Dinosaurs burrowed to avoid winter's chill

* 11:44 13 July 2009 **by Jeff Hecht**

Polar winters weren't as cold in the Cretaceous as they are today, but they were long and dark. That has posed a puzzle: how did small dinosaurs living in polar areas survive the lean months when little food was available? The discovery of three fossil burrows in south-eastern Australia suggests they may have dug in for the winter.

Fossils from "Dinosaur Cove" in Victoria show that small plant-eating dinosaurs called hypsilophodontids were common in the area about 110 million years ago, a time when the region was within the Antarctic Circle. The region was forested, with temperatures 6 or 7 °C warmer than such latitudes are today. But with the sun below the horizon for weeks or months of the winter, fresh vegetation would have been lacking.

Plant-eating dinosaurs had been thought to avoid the dark season by migrating long distances. The evidence for this was based on fossils from another related species, Edmontosaurus, which have been found from northern Alaska to Montana.

The Oryctodromeus cubicularis burrows down into it's hole. **(Image: James Hays, Fernbank Museum of Natural History)**

'Corkscrew' burrows

However, last year, Eric Snively of the University of Alberta in Edmonton, Canada, showed that the fossils could have been from a number of species, or that the dinosaurs may have lived at different times. "We don't think the evidence for migration is there," he told New Scientist. In this case, geography would have made north-south migration difficult or impossible in Australia and New Zealand, he adds.

Burrowing would have given small dinosaurs a refuge for the winter where they could rest and conserve energy, but evidence of this had been lacking. Then, two years ago, David Varricchio of Montana State University in Bozeman described a fossil burrow in Montana that contained the remains of one adult and two juvenile hypsilophodontids similar to those found in Australia.

Tony Martin of Emory University in Atlanta, Georgia, says that working with Varricchio on the Montana burrow primed him to spot the new burrows when he went to Australia looking for dinosaur tracks. "I turned around a corner at the outcrop, and I saw a structure identical to the one I saw in Montana," he says (see image). Further investigation uncovered a total of three burrows in the area, each very similar to those in Montana.

Flood risk

Although he didn't excavate the burrows or find any fossils, cross-sectional measurements of a corkscrew-shaped burrow 2.1 metres long suggest that it was dug by dinosaurs weighing 10 to 20 kilograms – the same size as the Montana burrow-diggers.

Martin says the burrows were dug in soil deposited in a valley by massive spring floods of meltwater similar to those seen today on the north slopes of Alaska. The dinosaurs could have lived in the burrows in autumn and winter, but would have had to move out before the following year's spring floods filled them with fresh sediment.

"His interpretation seems quite plausible," says palaeontologist Tom Rich of Museum Victoria in Melbourne, who visited the burrows with Martin. He hopes Martin will return to Australia and search for more burrows along the Victoria coast, which Rich would then excavate to hunt for fossil bones that might identify the occupants. *Journal reference: Cretaceous Research (DOI: 10.1016/j.cretres.2009.06.003)*

Hungry cats trick owners with baby cry mimicry

* 17:00 13 July 2009 **by Nic Fleming**

Cat owners will know the feeling. Your pet is purring loudly, demanding to be fed, and isn't going to give up until it gets what it wants. What most doting owners won't realise is that the cat is using an acoustic ruse.

According to Karen McComb of the University of Sussex, UK, domestic cats hide a plaintive cry within their purrs that both irritates owners and appeals to their nurturing instincts.

The team recorded the purrs of 10 different cats when they were soliciting food, and when they were purring in a different context. Fifty people who were asked to rate the purrs on how pleasant and urgent they sounded consistently rated the "solicitation purrs" as more urgent and less pleasant. Cat owners were especially good at distinguishing between the two kinds of purring.

Hear recordings of the cries here ***(urgent)*** and here ***(non-urgent)***.

When the team examined the sound spectrum of the solicitation purrs they saw an unusual peak in the 220 to 520-hertz frequency range embedded in the much lower frequencies of the usual purr. Babies' cries have a similar frequency range, 300 to 600 hertz, McComb says.

The louder this high-frequency element, the more urgent and less pleasant the purr was rated. Cats may be exploiting "innate tendencies in humans to respond to cry-like sounds in the context of nurturing offspring", McComb says. *Journal reference: Current Biology (DOI: 10.1016/j.cub.2009.05.033)*

How noise and nervous system get in way of reading skills

EVANSTON, Ill. --- A child's brain has to work overtime in a noisy classroom to do its typical but very important job of distinguishing sounds whose subtle differences are key to success with language and reading.

But that simply is too much to ask of the nervous system of a subset of poor readers whose hearing is fine, but whose brains have trouble differentiating the "ba," "da" and "ga" sounds in a noisy environment, according to a new Northwestern University study.

"The 'b,' 'd' and 'g' consonants have rapidly changing acoustic information that the nervous system has to resolve to eventually match up sounds with letters on the page," said Nina Kraus, Hugh Knowles Professor of Communication Sciences and Neurobiology and director of Northwestern's Auditory Neuroscience Laboratory, where the work was performed.

In other words, the brain's unconscious faulty interpretation of sounds makes a big difference in how words ultimately will be read. "What your ear hears and what your brain interprets are not the same thing," Kraus stressed.

The Northwestern study is the first to demonstrate an unambiguous relationship between reading ability and neural encoding of speech sounds that previous work has shown present phonological challenges for poor readers.

The research offers an unparalleled look at how noise affects the nervous system's transcription of three little sounds that mean so much to literacy.

The online version of the study will be published by the Proceedings of the National Academy of Sciences (PNAS) (http://www.pnas.org/papbyrecent.shtml) soon and is embargoed until 5 p.m. EDT Monday July 13.

The new Northwestern study as well as much of the research that comes out of the Kraus lab focuses on what is happening in the brainstem, an evolutionarily ancient part of the brain that scientists in the not too distant past believed simply relayed sensory information from the ear to the cortex.

As such, much of the earlier research relating brain transcription errors to poor reading has focused on the cortex -- associated with high-level functions and cognitive processing.

Focusing earlier in the sensory system, the study demonstrates that the technology developed during the last decade in the Kraus lab now offers a neural metric that is sensitive enough to pick up how the nervous system represents differences in acoustic sounds in individual subjects, rather than, as in cortical-response studies, in groups of people. Importantly, this metric reflects the negative influence of background noise on sound encoding in the brain.

"There are numerous reasons for reading problems or for difficulty hearing speech in noisy situations, and we now have a metric that is practically applicable for measuring sound transcription deficits in individual children," said Kraus, the senior author of the study. "Auditory training and reducing background noise in classrooms, our research suggests, may provide significant benefit to poor readers."

For the study, electrodes were attached to the scalps of children with good and poor speech-in-noise perception skills. Sounds were delivered through earphones to measure the nervous system's ability to distinguish between "ba," "da" and "ga." In another part of the study, sentences were presented in increasingly noisy environments, and children were asked to repeat what they heard.

"In essence, the kids were called upon to do what they would do in a classroom, which is to try to understand what the kid next to them is saying while there is a cacophony of sounds, a rustling of papers, a scraping of chairs," Kraus said.

In a typical neural system there is a clear distinction in how "ba," "da" and "ga" are represented. The information is more accurately transcribed in good readers and children who are good at extracting speech presented in background noise.

"So if a poor reader is having difficulty making sound-to-meaning associations with the 'ba,' 'da' and 'ga' speech sounds, it will show up in the objective measure we used in our study," Kraus said.

Reflecting the interaction of cognitive and sensory processes, the brainstem response is not voluntary.

"The brainstem response is just what the brain does based on our auditory experience throughout our lives, but especially during development," Kraus said. "The way the brain responds to sound will reflect what language you speak, whether you've had musical experience and how you have used sounds."

The Auditory Neuroscience Lab has been a frontrunner in research that has helped establish the relationship between sound encoding in the brainstem, and how this process is affected by an individual's experience throughout the lifespan. In related research with significant implications, recent studies from the Kraus lab show that the process of hearing speech in noise is enhanced in musicians.

"The very transcription processes that are deficient in poor readers are enhanced in people with musical experience," Kraus said. "It makes sense for training programs for poor readers to involve music as well as speech sounds."

The co-authors of the PNAS study, "Subcortical differentiation of voiced stop consonants: Relationships to reading and speech-in-noise perception" are Jane Hornickel, Erika Skoe, Trent Nicol, Steven Zecker and Nina Kraus.

Lightning may have cooked dinner for early life

* 18:00 13 July 2009 **by Kate Ravilious**

Early microbes may have relied on lightning to cook their dinner, say researchers.

When lightning strikes sand or sediment, the path followed by the bolt can fuse into a glassy tube called a fulgurite. A new analysis of these remnants suggests that lightning fries the nutrient phosphorus into a more digestible form. Most phosphorus on Earth exists as oxidised phosphate, but many microbes prefer a rarer, partially oxidised phosphorus – phosphite.

