for treatment and potential prevention of tumors of viral etiology. PLOS One 2(10): e1114.
Dadachova E, Casadevall A.(2009) Radioimmunotherapy of infectious diseases. Semin Nucl Med. 39(2):146-53.

Galaxy has 'billions of Earths'

There could be one hundred billion Earth-like planets in our galaxy, a US conference has heard.

Dr Alan Boss of the Carnegie Institution of Science said many of these worlds could be inhabited by simple lifeforms.

He was speaking at the annual meeting of the American Association for the Advancement of Science in Chicago.

So far, telescopes have been able to detect just over 300 planets outside our Solar System.

Very few of these would be capable of supporting life, however. Most are gas giants like our Jupiter; and many orbit so close to their parent stars that any microbes would have to survive roasting temperatures.

But, based on the limited numbers of planets found so far, Dr Boss has estimated that each Sun-like star has on average one "Earth-like" planet.

The number of stars points to there being many rocky planets

This simple calculation means there would be huge numbers capable of supporting life.

"Not only are they probably habitable but they probably are also going to be inhabited," Dr Boss told BBC News. "But I think that most likely the nearby 'Earths' are going to be inhabited with things which are perhaps more common to what Earth was like three or four billion years ago." That means bacterial lifeforms.

Dr Boss estimates that Nasa's Kepler mission, due for launch in March, should begin finding some of these Earth-like planets within the next few years.

Recent work at Edinburgh University tried to quantify how many intelligent civilisations might be out there. The research suggested there could be thousands of them.

Study finds behavioral link between insomnia and tension-type headaches

Findings of this study suggest that efforts to manage tension-type headache pain by going to sleep might serve as a behavioral risk factor for developing insomnia

Westchester, Ill. –A study in the Feb. 15 issue of the Journal of Clinical Sleep Medicine shows that the use of sleep or napping to relieve chronic pain caused by tension-type headaches could have the unwanted effect of decreasing the homeostatic drive for sleep, leading to reduced ability to initiate and maintain sleep at night. Use of sleep as a coping mechanism for pain over time could lead to the development of poor sleep hygiene and serve as a perpetuating factor for chronic insomnia.

Group comparisons on triggers of headache indicate that a significantly greater proportion of the headache group relative to the control group (58 versus 18 percent) reported sleep problems as a trigger of headaches, and women in the headache group reported a significantly higher rating of pain interfering with sleep. Eighty-one percent of women who suffer from tension-type headaches reported going to sleep as a way of managing their headaches; this method was also rated as the most effective self-management strategy for pain.

Principal investigator and lead author, Jason C. Ong, PhD, assistant professor of behavioral sciences at Rush University Medical Center, said the extent to which headache sufferers rated sleep as being an effective method for coping with pain was somewhat surprising

"Insomnia is a common complaint among headache sufferers. While napping may relieve pain, it may also result in poor sleep hygiene, thus triggering sleep disturbance or perpetuating an insomnia episode," said Ong.

A high proportion of both the headache and control groups (97 and 70 percent) reported stress as a trigger of headaches. No significant differences were found between the groups on use of medication to relieve headaches.

A total of 65 women were recruited from undergraduate psychology courses at a university located in the southeastern U.S.; 32 participants who were confirmed to have tension-type headaches, as classified by the International Headache Society System, were placed in a headache group, and 33 were classified as controls who experience minimal pain. The average age of members of the headache group was 21.9 years, while the average age of the control group was 18.9 years.

The average time since the first headache of any type was 9.4 years for participants in the headache group, with an average of 8.11 headache days per month. Participants reported an average of 12.2 tension-type headaches over the past year, and 2.1 tension-type headaches in the past month, with a median duration of 2.0 hours. The average tension-type headache intensity rating using a 0-to-10 scale was 5.6. Six participants in the headache group also met criteria for migraine disorder.

Secondary analyses were conducted on self-report data from participants who completed a psychophysiologic assessment investigating the pattern of physiologic, affective and behavioral responses to a picture-viewing task. All participants completed a research questionnaire packet that included measures pertaining to pain history and pain coping strategies. Electromyographic (EMG) activity, self-reported affect and self-reported oral motor behaviors were also analyzed.

The authors conclude that the assessment of daytime napping behaviors among individuals who report insomnia and headaches may be important for developing behavioral sleep interventions. They also note that clinicians should be aware of the challenges of managing pain without causing sleep disturbances.

A media fact sheet about insomnia is available from the AASM at http://www.aasmnet.org/Resources/FactSheets/Insomnia.pdf, and information for patients and the public is available at http://www.sleepeducation.com/Disorder.aspx?id=42.