Life fuel

Matthew Pasek and Kristin Block of the University of Arizona, Tucson, used an MRI scanner on 10 fulgurites and found that five contained phosphite. The surrounding soil only contained phosphate. They suggest that the high energy of a lightning strike strips an oxygen atom from phosphate compounds, creating phosphites. "Early life may have used phosphite to form its key biomolecules, like RNA and DNA," says Pasek.

Today, anthropogenic influences such as steel corrosion, provide the primary source of phosphites in the environment, but prior to anthropogenic input Pasek and Block believe lightning would have been the main source, producing up to 3000 kilograms of phosphites per year.

Journal reference: Nature Geoscience (DOI: 10.1038/ngeo580)

New drugs faster from natural compounds: A UC San Diego breakthrough

New or not? Cracking cyclic natural products for new drugs

Researchers have invented computational tools to decode and rapidly determine whether natural compounds collected in oceans and forests are new - or if these pharmaceutically promising compounds have already been described and are therefore not patentable.

This University of California, San Diego advance will finally enable scientists to rapidly characterize ring-shaped nonribosomal peptides (NRPs) - a class of natural compounds of intense interest due to their potential to yield or inspire new pharmaceuticals. The study will be published in the July 13 online issue of journal Nature Methods.

"These advances will speed the process by which we discover and describe new and biologically active molecules from organisms such as marine cyanobacteria, also known as blue-green algae. This, in turn, will accelerate the timeline for bringing new experimental therapies into clinical application," said William Gerwick, an author on the paper and a professor with the UC San Diego Scripps Institution of Oceanography Center for Marine Biotechnology and Biomedicine and the UCSD Skaggs School of Pharmacy and Pharmaceutical Sciences.

Researchers have invented computational tools to decode and rapidly determine whether natural compounds collected in oceans and forests are new -- or if these pharmaceutically promising compounds have already been described and are therefore not patentable. This University of California, San Diego, advance will finally enable scientists to rapidly characterize ring-shaped nonribosomal peptides -- a class of natural compounds of intense interest due to their potential to yield or inspire new pharmaceuticals. The study will be published in the July 13 online issue of journal Nature Methods. **UC San Diego**

(Read about Gerwick's work to discover drugs and protect Panama's natural and cultural resources at: http://explorations.ucsd.edu/Features/2009/Discovering_Diversity/images/07_2009_Feature.pdf)

Nonribosomal peptides (NRPs) often serve as chemical defenses for the bacteria that manufacture them. Starting from penicillin, NRPs have an unparalleled track record in pharmacology: most anti-cancer and anti-microbial agents are natural products or their derivatives. However, it is currently difficult, time-consuming and

costly to determine the molecular structure of NRPs which, by definition, are not directly inscribed in the genomes of the organisms that produce them.

"NRPs are one of the last bastions of pharmacologically important biological compounds that remain virtually untouched by computational research. As a result, it is currently one of the most painfully slow processes, it is a real bottleneck that we have now removed," said Pavel Pevzner, a computer science professor at UC San Diego's Jacobs School of Engineering and the corresponding author on the Nature Methods paper.

Researchers can now separate known compounds from those that are unknown.

"If I collect 1,000 ocean compounds, why waste time with compounds that are already known or patented?" added Nuno Bandeira, co-lead author on the paper, director of UC San Diego's Center for Computational Mass Spectrometry (CCMS) and a researcher at the UC San Diego division of Calit2, the California Institute of Telecommunications and Information Technology.

"Our algorithms can tell natural product researchers what their compounds are. Manual annotations should be something of the past," said Julio Ng, a co-lead author on the Nature Methods paper and a doctoral student in Bioinformatics at UC San Diego.

"Compound 879," for example, is a cyclic NRP discussed in the Nature Methods paper that was thought to be novel when it was isolated. A lengthy and expensive patenting process, however, uncovered that compound 879 had already been described as an antibiotic and named neoviridogrisen. The new UC San Diego algorithms would have quickly identified this fact. These algorithms make sense of the flood of tiny peptide fragments that are generated by machines called mass spectrometers that blast nonribosomal peptides apart and determine their sizes.

Two complementary processes are used to glean insights from data generated from the mass spectrometers that break the cyclic peptides into smaller and smaller linear pieces.

First, the authors present new algorithms that computers use to piece these peptide fragments back together in order to determine the chemical structure of a cyclic NRP. This is called "De Novo sequencing of NRPs."

Second, the researchers created "dereplication" tools for moving the other direction: taking the chemical structures of known NRPs and other related information and determining what the data signature would look like if a mass spectrometer had blown the compound part.

"Natural products have a long history in therapeutic development and many were discovered before the digital recording of mass spectrometry data. Therefore, we do not have an extensive mass spectrometry database for natural products as we do for proteomics. Our new tools enable dereplication without an experimental database to compare to," said Pieter Dorrestein, assistant professor in the UC San Diego Skaggs School of Pharmacy and Pharmaceutical Sciences and the Departments of Pharmacology, Chemistry and Biochemistry.

By using these two approaches, the researchers have created tools that enable researchers to both characterize the compound they have isolated and check to see if it, or something similar, has been previously described. With dereplication, researchers can leverage known information and are not forced to start from scratch each time a new compound needs to be identified.

"As long as the structure of the therapeutic or a related therapeutic or natural product is in the library, we can accurately dereplicate the molecule. This is the first generation of algorithms that can accomplish this and is a glimpse into the future of modern drug discovery."

Performing de novo sequencing without knowing amino acid masses is completely novel, according to Bandeira. "Until we created them, there were no algorithmic approaches available to do this from mass spectrometry data and it was generally thought to be impossible," said Bandeira, who earned his Ph.D. in computer science from the UC San Diego Jacobs School of Engineering.

The work allows mass spectrometry to go into the natural products field and actually do the identification and characterization of natural products in a high throughput fashion, explained Ng, a bioinformatics PhD student advised by Pavel Pevzner in computer science and Pieter Dorrestein in the Skaggs School of Pharmacy.

The researchers note that currently there is no one place to look for known NRPs, a situation they are trying to change with a new data repository effort.

The UC San Diego web-based tools for sequencing nonribosomal peptides (at not cost to researchers) are available at: bix.ucsd.edu/nrp

"This new study has shown that marine cyanobacteria are incredible sources of new molecules that may have medical value, especially in cancer, infectious diseases and neurological disorders," said Gerwick.

This project was supported by US National Institutes of Health grants 1-P41-RR024851-01, GM086283 and cA10u851, and by the PhRMA foundation.

"Dereplication and De Novo Sequencing of Nonribosomal Peptides," by Julio Ng,1,8 Nuno Bandeira,2,8 Wei-Ting Liu,3 Majid Ghassemian,3 Thomas L. Simmons,4 William Gerwick,4,5 Roger Linington,6 Pieter Dorrestein,3,5 and Pavel Pevzner2,7
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Study finds citrus-derived flavonoid prevents obesity

A flavonoid derived from citrus fruit has shown tremendous promise for preventing weight gain and other signs of metabolic syndrome which can lead to Type 2 Diabetes and increased risk of cardiovascular disease. The study, led by Murray Huff of the Robarts Research Institute at The University of Western Ontario looked at a flavonoid (plant-based bioactive molecule) called naringenin. The findings are published online in the journal Diabetes.

In the study one group of mice was fed a high-fat (western) diet to induce the symptoms of metabolic syndrome. A second group was fed the exact same diet and treated with naringenin. Naringenin corrected the elevations in triglyceride and cholesterol, prevented the development of insulin resistance and completely normalized glucose metabolism. The researchers found it worked by genetically reprogramming the liver to burn up excess fat, rather than store it.

"Furthermore, the marked obesity that develops in these mice was completely prevented by naringenin," says Huff, Director of the Vascular Biology Research Group at Robarts and Professor of Medicine and Biochemistry at the Schulich School of Medicine & Dentistry. "What was unique about the study was that the effects were independent of caloric intake, meaning the mice ate exactly the same amount of food and the same amount of fat. There was no suppression of appetite or decreased food intake, which are often the basis of strategies to reduce weight gain and its metabolic consequences."

While grapefruit has long been linked to weight loss diets, the concentrations of the citrus-derived flavonoid being studied are at higher levels than you could get from dietary components. "We are examining the pharmacological properties of naringenin," explains Huff. "The next step is to find out if naringenin prevents heart disease in animal models and to explore the feasibility of clinical trials to determine its safety and efficacy in humans."

This study investigated naringenin's preventative properties, but Huff is also investigating whether it can treat obesity and other existing metabolic problems. "These studies show naringenin, through its insulin-like properties, corrects many of the metabolic disturbances linked to insulin resistance and represents a promising therapeutic approach for metabolic syndrome."

The co-first authors on the paper are Erin Mulvihill and Emma Allister. The research was funded primarily by the Heart and Stroke Foundation of Ontario.

Regular moderate alcohol intake has cognitive benefits in older adults

WINSTON-SALEM, N.C. – A glass of wine here, a nightcap there – new research out of Wake Forest University School of Medicine suggests that moderate alcohol intake offers long-term cognitive protection and reduces the risk of dementia in older adults. The study is being presented at the Alzheimer's Association 2009 International Conference on Alzheimer's Disease (ICAD), in Vienna on July 13.

While previous studies have shown that moderate alcohol intake, particularly wine, is linked with lower risk of heart attacks and dementia, most of the studies have been done in middle-aged people, and it has remained unclear if the benefits of alcohol also apply to older adults in general or to older adults who might already have some mild memory problems. This is the largest, longest U.S. study to look at the effects of regular alcohol intake on dementia in seniors, both with and without memory problems.

"As of yet, we still have no cure for Alzheimer's disease and other dementias, so it is important to look for things that might help people prevent the disease," said Kaycee Sink, M.D., M.A.S (Masters of Advanced Studies in clinical research), a geriatrician and senior author of the paper. Moderate alcohol intake has been linked to lower risk of heart attacks, stroke, dementia, and death in middle-aged adults, but there is still controversy about alcohol intake in older adults."

For the study, researchers began by examining and interviewing 3,069 individuals, 75 years or older and most without any memory or thinking problems, about their drinking habits. Participants were asked about beer, wine, and liquor. The investigators then categorized the individuals as abstainers (non-drinkers), light drinkers (one to seven drinks per week), moderate drinkers (eight to 14 drinks per week), or heavy drinkers (more than 14 drinks per week). All types of alcohol were included.

The study subjects were then examined and interviewed every six months for six years to determine changes in their memory or thinking abilities and to monitor who developed dementia.

Researchers found that individuals who had no cognitive impairment at the start of the study and drank eight to 14 alcoholic beverages per week, or one to two per day, experienced an average 37 percent reduction in risk of developing dementia compared to individuals who did not drink at all and were classified as abstainers. The type of alcohol consumed did not matter.

For older adults who started the study with mild cognitive impairment, however, consumption of alcohol, at any amount, was associated with faster rates of cognitive decline. In addition, those who were classified in the heavy drinker category, consuming more than 14 drinks per week, were almost twice as likely to develop dementia during the study compared to non-drinkers with mild cognitive impairment.

"We were excited to see that even in older adults, moderate alcohol intake decreases the risk of dementia," Sink said. "It is important to note, however, that our study found a significantly higher risk of dementia for heavy drinkers who started the study with mild cognitive impairment."

The results are consistent with previous studies of middle-aged adults that suggest mild to moderate alcohol intake may reduce the risk of dementia, except in the case of individuals who already have mild to moderate cognitive impairment. The researchers' findings support current recommendations not to exceed one drink per day for women and two for men.

It is unclear from this study whether an abstainer who begins drinking moderately in his/her 70s will experience the same benefit or if the benefit is associated with a long pattern of moderate alcohol intake that continues on into old age.

"Our results suggest that older adults who are normal cognitively and drink moderately do not need to change their drinking behavior," Sink said. "If you have mild cognitive impairment however, it might benefit you to restrict your drinking and certainly not exceed one drink a day for women and two drinks a day for men.

"The participants in this study self-reported their alcohol intake at the start, but it is unusual for people to start drinking in their 70s, so we assume that the habits they reported at the start of the study reflect stable drinking habits," Sink added. "Without scientific data showing that it is beneficial, I wouldn't recommend that non-drinkers start drinking in their 70s.

"We are starting to make progress in understanding how to prevent and treat Alzheimer's and other dementias," she said. "It is a very exciting time to be involved in geriatrics research."

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Obesity contributes to rapid cartilage loss

OAK BROOK, Ill. – Obesity, among other factors, is strongly associated with an increased risk of rapid cartilage loss, according to a study published in the August issue of Radiology.

"We have isolated demographic and MRI-based risk factors for progressive cartilage loss," said the study's lead author, Frank W. Roemer, M.D., adjunct associate professor at Boston University and co-director of the Quantitative Imaging Center at the Department of Radiology at Boston University School of Medicine. "Increased baseline body mass index (BMI) was the only non-MRI-based predictor identified."

Tibio-femoral cartilage is a flexible connective tissue that covers and protects the bones of the knee. Cartilage damage can occur due to excessive wear and tear, injury, misalignment of the joint or other factors, including osteoarthritis.

Osteoarthritis is the most common form of arthritis, affecting 27 million Americans, according to the National Institute of Arthritis and Musculoskeletal and Skin Diseases. In osteoarthritis, the cartilage breaks down and, in severe cases, can completely wear away, leaving the joint without a cushion. The bones rub together, causing further damage, significant pain and loss of mobility.

The best way to prevent or slow cartilage loss and subsequent disability is to identify risk factors early.

"Osteoarthritis is a slowly progressive disorder, but a minority of patients with hardly any osteoarthritis at first diagnosis exhibit fast disease progression," Dr. Roemer said. "So we set out to identify baseline risk factors that might predict rapid cartilage loss in patients with early knee osteoarthritis or at high risk for the disease."

The researchers recruited patients from the Multicenter Osteoarthritis (MOST) Study, a prospective study of 3,026 people, age 50 – 79, at risk for osteoarthritis or with early x-ray evidence of the disease. The study is funded by the National Institute on Aging.

Dr. Roemer's study consisted of 347 knees in 336 patients. The patient group was comprised of 65.2 percent women, mean age 61.2, with a mean BMI of 29.5, which is classified as overweight. Recommended BMI typically ranges from 18.5 to 25. Only knees with minimal or no baseline cartilage damage were included. Of 347 knees selected for the study, 20.2 percent exhibited slow cartilage loss over the 30-month follow-up period

and 5.8 percent showed rapid cartilage loss. Rapid cartilage loss was defined by a whole organ magnetic imaging score of at least 5, indicating a large full thickness loss of 75 percent in any subregion of the knee during the follow-up period.

The results showed that the top risk factors contributing to rapid cartilage loss were baseline cartilage damage, high BMI, tears or other injury to the meniscus (the cartilage cushion at the knee joint) and severe lesions seen on MRI at the initial exam. Other predictors were synovitis (inflammation of the membrane that lines the joints) and effusion (abnormal build-up of joint fluid).

Excess weight was significantly associated with an increased risk of rapid cartilage loss. For a one-unit increase in BMI, the odds of rapid cartilage loss increased by 11 percent. No other demographic factors - including age, sex and ethnicity - were associated with rapid cartilage loss.

"As obesity is one of the few established risk factors for osteoarthritis, it is not surprising that obesity may also precede and predict rapid cartilage loss," Dr. Roemer said. "Weight loss is probably the most important factor to slow disease progression."

"Risk Factors for MRI-detected Rapid Cartilage Loss of the Tibio-femoral Joint over a 30-month Period: the MOST Study." Collaborating with Dr. Roemer were Yuqing Zhang, D.Sc., Jingbo Niu, M.D., John A. Lynch, Ph.D., Michel D. Crema, M.D., Monica D. Marra, M.D., Michael C. Nevitt, Ph.D., David T. Felson, M.D., M.P.H., Laura Hughes, Georges El-Khoury, M.D., Martin Englund, M.D., Ph.D., and Ali Guermazi, M.D., for MOST study investigators.

Repair of heart defect discovered incidentally during surgery may not have clear benefit

CHICAGO – Patients who have a heart defect known as patent foramen ovale incidentally discovered and repaired during surgery for a different condition may have an increased odds of postoperative stroke, along with no clear benefit on short-term outcomes or long-term survival, according to a study in the July 15 issue of JAMA.

Patent foramen ovale (PFO) is an opening in the upper chambers of the heart that failed to close naturally shortly after birth. The role that PFO plays in cryptogenic (of unknown cause) stroke remains controversial. "The debate over an association has existed for more than a century, but causal data linking PFO and cryptogenic stroke remain anecdotal. Epidemiological evidence is consistent with an increased risk of stroke associated with PFO but data are not conclusive. The paucity of evidence supporting PFO as the mechanism for cryptogenic stroke has left many questions in the field unanswered, including when PFO repair is appropriate," the authors write. A recent survey suggested that cardiothoracic (heart and chest) surgeons may alter planned procedures to repair a newly discovered PFO. How frequently this occurs and the impact on outcomes has been unknown, according to background information in the article.

Richard A. Krasuski, M.D., of the Cleveland Clinic, and colleagues examined the prevalence of PFO incidentally discovered during cardiothoracic surgery and investigated the relationship of repair (closure of the opening) on outcomes and long-term survival. The researchers reviewed the intraoperative transesophageal (through the esophagus) echocardiograms of 13,092 patients without prior diagnosis of PFO or atrial septal defect (an abnormal opening between the left and right atria of the heart) undergoing surgery at the Cleveland Clinic from 1995 through 2006. Postoperative outcomes were collected until discharge.

The authors found that PFO was intraoperatively discovered in 2,277 patients (17 percent). Of patients with newly discovered PFO, 639 (28 percent) underwent surgical repair, nearly all of which were suture closures (97 percent). Patients undergoing repair were more likely to be women, be younger, have a history of stroke or atrial fibrillation.

Further analysis indicated that patients with intraoperatively diagnosed PFO had similar rates of in-hospital stroke and hospital death. Length of hospital stay and days spent in the ICU were also similar between those with intraoperatively diagnosed PFO and those without.