Researchers shed new light on connection between brain and loneliness

Work is part of emerging field examining brain mechanisms

Social isolation affects how people behave as well as how their brains operate, a study at the University of Chicago shows.

The research, presented Sunday at a symposium, "Social Emotion and the Brain," at the annual meeting of the American Association for the Advancement of Science, is the first to use fMRI scans to study the connections between perceived social isolation (or loneliness) and activity in the brain. Combining fMRI scans with data relevant to social behavior is part of an emerging field examining brain mechanisms—an approach to psychology being pioneered at the University of Chicago.

Researchers found that the ventral striatum—a region of the brain associated with rewards—is much more activated in non-lonely people than in the lonely when they view pictures of people in pleasant settings. In contrast, the temporoparietal junction—a region associated with taking the perspective of another person—is much less activated among lonely than in the non-lonely when viewing pictures of people in unpleasant settings.

"Given their feelings of social isolation, lonely individuals may be left to find relative comfort in nonsocial rewards," said John Cacioppo, the Tiffany and Margaret Blake Professor in Psychology at the University. He spoke at the briefing along with Jean Decety, the Irving B. Harris Professor in Psychology and Psychiatry at the University.

The ventral striatum, which is critical to learning, is a key portion of the brain and is activated through primary rewards such as food and secondary rewards such as money. Social rewards and feelings of love also may activate the region.

Cacioppo, one of the nation's leading scholars on loneliness, has shown that loneliness undermines health and can be as detrimental as smoking. About one in five Americans experience loneliness, he said. Decety is one of the nation's leading researchers to use fMRI scans to explore empathy.

They were among five co-authors of a paper, "In the Eye of the Beholder: Individual Differences in Perceived Social Isolation Predict Regional Brain Activation to Social Stimuli," published in the current issue of the Journal of Cognitive Neuroscience.

In the study, 23 female undergraduates were tested to determine their level of loneliness. While in an fMRI scanner, the subjects were shown unpleasant pictures and human conflict as well as pleasant things such as money and happy people.

The subjects who rated as lonely were least likely to have strong activity in their ventral striata when shown pictures of people enjoying themselves.

Although loneliness may be influence brain activity, the research also suggests that activity in the ventral striatum may prompt feelings of loneliness, Decety said. "The study raises the intriguing possibility that loneliness may result from reduced reward-related activity in the ventral striatum in response to social rewards."

In addition to differing responses in the ventral striatum, the subjects also recorded differing responses in parts of the brain that indicated loneliness played a role in how their brain operates.

Joining Decety and Cacioppo in writing the Journal of Cognitive Science paper were Catherine Norris, Assistant Professor of Psychology at Dartmouth College; George Monteleone, a graduate student at the University of Chicago; and Howard Nusbaum, Chair of Psychology at the University of Chicago.

Decety and Cacioppo discussed the new field of brain mechanism in a paper in the current issue of Perspectives on Psychological Science, "What Are the Brain Mechanisms on Which Psychological Processes are Based?" The new field extends the work of Charles Darwin, who "regarded the brain as a product of evolution and the science of psychology as concerned with these foundations," they wrote.

By studying brain mechanisms, researchers hope to gain new insights by examining mental activities surrounding consciousness, perception and thought through an understanding of how columns of neurons stacked next to each other form elementary circuits to function as a unit, they wrote.

New visualization tools such as three-dimensional imaging will help scholars develop a new way of studying psychology, they said.

"Psychological science in the 21st century can, and should, become not only the science of overt behavior, and not only the science of the mind, but also the science of the brain," they concluded.

Ice oceans 'are not poles apart'

By Mark Kinver Science and environment reporter, BBC News

At least 235 marine species are living in both polar regions, despite being 12,000km apart, a census has found.

Limacina helicina occurs in both Arctic and Antarctic waters

Scientists were surprised to find the same species of "swimming snails" at both poles, raising questions about how they evolved and became so dispersed.

The census, involving 500 researchers from more than 25 nations, was carried out during International Polar Year.

The findings form part of the global Census of Marine Life (CoML) report, which will be published in 2010.

"Some of the more obvious species like birds and whales migrate between the poles on an annual basis," explained Ron O'Dor, CoML's co-senior scientist.

But he added the presence of smaller creatures, such as worms living in mud, sea cucumbers and "swimming snails", at both locations had particularly interested researchers on the project.

'Conveyor belt'

One of the swimming snails, or sea butterflies, found in the icy waters of both the Arctic and Antarctic was Cliona limacina .

The creature feeds on Limacina helicina , which is another swimming snail found in the waters of both poles.