Regarding outcomes for patients who underwent PFO repair compared with those who did not, the primary difference noted between the 2 groups was the rate of in-hospital stroke, which was 2.8 percent in the repaired group vs. 1.2 percent in the unrepaired group, representing a nearly 2.5 times greater odds of having in-hospital stroke. The rate of hospital deaths, hospital length of stay, ICU length of stay and time on cardiopulmonary bypass were all similar. Long-term analysis demonstrated that PFO repair was associated with no survival difference.

"In summary, PFO is commonly detected during intraoperative imaging at the time of cardiothoracic surgery. When incidentally discovered, it appears to have a benign short-term and long-term clinical course. While the number of events is small, there was no clear benefit of closure on short-term perioperative outcomes or longer-term mortality. The finding that repair may increase postoperative stroke risk should discourage routine surgical closure and foster further investigation to delineate whether there is any benefit in terms of long-term stroke prevention and which patients might benefit from this intervention," the authors conclude.

(JAMA. 2009;302[3]:290-297. Available pre-embargo to the media at www.jamamedia.org)

'Copernicium' proposed as name for newly discovered element 112

Proposed name honors astronomer Nicolaus Copernicus

This release is available in German.

In honor of scientist and astronomer Nicolaus Copernicus (1473-1543), the discovering team around Professor Sigurd Hofmann suggested the name “copernicium" with the element symbol "Cp" for the new element 112, discovered at the GSI Helmholtzzentrum für Schwerionenforschung (Center for Heavy Ion Research) in Darmstadt. It was Copernicus who discovered that the Earth orbits the Sun, thus paving the way for our modern view of the world. Thirteen years ago, element 112 was discovered by an international team of scientists at the GSI accelerator facility. A few weeks ago, the International Union of Pure and Applied Chemistry, IUPAC, officially confirmed their discovery. In around six months, IUPAC will officially endorse the new element's name. This period is set to allow the scientific community to discuss the suggested name "copernicium" before the IUPAC naming.

"After IUPAC officially recognized our discovery, we – that is all scientists involved in the discovery – agreed on proposing the name "copernicium" for the new element 112. We would like to honor an outstanding scientist, who changed our view of the world", says Sigurd Hofmann, head of the discovering team.

Copernicus was born 1473 in Torun; he died 1543 in Frombork, Poland. Working in the field of astronomy, he realized that the planets circle the Sun. His discovery refuted the then accepted belief that the Earth was the center of the universe. His finding was pivotal for the discovery of the gravitational force, which is responsible for the motion of the planets. It also led to the conclusion that the stars are incredibly far away and the universe inconceivably large, as the size and position of the stars does not change even though the Earth is moving. Furthermore, the new world view inspired by Copernicus had an impact on the human self-concept in theology and philosophy: humankind could no longer be seen as the center of the world.

With its planets revolving around the Sun on different orbits, the solar system is also a model for other physical systems. The structure of an atom is like a microcosm: its electrons orbit the atomic nucleus like the planets orbit the Sun. Exactly 112 electrons circle the atomic nucleus in an atom of the new element "copernicium".

Element 112 is the heaviest element in the periodic table, 277 times heavier than hydrogen. It is produced by a nuclear fusion, when bombarding zinc ions onto a lead target. As the element already decays after a split second, its existence can only be proved with the help of extremely fast and sensitive analysis methods. Twenty-one scientists from Germany, Finland, Russia and Slovakia have been involved in the experiments that led to the discovery of element 112.

Since 1981, GSI accelerator experiments have yielded the discovery of six chemical elements, which carry the atomic numbers 107 to 112. The discovering teams at GSI already named five of them: element 107 is called bohrium, element 108 hassium, element 109 meitnerium, element 110 darmstadtium, and element 111 is named roentgenium.

Study pinpoints drugs that prevent epilepsy, seizures after severe brain injury

Rat study shows blocking TGF-beta halts brain changes associated with epilepsy

Drugs that block a growth factor receptor on brain cells may prevent epilepsy after brain damage, according to a new study appearing in the July 15 issue of the Journal of Neuroscience.

Daniela Kaufer, an assistant professor of integrative biology at the University of California, Berkeley, graduate student Luisa P. Cacheaux, and their Israeli colleagues, graduate student Yaron David and neurosurgeon Alon Friedman, found that they could prevent the brain changes leading to epilepsy in rats by treating the animals with a drug that blocks transforming growth factor-beta (TGF-beta) receptors.

"When we add the blockers, the hyper-excitability that you normally see after brain trauma is gone," Cacheaux said. "The blockers also prevent a majority of the gene expression changes that we see following brain insult."

While seizures can take weeks to show up in rats, for the current paper, the researchers followed the rats for only four days after brain injury and treatment with TGF-beta blockers. Nevertheless, preliminary EEG studies of the rats' brains indicated that most animals remained seizure-free after a month.

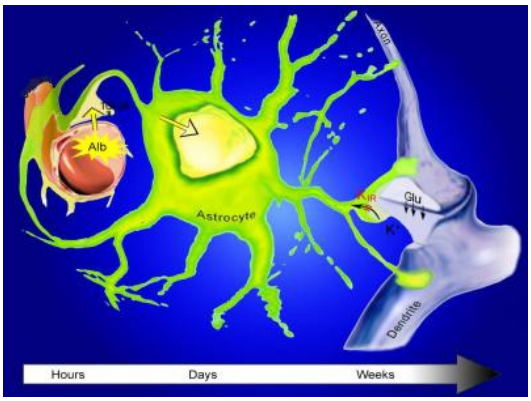
If the findings are confirmed in humans, a TGF-beta blocker may prevent many cases of epilepsy in accident victims or Iraqi war GIs who are victims of roadside bombs. Because of better medical care, many soldiers now survive severe traumatic brain injuries, yet those with severe head injuries are thought to have a 25 to 50 percent chance of eventually developing epileptic seizures. No treatment exists to prevent the development of epilepsy. Once epilepsy develops, drugs are the only option, and even those fail to control seizures in 30 percent of cases.

Because seizures develop weeks to years after an injury, there is a large window of opportunity in which patients could be treated with drugs to prevent the development of seizures, Kaufer said.

"The idea is to identify the brain injury patients that are very susceptible to epilepsy development – which may be possible to achieve using brain imaging – and then treat only those, not everybody, with a pretty benign drug that blocks the growth factors," she said. "At least in the rats, that works now."

The results are the culmination of more than 14 years of research to explore the hypothesis that trauma-induced epilepsy is caused by leakage of blood into the brain after injury, whether caused by trauma, brain tumors or infection, meningitis, or a hemorrhagic or ischemic stroke.

The idea originated with Friedman, who at the time was a physician in the Israeli army. Friedman, now an associate professor of physiology and neurosurgery at Israel's Ben-Gurion University of the Negev, hypothesized that breech of the blood brain barrier – a sheath of tightly joined cells that lines the capillaries in the brain to prevent intrusion of bacteria and potentially dangerous blood-borne molecules – somehow triggers events that destroy brain cells.



Friedman teamed up with Kaufer, then a graduate student at Hebrew University, on a series of experiments that has gradually provided support for the hypothesis and convinced many that this is a totally new and valuable way of looking at epilepsy. Over the years, their team systematically sifted through the components of the blood and, in 2007, reported that the main culprit in epileptogenesis seemed to be albumin, the main protein in blood serum.

The blood-brain barrier lining the capillaries often breaks after a severe brain injury, leaking blood into the brain. Albumin (Alb) in the blood serum binds to the TGF-beta receptor on astrocytes, often called glial cells, triggering a host of changes. One of these prevents astrocytes from mopping up the two neurotransmitters, glutamate (Glu) and potassium ions (K). As a result, these neurotransmitters flood the synapses between nerve cells, where the axon of one neuron touches the dendrite of another neuron, causing a constant excitation that can destroy the nerve cell. These changes take place over a period of days, providing a large window for delivery of drugs to block TGF-beta and prevent such changes. Dr. Dominik Zumsteg/Zurich University **Hospital**

In the current experiment, the researchers used serum albumin to trigger epileptogenesis in rats' brains and showed that albumin binds to TGF-beta receptors – there are two of them – and triggers the expression of a myriad of genes that are also turned on when the blood-brain barrier is breeched by other means. The genes expressed involve not only the normal TGF-beta pathway, but also genes involved in inflammation and in reducing inhibition of neurons. The actual damage is thought to be caused by uninhibited firing of neurons, so called hyper-excitability, that can exhaust and kill the neurons. Neuron death alters the nerve network in the brain, leading to a reorganization of neurons that creates short-circuits that precipitate seizures.

"Epilepsy is neurons firing together in synchrony, which leads to a storm of electricity," Kaufer said. "The brain by itself has mechanisms – release of inhibitory signals through inhibitory neurotransmitters – to shut down the firing. In epilepsy, you don't get shutdown of firing, and it spirals out of control.

"Here we have shown the beginning stages of the hyper-excitable state when a lot of inhibitory genes are being down-regulated, so that you don't have as much inhibition. And then the synchrony begins."

The team triggered the same processes by squirting TGF-beta1 into the brain, and were able to block these genetic changes by treating the brain with drugs that block both TGF-beta receptor 1 and TGF-beta receptor 2.

Kaufer noted that TGF-beta blockers might also prevent further damage in those with persistent seizures – a condition known as status epilepticus – because these non-stop seizures also open the blood brain barrier.

Interestingly, the albumin initially seems to be activating receptors on astrocytes, not neurons. Astrocytes, also called glial cells, are a population of "support cells" in the brain that research is showing may play an important role in many disease processes.

"The astrocytes really work well as sponges for glutamate and potassium ions, controlling neuronal excitability," Kaufer said. "Signaling in the TGF-beta pathway changes the properties of astrocytes, so you get higher potassium and glutamate in the vicinity of neurons and hyper-excitability, which makes the neurons start firing together, you get synchronous activity developing, and epilepsy follows."

Friedman continues to monitor treated rats with an electroencephalograph (EEG) to see what percentage of the rats goes on to develop epileptic seizures. Friedman and his group in Ben-Gurion's Brain Imaging Research Center are developing new imaging tools that allow measuring the blood-brain barrier opening in humans with brain injuries.

"You can have somebody with no epileptic seizures, but the barrier is open for weeks and months after the trauma. We have initial evidence to suggest that these patients are much more susceptible to the development of epilepsy," Friedman said.

Kaufer and her lab colleagues continue to explore the role of blood-brain barrier breech in epilepsy, and the impact of stress on the brain.

Other coauthors of the paper are Sebastian Ivens, a psychiatry resident, and Uwe Heinemann, a neurophysiologist, from the Institute of Neurophysiology at Charité Universitätsmedizin in Berlin; Alexander J. Lakhter and Guy Bar-Klein of the Zlotowski Center for Neuroscience at Ben-Gurion University; and Michael Shapira, UC Berkeley assistant professor of integrative biology. Kaufer and Cacheaux are also affiliated with UC Berkeley's Helen Wills Neuroscience Institute.

The work was supported by the CURE Foundation, German National Science Foundation, Mary Elizabeth Rennie Epilepsy Foundation, Israel Science Foundation and United States-Israel Binational Science Foundation.

The last supper of the hominids establishes the times they lived at the sites

In the French cave of Arago, an international team of scientists has analyzed the dental wear of the fossils of herbivorous animals hunted by Homo heidelbergensis. It is the first time that an analytical method has allowed the establishment of the length of human occupations at archaeological sites. The key is the last food that these hominids consumed.

For many years, the mobility of the groups of hominids and how long they spent in caves or outdoors has been a subject of discussion among scientists. Now, an international team headed by researchers from the Catalan Institute of Human Paleoecology and Social Evolution (IPHES) in Tarragona has based its studies on the dental fossils of animals hunted by hominids in order to determine the vegetation in the environment and the way of life of Homo heidelbergensis.

Florent Rivals is the main author and a researcher from the Catalan Institute for Research and Advanced Studies (ICREA), attached to the IPHES in Tarragona. "For the first time, a method has been put forward which allows us to establish the relative length of the human occupations at archaeological sites as, up until now, it was difficult to ascertain the difference between, for example, a single long-term occupation and a succession of shorter seasonal occupations in the same place", he explained to SINC.

In the study, recently published in the Journal of Human Evolution, the researchers analyze the dental wear of the ungulates (herbivorous mammals) caused by microscopic particles of opaline silica in plants. These marks appear when eating takes place and erase the previous ones. This is why they are so useful.

Thanks to the "last supper phenomenon", the scientists have been able to analyze the last food consumed by animals such as the Eurasian wild horse (Equus ferus), the mouflon (Ovis ammon antiqua) and the reindeer (Rangifer tarandus). "This method allows us to confirm the seasonal nature of the occupation", Rivals added. According to the team, the microwear of the teeth is sensitive to seasonal changes in the diet.

The application has allowed the researchers to estimate the length of the occupation of the site from the Lower Paleolithic Age in the cave of Arago (France) by the number of marks on the fossils and, therefore, the variation in the diet of several species of herbivores, as "each season presented food resources which were limited and different in the environment", the paleontologist clarified.

High and low periods of occupation

After confirming the hypothesis in present-day animals whose age and date of death was known to the scientists, the researchers demonstrated that, if a group of animals is seen during a specific season (a short-term occupation), the signs of dental wear undergo little variation. But if the occupation lasts several seasons, the dental marks are more diverse.

"If the animals are hunted during long periods of occupation, more variable dental wear would be expected", Rivals declared. In the case of the French cave of Arago, the study of the dental wear confirms that it was occupied in different ways. "With this method, we were able to prove that at the site, which belonged to Homo heidelbergensis, there is evidence of differing mobility, as there were highly mobile groups and others with little mobility", the scientist confirmed.

The Spanish and German researchers have combined this application with multidisciplinary studies of archaeological sites in order to apply it to other settlements of the Mid-Paleolithic Age such as Payre (France), Taubach (Germany) and Abric Romani (Spain).

References: *Rivals, Florent; Schulz, Ellen; Kaiser, Thomas M. "A new application of dental wear analyses: estimation of duration of hominid occupations in archaeological localities" Journal of Human Evolution 56(4): 329-339 abril de 2009.*

U of M Researchers Find Childhood Cancer Risk Rises with Mother's Age

MINNEAPOLIS/ST. PAUL (July 13, 2009) – Research from the Masonic Cancer Center, University of Minnesota indicates that a baby born to an older mother may have a slightly increased risk for many of the cancers that occur during childhood.

“Our finding shows that although the absolute risk is low, advancing maternal age may be a factor and explain why, after other factors are adjusted for, some children get cancer,” said Logan Spector, Ph.D., assistant professor of pediatrics and cancer epidemiology researcher. Spector and Kimberly Johnson, Ph.D., post

doctoral fellow in pediatric epidemiology, led the research team on this study. The results are published in the July 2009 issue of the journal Epidemiology.

Currently, about 1 in 435 children under the age of 15 in the United States gets cancer. Types of cancers most often affecting children include leukemia, lymphoma, central nervous system tumor, neuroblastoma, Wilms' tumor, bone cancer, and soft tissue sarcoma.

For this population-based case-control study, Spector and Johnson used information from birth registry records in New York, Washington, Minnesota, Texas, and California. The study included the records of 17,672 children in those states diagnosed with cancer at ages 0-14 years between 1980 and 2004 and 57,966 children not diagnosed with cancer.

"We saw that the risk of 7 of the 10 most common childhood cancers increased slightly, about 7-10 percent, with every five-year increase in maternal age," Spector said.

The researchers noted the father's age did not seem to matter once the mother's age was taken into account.

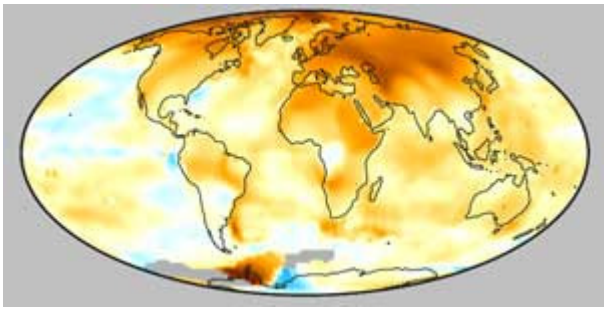
Spector and Johnson say more research needs to be done on why the risk for childhood cancer increases with advancing maternal age. Some of the possible explanations could be age-related changes in hormonal levels during pregnancy and alterations in DNA markings in eggs that can be transmitted to the offspring. "A mechanism of inherited mutation is consistent with our finding that the maternal age effect was strongest among children diagnosed with cancer at the earliest age," Spector said.

He anticipates that such research will take on greater importance as more women delay having children until older. Statistics show the percentage of babies born to women 30 years of age or older in the United States has risen from about 18 percent in 1970 to 37 percent in 2005.

This study was supported by the Children's Cancer Research Fund, Minneapolis; National Cancer Institute, Washington, D.C.; Fred Hutchinson Cancer Research Center, Seattle; and Centers for Disease Control and Prevention's National Program of Cancer Registries, Atlanta. Researchers working with Spector and Johnson on this study included Susan Carozza and Scott Horel, Texas A&M Health Science Center; Eric Chow, Fred Hutchinson Cancer Research Center and University of Washington; Erin Fox, Texas Department of State Health Services; Colleen McLaughlin, New York State Cancer Registry; Beth Mueller, University of Washington; Peggy Reynolds and Julie Von Behren, Northern California Cancer Center; and Susan Puumala, University of Minnesota.

Global warming: Our best guess is likely wrong

Unknown processes account for much of warming in ancient hot spell



No one knows exactly how much Earth's climate will warm due to carbon emissions, but a new study this week suggests scientists' best predictions about global warming might be incorrect.

The study, which appears in Nature Geoscience, found that climate models explain only about half of the heating that occurred during a well-documented period of rapid global warming in Earth's ancient past. The study, which was published online today, contains an analysis of published records from a period of rapid climatic warming about 55 million years ago known as the Palaeocene-Eocene thermal maximum, or PETM.