Dr O'Dor said that although there was 12,000km separating the two habitats, it did not create a huge barrier for marine wildlife, as a mountain range does for terrestrial species.

"The oceans are a mixing ground," he told BBC News. "There are all kinds of currents that allow things to move around."

He also added that the temperature differences in the oceans did not vary enough to act as a thermal barrier.

"The deep ocean at the poles falls as low as -1C (30F), but the deep ocean at the equator might not get above 4C (39F).

"There is continuity in the ocean as a result of the major current systems, which we call the 'conveyor belt'; a lot of these animals have egg and larvae stages that can get transferred in this water."

'Barcode of life'

Dr O'Dor said that part of the CoML's work included examining organisms' genetic information, which would help the scientists to identify any differences between the seemingly identical species.

"The traditional approach was to describe an organism's physical features, so if these organisms lived in very similar habitats, did very similar jobs and ate similar food, then they often looked very alike even if they came from different origins.

"So we are also working very closely with the Barcode of Life team at the University of Guelph (Canada), and we hope that by 2010 that we will have about 90% of marine species barcoded."

The project aims to develop DNA barcoding as a global standard for identifying species using key genetic markers - much like a shop barcode uniquely identifies a retail product.

"It's a new way to mark or classify things," Dr O'Dor observed.

"Even though organisms look exactly the same and have been identified as being the same type by traditional methods, genetic information can reveal them to be a sub-species or different populations."

COML, which began back in 2000, carried out 17 regional censuses involving more than 2,000 scientists from 82 nations.

Currently, the census teams are collating and examining the data collected by the various surveys, ahead of the publication in October 2010 of the first global Census of Marine Life.

Science intends to tag all life

By Jonathan Amos BBC News science reporter

Scientists are to establish a giant catalogue of life - to, in effect, "barcode" every species on Earth, from tiny plankton to the mighty blue whale.

Science is engaged in a huge stocktaking exercise

Initial projects will focus on birds and fish, recording details in their genetic make-up that can be used to tell one life form from another. The initiative was launched in London at the International Conference for the Barcoding of Life. Researchers concede it will take many years to complete the task.

"About 1.7 million species are known - we suspect there are anything from 10-30 million species on Earth," explained Dr Richard Lane, director of science at London's Natural History Museum. "We have discovered that it is quite possible to have a short DNA sequence that can characterise just about every form of life on the planet."

Opening up

At the cost of about £1 ($1.80) per genetic test, many specimens for each species will now be analysed to obtain their barcode information. This data will then be put into a giant database which the Consortium for the Barcode of Life (CBOL) hopes can be used to link off to all the knowledge acquired by science on particular organisms.

And just as one might Google a species name today to find pictures or a description of an individual insect, the time may come when we have Star Trek-style mobile computers that can read off barcodes and access species information in the field.

"It's like a policeman who sees a car breaking the law and all he's got is the license plate number - but with that number he's got the owner and when the car was bought," Professor Dan Janzen, from the University of Pennsylvania, US, said. "And that's what a barcode is - it's that thing that links you to the body of information the taxonomists, the natural historians and ecologists have been accumulating for 200 years."

The consortium pulls together a range of world museums, zoos, research organisations and companies concerned with taxonomy and biodiversity.

DNA barcodes should make species recognition in the field much easier, especially where traditional methods are not practical. In addition, species identification should become more reliable, particularly for non experts.

Just knowing every species on Earth would help answer some fundamental questions in ecology and evolution. And with this information we would get very much better polices to manage and conserve the world around us.

Short code

Dr Scott Miller, the chairman of the CBOL, added: "DNA barcoding will make a huge difference to our knowledge and understanding of the natural world. "The Barcode of Life initiative aims to complement existing taxonomic practice to build on it and expand its power and use."

The segment of DNA to be used in the project is found in a gene known as cytochrome c oxidase I, or COI.

This is involved in the metabolism of all creatures but is coded slightly differently in each one of them.

In humans, for example, COI barcodes differ from one another by only one or two DNA "letters" out of 648; while we differ from chimpanzees at about 60 locations, and in gorillas by about 70.

DNA - BARCODING LIFE

The double-stranded DNA molecule is held together by four chemical components, or bases

Adenine (A) bonds with thymine (T); cytosine(C bonds with guanine (G)

Written in these "letters" are genes - starting template that drive the production of proteins which build and maintain an organism's body

Barcoding records the order of DNA letters in a particular gene found in mitochondria, the "power units" in cells

This DNA changes rapidly over time as species evolve

These differences can be used to distinguish species

Wednesday saw the announcement at the London conference of a project to get comprehensive barcode data on all known fish types - currently thought to number 15,000 marine and 8,000 freshwater species.