"In a nutshell, theoretical models cannot explain what we observe in the geological record," said oceanographer Gerald Dickens, a co-author of the study and professor of Earth science at Rice University. "There appears to be something fundamentally wrong with the way temperature and carbon are linked in climate models."

During the PETM, for reasons that are still unknown, the amount of carbon in Earth's atmosphere rose rapidly. For this reason, the PETM, which has been identified in hundreds of sediment core samples worldwide, is probably the best ancient climate analogue for present-day Earth.

In addition to rapidly rising levels of atmospheric carbon, global surface temperatures rose dramatically during the PETM. Average temperatures worldwide rose by about 7 degrees Celsius -- about 13 degrees Fahrenheit -- in the relatively short geological span of about 10,000 years.

Many of the findings come from studies of core samples drilled from the deep seafloor over the past two decades. When oceanographers study these samples, they can see changes in the carbon cycle during the PETM.

"You go along a core and everything's the same, the same, the same, and then suddenly you pass this time line and the carbon chemistry is completely different," Dickens said. "This has been documented time and again at sites all over the world."

Based on findings related to oceanic acidity levels during the PETM and on calculations about the cycling of carbon among the oceans, air, plants and soil, Dickens and co-authors Richard Zeebe of the University of

Hawaii and James Zachos of the University of California-Santa Cruz determined that the level of carbon dioxide in the atmosphere increased by about 70 percent during the PETM.

That's significant because it does not represent a doubling of atmospheric carbon dioxide. Since the start of the industrial revolution, carbon dioxide levels are believed to have risen by about one-third, largely due to the burning of fossil fuels. If present rates of fossil-fuel consumption continue, the doubling of carbon dioxide from fossil fuels will occur sometime within the next century or two.

Probing the past. ***Dark, red-brown ocean sediment layers reveal a telltale warming episode in cores retrieved by the drill ship JOIDES Resolution (inset).*** **J. C. Zachos*; (inset)* Integrated Ocean Drilling Program**

Doubling of atmospheric carbon dioxide is an oft-talked-about threshold, and today's climate models include accepted values for the climate's sensitivity to doubling. Using these accepted values and the PETM carbon data, the researchers found that the models could only explain about half of the warming that Earth experienced 55 million years ago.

The conclusion, Dickens said, is that something other than carbon dioxide caused much of the heating during the PETM. "Some feedback loop or other processes that aren't accounted for in these models -- the same ones used by the IPCC for current best estimates of 21st Century warming -- caused a substantial portion of the warming that occurred during the PETM."

To see the complete study, visit http://www.nature.com/ngeo/journal/vaop/ncurrent/abs/ngeo578.html.

Drug rescues memory lost to Alzheimer's disease

Study in mice finds oral therapy helps prevent neuron damage

Irvine, Calif., July 14, 2009 – A drug similar to one used in clinical trials for treatment of rheumatoid arthritis and psoriasis has been found to rescue memory in mice exhibiting Alzheimer's symptoms.

The discovery by UC Irvine scientists offers hope that a new treatment may be on the horizon for people in the early stages of Alzheimer's, the leading cause of elderly dementia afflicting more than 5 million people in the U.S. and for which no cure exists.

The drug, called PMX205, prevented inflamed immune cells from gathering in brain regions with Alzheimer's lesions called amyloid plaques. Cell inflammation in these areas accelerates neuron damage, exacerbating the disease.

"We used a multidisciplinary approach combining an understanding of immunology and neurobiology to uncover a completely different target than other therapies," said Andrea Tenner, lead author of the study that led to the findings and a molecular biology & biochemistry professor at UCI.

Study results are reported in the July 15 edition of the Journal of Immunology.

For 12 weeks, Tenner and colleagues added PMX205 to the drinking water of mice genetically altered to develop age-related Alzheimer's-like symptoms. The treatment occurred at an age when plaques were accumulating in their brains.

Scientists gave the treated mice learning and memory tests and then examined their brains for evidence of the disease. Alzheimer's mice that were not given the drug performed significantly worse on the test than normal mice. But – in all but one case – the treated Alzheimer's mice performed almost as well as the normal mice. Those with the rescued cognitive ability had more than 50 percent fewer Alzheimer's lesions and inflammatory immune cells than the untreated diseased mice.

PMX53, a similar drug that can be taken orally, passed Phase 1 human clinical trials for safety with no major problems reported. Possible side effects include an increased susceptibility to some infections. PMX205 is a modified version that may be more potent for treatment of brain disorders.

"This approach may work even better if combined with treatments targeting other problems in the Alzheimer's brain," said Tenner, also a professor of pathology and neurobiology & behavior, as well as a member of UCI's Institute for Immunology and Institute for Memory Impairments and Neurological Disorders, or UCI MIND.

In addition to Tenner, UCI graduate student Rahasson Ager and senior researcher Marisa Fonseca worked on this study. They collaborated with Australian scientists Trent Woodruff and Steve Taylor, who demonstrated the drug's effectiveness in rat models of other diseases.

The research was supported by the National Institutes of Health and the National Health and Medical Research Council of Australia.

Phase 3 Alzheimer's drug increases toxic beta amyloid in the brain - but still provides benefits

Immunotherapy targets Alzheimer's tau tangles and more doctors are diagnosing and treating mild cognitive impairment

Vienna, July 15, 2009 – New insights into how a Phase III Alzheimer's drug might work were among the advances in potential therapies targeting two abnormal brain proteins – beta amyloid and phosphorylated tau – that were reported today at the Alzheimer's Association 2009 International Conference on Alzheimer's Disease (ICAD 2009) in Vienna. Scientists also reported on how clinicians view and treat mild cognitive impairment (MCI), a research category used to define the state between normal aging and Alzheimer's, that is now being used widely in clinical practice.

"There are now more than 5 million people living with Alzheimer's disease in the United States. The cost of caring for people who now have Alzheimer's, and those who will get it in the next few years, will bankrupt the healthcare system and devastate Medicare and Medicaid," said Ralph Nixon, PhD, MD, vice chair of the Alzheimer's Association Medical & Scientific Advisory Council.

"But, as these studies and many hundreds more reported at ICAD 2009 show, there is hope. There are currently dozens of drugs in Phase II and III clinical trials for Alzheimer's. This, combined with advancements in diagnostic tools, has the potential to change the landscape of Alzheimer's in our lifetime. How fast we get there depends completely on the investment in research. We need more government and private dollars for Alzheimer's research now to capitalize on the progress we've made in the last decade," Nixon added.

Surprisingly, Dimebolin Increases Brain Beta Amyloid in Alzheimer's Mouse Models

Recent evidence suggests that dimebolin (Dimebon®, Medivation) may improve cognitive function in aged rodents and in people suffering from mild to moderate Alzheimer's, but how the drug produces these benefits remains unclear.

Samuel Gandy, MD, PhD, Mount Sinai Professor in Alzheimer's Disease Research; Professor of Neurology and Psychiatry; and Associate Director, Alzheimer's Disease Research Center at the Mount Sinai School of Medicine, New York, and colleagues John Cirrito PhD, and David M. Holtzman, MD, Professor and Chairman of the Department of Neurology at Washington University in St. Louis, MO, conducted a series of experiments in cells and in Alzheimer's mouse models to assess the effects of dimebolin on beta amyloid and other brain proteins known to be related to Alzheimer's disease.

Beta amyloid is a protein that is the main constituent of amyloid plaques found in the brains of people with Alzheimer's disease. It is widely considered a key player in the development and progression of Alzheimer's. The goal of anti-amyloid drugs that are currently in clinical trials is to reduce beta amyloid levels in the brain.

In a surprising result, the researchers at Washington University in St. Louis found that treatment with dimebolin caused an acute increase in brain beta amyloid levels in the animal models.

"This result is highly unexpected in what may prove to be a clinically beneficial Alzheimer's drug," Gandy said. "We need more research to further clarify how dimebolin affects beta amyloid levels in the brain."

"A number of ideas need to be pursued. It may turn out that the drug works by getting toxic amyloid out of brain nerve cells. Or, the effects of dimebolin on other brain systems may override its effect on increasing beta amyloid. Finally, the drug's beneficial actions might have nothing to do with amyloid, which, if true, indicates the existence of important therapeutic targets independent of beta amyloid," Gandy added.

The researchers note that so far they only studied acute systems, and it is conceivable that the chronic effect of dimebolin could be amyloid-lowering.

Immunotherapy Against Tau Tangles in Alzheimer's Mouse Models

Immunotherapy (treatment by inducing, enhancing, or suppressing an immune response) targeting beta amyloid is being researched widely by companies and academics as a therapeutic option for Alzheimer's disease. Earlier, late stage, anti-amyloid immunotherapy trials in people were complicated, and eventually stopped, when about six percent of participants developed brain inflammation. Current trials in this area are working in a variety of ways to eliminate this side effect.

Tau tangles, the other major Alzheimer's brain pathology, are now also receiving attention as a target for immunotherapy. Also known as neurofibrillary tangles (NFTs), these lesions consist of an abnormal folded protein (phosphorylated tau), and research shows their accumulation in the brain is more closely associated with the progression of Alzheimer's symptoms than amyloid.

Building on previous studies using this approach (for example, Asuni et al. (2007)), Hanna Rosenmann, Ph.D., head of the Laboratory of Molecular Neurogenetics, Department of Neurology, Hadassah University Hospital, Ein Kerem, and an Investigator (Associate-Senior Lecturer) at the Hebrew University Hadassah School of Medicine, Jerusalem, Israel, and colleagues performed immunization studies against tau tangle

pathology by immunizing NFT mice with a mixture of three phosphorylated-tau peptides (shortened versions of the full length tau protein that are phosphorylated like the NFTs). Previous experiments by this lab with non-phosphorylated full length tau caused brain inflammation in the animal models.

The researchers observed a robust decrease in the number of tau tangles in the brains of the mice immunized with the phosphorylated tau-peptides (~40%; $p < 0.001$), and detected anti-phosphorylated-tau antibodies in mouse serum. They found no evidence or symptoms of brain inflammation in the immunized mice.

According to Rosenmann, the decrease in tau tangles observed by her team is in accord with previous findings by Asuni's group, though Asuni immunized with a different phosphorylated tau peptide and immunization protocol.

"We believe that these results point to the therapeutic potential of phosphorylated-tau-immunotherapy in Alzheimer's," Rosenmann said. "We devoted significant effort to address not only the anti-tangle effect but also safety of a phosphorylated-tau vaccine. This was done in order to identify early in the preclinical stage any potential hazard of this potential Alzheimer's therapy."

Neurologists Views MCI as a Useful Clinical Diagnosis – Practice Guidelines Are Needed

Mild cognitive impairment (MCI) is a category of cognitive status that is used in research to define the state between normal aging and Alzheimer's, and it is now entering clinical practice. Little is known about how it is being used by clinicians or how they view the benefits and limitations of MCI as a clinical category.

In MCI, a person has problems with memory, language, or another mental function severe enough to be noticeable to other people and to show up on tests, but not serious enough to interfere with their daily life. Because the problems do not interfere with daily activities, the person is not diagnosed with Alzheimer's or another dementia. The best-studied type of MCI involves a memory problem and is called "amnestic MCI."

Research has shown that people with MCI have an increased risk of developing Alzheimer's over the next few years, especially when their main problem is memory. However, not everyone diagnosed with MCI goes on to develop Alzheimer's. There is currently no treatment for MCI approved by the FDA. Numerous clinical trials are investigating treatments to delay or prevent Alzheimer's in MCI populations.

Scott Roberts, PhD, Assistant Professor of Health Behavior & Health Education at the University of Michigan's School of Public Health; Jason Karlawish, MD, Associate Professor of Medicine and Medical Ethics with tenure, Senior Fellow of the Center for Bioethics and the Leonard Davis Institute of Health Economics, and Associate Scholar at the Center for Clinical Epidemiology and Biostatistics at the University of Pennsylvania; and colleagues sought to assess how neurologists are diagnosing and treating patients with mild cognitive symptoms and how they view MCI as a clinical diagnosis. They surveyed members of the American Academy of Neurology (AAN) who had indicated a clinical practice focus on aging/dementia or behavioral neurology in a recent AAN Member Census using mail, fax and the Internet.

420 clinicians (response rate=48%) completed the survey. 88% reported at least monthly encounters with patients experiencing mild cognitive symptoms. Most respondents recognize MCI as a clinical diagnosis (90%) and use its diagnostic code for billing purposes (70%). When seeing this population, most respondents report routinely making recommendations for monitoring and follow-up (88%), counseling patients on physical (78%) and mental exercise (75%), and communicating about risk of dementia (63%).

Most respondents (70%) prescribe cholinesterase inhibitors at least sometimes for this population, with memantine (39%) and "other" agents (e.g., vitamin E, gingko) prescribed less frequently. Cholinesterase inhibitors and memantine are FDA-approved drugs for Alzheimer's. Relatively few respondents routinely provide information on support services (27%) or a written summary of findings (15%).

Respondents endorsed several benefits of making a clinical diagnosis of MCI:

1 Labeling the problem is helpful (91%)
2 Involving the patient in planning for the future (87%)
3 Motivating the patient's risk reduction activities (85%)
4 Helping the family with financial planning (72%)
5 Prescribing medications useful for treating MCI (65%)

Some respondents noted potential drawbacks of MCI as a clinical diagnosis, including:

1 It is too difficult to diagnose accurately or reliably (23%)
2 It is usually better described as early AD (21%)
3 A diagnosis can cause unnecessary worry (20%)

"Our results show that neurologists regularly see and treat people with MCI, despite the fact that the medications they are prescribing are not FDA-approved for this particular diagnostic category," Roberts said. "Clinicians vary greatly in the education and support they provide or recommend for people with MCI, suggesting that there is a need for practice guidelines in this area. Millions of people can be classified as having MCI, and these numbers are expected to rise in coming years. It is important to establish professional consensus about appropriate care for this population."

According to Roberts, the AAN is currently engaged in an evidence-based medicine review of the literature to develop a new practice parameter for MCI.

Alzheimer's risk: Would you want to know?

ANN ARBOR, Mich.---When people learn they are predisposed to Alzheimer's disease, any depression or anxiety is not long lasting, a new study indicates.

These findings help address a longstanding debate about whether learning such information might cause lasting psychological harm, at least among those with a family history of Alzheimer's disease, says Scott Roberts, a University of Michigan researcher at the School of Public Health and co-author of the study findings, which appear today in the New England Journal of Medicine.

People with a family history are already at higher risk, which is further increased if they also carry a certain version of the gene called Apolipoprotein E (APOE).

Roberts and colleagues at Boston University, Case Western Reserve University, and Cornell Medical College tested 162 people with a parent with Alzheimer's, which means their risk for developing the disease by age 85 is about 30-35 percent, compared with the general population risk of about 10-15 percent.

After an educational session about Alzheimer's and genetic testing, researchers tested people for their APOE genotype to learn if they carried the genetic variant. The presence of the gene increases the risk for those with a family history of Alzheimer's to more than 50 percent. For subjects who did agree to the test, specially trained genetic counselors then disclosed results and researchers followed participants over one year to determine the impact of risk information.

The researchers measured anxiety, depression and test-related distress after six weeks, six months, and one year. Test-related distress did increase slightly at six weeks for people with the risk-increasing form of the gene, but not at 6 months or one year, Roberts said. Anxiety and depression levels remained stable.

"Some people might say, 'I'm thinking about this a lot,' but it didn't translate into long-term depression or anxiety," Roberts said. "The findings show if you do (disclose this genetic information) genetic counseling may be an important component to ensure that most people do not respond with significant distress.

"Genetic counselors help put the test results in context so that people understand the meaning and limits of the results," Roberts said. For example, for participants with a 55 percent lifetime risk, counselors explained that there was a 45 percent chance that they would never develop the disease.

The APOE link to Alzheimer's was identified in the 1990s, and traditionally, the medical community doesn't favor disclosure of the APOE genotype---or other genetic markers---unless telling patients directly impacts clinical treatment, Roberts says. However, now that private companies offer genetic testing for a variety of conditions, the debate over clinical utility versus personal utility is growing.

Some argue it's paternalistic to tell people what information they can or cannot know about their own genome, he says. After the initial educational session, 20 percent of the subjects opted out of the actual test, which means the majority wanted to know.

"I think most adult children of Alzheimer's patients would favor the right to at least have the choice," he said.

Roberts conducted this research while at Boston University. He came to the U-M in 2006.

Roberts is second author on the paper, called "Disclosure of APOE Genotype for Risk of Alzheimer's Disease," and co-principal investigator on the Risk Evaluation and Education for Alzheimer's Disease (REVEAL), a series of randomized clinical trials examining the impact of a genetic susceptibility testing program for adult children of people with Alzheimer's.

How the moon got its stripes

A new study has revealed the origins of tiger stripes and a subsurface ocean on Enceladus- one of Saturn's many moons. These geological features are believed to be the result of the moon's unusual chemical composition and not a hot core, shedding light on the evolution of planets and guiding future space exploration.

Dr Dave Stegman, a Centenary Research Fellow in the School of Earth Sciences at the University of Melbourne, led the study and says that part of the intrigue with Enceladus is that it was once presumed to be a lifeless, frozen ice ball until a water vapour plume was seen erupting from its surface in 2006.

"NASA's Cassini spacecraft recently revealed Enceladus as a dynamic place, recording geological features such as geysers emerging from the 'tiger stripes' which are thought to be cracks caused by tectonic activity on the south pole of the moon's surface," says Dr Stegman.