The bird project will list the world's 10,000 known avian species.

A third project will genetically label the 8,000 kinds of plants in Costa Rica, Central America.

Some doubts

It has to be said that not all of the science community shares the unrestrained enthusiasm of the barcoders.

Some researchers are concerned that taxonomic skills that have traditionally been used to catalogue individual species may suffer - fewer and fewer students now take up the discipline, especially in Western

universities. Doubt has also been expressed that the COI approach will prove as reliable in distinguishing species as is claimed.

This view is shared by butterfly expert James Mallet, from University College London. Although he supports the project, Professor Mallet thinks it may struggle in several settings, such as when a new or hybrid species has just emerged in a population. "I just wish it hadn't been called barcoding because this should mean things are identical - for any retail product that's how you recognise them," he said. "This is not true of mitochondrial DNA and in an evolutionary setting where species grade into other species, this is a lot more tricky.

"Closely related species are going to give you more difficulty and the more data you have, including DNA data - and DNA data is very powerful - the better."

First gravity map of Moon's far side unveiled

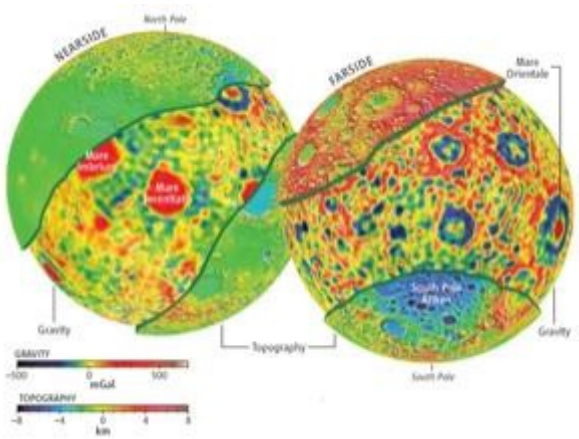
* 00:00 16 February 2009 **by Rachel Courtland**

The first detailed map of the gravity fields on the Moon's far side shows that craters there are different than those on the near side. The results could reveal more about the Moon as it was billions of years ago, when magma flowed across its surface.

The new gravity map was collected by the Japanese lunar satellite Kaguya, which released two small probes into orbit around the Moon in 2007. The motions of the three spacecraft, which are sensitive to variations in the Moon's gravity field, were measured by tracking their radio signals.

Crucially, while the main Kaguya spacecraft was on the far side of the Moon and therefore out of direct contact with Earth, one of the small probes relayed its signals to Earth.

The resulting map - the first detailed one completed of the Moon's far side - shows that craters on the far side have a markedly different gravity signature from those on the side that always faces Earth.



The Japanese probe Kaguya has created the first map of gravity differences on the far side of the Moon, which always points away from Earth. The gravity signatures of some craters suggests the far side might have been stiffer and cooler than expected **(Illustration: Namiki et al/AAAS)**

'Fabulous data'

That suggests that billions of years ago, there might have been large differences in the temperature or thickness of the Moon's two halves.

"It's fabulous new data," says Walter Kiefer, a planetary geophysicist with the Lunar and Planetary Institute in Houston, Texas, who was not part of the study. "We haven't been able to get a good look at the far side until now."

Most of the large craters on the Moon formed more than 3.8 billion years ago. These were partly filled in by magma that flowed on the surface before the Moon cooled and its geological activity died down.

But a number of craters also seem to have been filled in from below. Researchers believe material from the mantle also rose up in craters, since these are sites where impacts had thinned the Moon's crust.

The new Kaguya measurements reveal some craters on the far side that seem to have been filled only with mantle. These craters have higher-than-normal gravity at the centre, surrounded by a thick ring of low gravity that closely matches the original low elevation of the crater.

Opposite conclusions

It is not yet clear what these new crater measurements suggest about the early Moon. In order for these structures to survive, the lunar far side must have been too cool and stiff to allow the mantle at the craters' centres to smooth out much over time, says team leader Noriyuki Namiki, of Japan's Kyushu University. "The surface had to be very rigid to support these structures," Namiki says.

But Keifer says the low gravity rings could argue for the opposite scenario. The structures in the centres of the craters might be narrow because the top layer of the Moon's far side was too thin and warm to be able to hold up anything larger. Comparing the Kaguya observations with models could help settle the question, Kiefer says.

The Moon's two halves also show other striking differences. NASA's Lunar Prospector, which operated in the late 1990s, found that radioactive elements seem to be concentrated on the near side. The far side also shows less evidence of past volcanic activity. *Journal reference: Science (vol 323, p 900)*