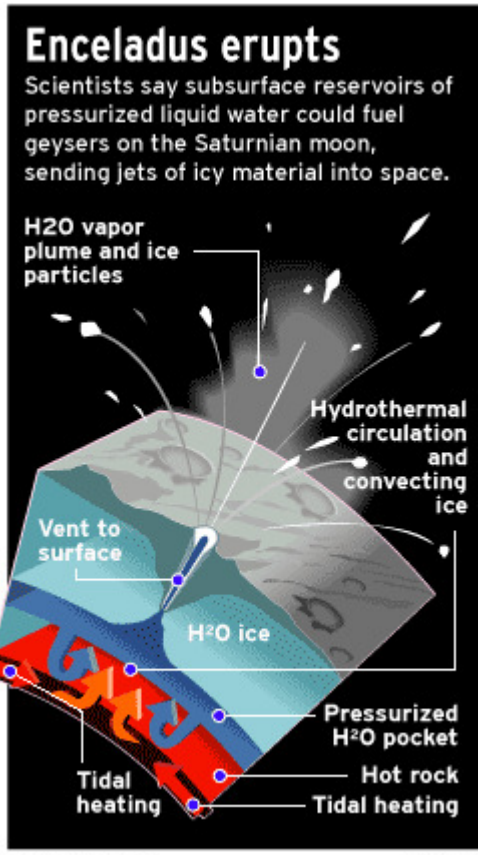
The moon is also one of the brightest objects in our solar system because the ice covering its surface reflects almost 100 percent of the sunlight that strikes it. One of Saturn's 53 moons (so far identified) Enceladus reflects so much of the sun's energy that its surface temperature is about -201° C (-330° F).

Grappling with how an inaccessible small moon with a completely frozen interior was capable of displaying geological activity, Dr Stegman and colleagues used computer simulations to virtually explore it.

Ammonia, usually found on Earth as an odorous gas used to make fertilizers, has been indirectly observed to be present in Enceladus and formed the basis of the study which is the first to reveal the origins of the subsurface ocean.

The model reveals that Enceladus initially had a frozen shell composed of a mixture of ammonia and water ice surrounding a rocky core. Over time, as Enceladus interacted with other moons, a small amount of heat was generated above the silicate core which made the ice shell separate into chemically distinct layers. An ammonia-enriched liquid layer formed on top of the core while a thin layer of pure water ice formed above that. The work will be published in the August issue of the planetary science journal, Icarus.

"We found that if a layer of pure water ice formed near the core, it would have enough buoyancy to rise upwards, and such a redistribution of mass can generate large tectonic stresses at the surface," says Dr Stegman. "However, the pure water ice rising up is also slightly warmer which causes the separation to occur again, this time forming an ammonia-enriched ocean just under the surface. The presence of ammonia, which acts as an anti-freeze, then helps keep the ocean in its liquid state."



"These simulations are an important step in understanding how planets evolve and provide questions to focus future space exploration and observations. It will hopefully progress our understanding of how and why planets and moons are different to each other."

Study reveals major genetic differences between blood and tissue cells

Important questions raised about genetic research based only on blood samples; new treatment in vascular disease foreseen at the same time

Research by a group of Montreal scientists calls into question one of the most basic assumptions of human genetics: that when it comes to DNA, every cell in the body is essentially identical to every other cell. Their results appear in the July issue of the journal Human Mutation.

This discovery may undercut the rationale behind numerous large-scale genetic studies conducted over the last 15 years, studies which were supposed to isolate the causes of scores of human diseases.

Except for cancer, samples of diseased tissue are difficult or even impossible to take from living patients. Thus, the vast majority of genetic samples used in large-scale studies come in the form of blood. However, if it turns out that blood and tissue cells do not match genetically, these ambitious and expensive genome-wide association studies may prove to have been essentially flawed from the outset.

This discovery sprang from an investigation into the underlying genetic causes of abdominal aortic aneurysms (AAA) led by Dr. Morris Schweitzer, Dr. Bruce Gottlieb, Dr. Lorraine Chalifour and colleagues at McGill University and the affiliated Lady Davis Institute for Medical Research at Montreal's Jewish General Hospital. The researchers focused on BAK, a gene that controls cell death.

What they found surprised them.

AAA is one of the rare vascular diseases where tissue samples are removed as part of patient therapy. When they compared them, the researchers discovered major differences between BAK genes in blood cells and tissue cells coming from the same individuals, with the suspected disease "trigger" residing only in the tissue. Moreover, the same differences were later evident in samples derived from healthy individuals.

"In multi-factorial diseases other than cancer, usually we can only look at the blood," explained Gottlieb, a geneticist with McGill's Centre for Translational Research in Cancer. "Traditionally when we have looked for genetic risk factors for, say, heart disease, we have assumed that the blood will tell us what's happening in the tissue. It now seems this is simply not the case."

"From a genetic perspective, therapeutic implications aside, the observation that not all cells are the same is extremely important. That's the bottom line," he added. "Genome-wide association studies were introduced with enormous hype several years ago, and people expected tremendous breakthroughs. They were going to draw blood samples from thousands or hundreds of thousands of individuals, and find the genes responsible for

disease. "Unfortunately, the reality of these studies has been very disappointing, and our discovery certainly could explain at least one of the reasons why."

AAA is a localized widening and weakening of the abdominal aorta, and primarily affects elderly Caucasian men who smoke, have high blood pressure and high cholesterol levels. It often has no symptoms, but can lead to aortic ruptures which are fatal in 90 per cent of cases.

If the mutations discovered in the tissue cells actually predispose for AAA, they present an ideal target for new therapies, and may have even wider therapeutic implications.

"This will probably have repercussions for vascular disease in general," said Schweitzer, of McGill's Department of Medicine. "We have not yet looked at coronary or cerebral arteries, but I would suspect that this mutation may be present across the board."

Schweitzer is optimistic that this discovery may lead to new treatments for vascular disease in the near to medium term. "The timeline might be five to 10 years," he said. "We have to do in-vitro cell culture experiments first, prove it in an animal model, and then develop a molecule or protein which will affect the mutated gene product. This is the first step, but it's an important step."

Pluto's kin may have invaded asteroid belt

* 18:55 15 July 2009 **by Rachel Courtland**

Millions of objects in the solar system's main asteroid belt may be icy interlopers from beyond Neptune that were flung into their present orbits after a violent migration of the giant planets, a new simulation suggests.

The solar system's main asteroid belt is a diverse mix of objects that orbit between Mars and Jupiter. These asteroids are generally thought to have formed close to their present locations, so their compositions should reflect the original distribution of gas and dust that surrounded the sun there and eventually condensed into solid bodies.

"People have just been assuming that what we see there, formed there," says Hal Levison of the Southwest Research Institute in Boulder, Colorado.

But Levison and others suspect some 20 per cent of the asteroids in the belt may be comet-like objects that were born in colder climes, beyond the orbit of Neptune.

Scattered objects

The results come from new simulations using a theory called the Nice model, which suggests the solar system's giant planets were born closer together and were surrounded by a vast disc of leftovers from the planets' formation called planetesimals (see Gas giants credited for solar system formation).

According to the model, Jupiter and Saturn entered a tight orbital dance about 700 million years after the solar system formed. Their gravity then flung Uranus and Neptune out into the planetesimal disc like bowling balls, causing the objects there to scatter like pins.

Past simulations have tracked the trajectories of these planetesimals and showed they wind up forming the Kuiper belt of icy debris where Pluto sits, some of the distant satellites of Jupiter and Saturn, and Trojan asteroids, bodies that share Jupiter's orbit but are centred at two points ahead of and behind the planet.

Outer belt

The new simulation fed planetesimals into the region surrounding Jupiter and Saturn as the planets moved to see how many would be captured as so-called Hilda asteroids, a group outside the main asteroid belt that orbits the sun three times for each two orbits of Jupiter.

While some of the objects became Hildas and Trojans, most of the captured objects wound up in the outer portion of the solar system's main asteroid belt.

Indeed, the outer asteroid belt boasts objects that are thought to have ice, while the inner asteroid belt is dominated by rocky bodies. "The interpretation has been that this represents a change in the nebula or the disc from which the planets formed," Levison told New Scientist.

Partial simulation

But if these icy outer objects are newcomers, it would mean that objects in the asteroid belt did not all form close to their present locations. "It says to the community that the assumptions you've been making don't necessarily have to be true," Levison says.

But Renu Malhotra of the University of Arizona in Tucson says she's not convinced of the estimate that 20 per cent of the asteroids in the main belt could come from the outer solar system.

That's because the new study didn't simulate the process that knocked the planetesimals out of their original orbits – it only sent objects from the outer solar system inwards to find out what fraction would be captured.

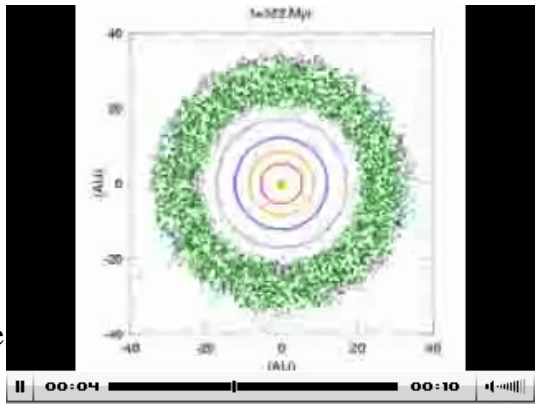
"It's not clear how statistically probable this is," Malhotra says. The simplest hypothesis, she adds, is that most of the asteroids in the main belt are original residents. *Journal reference: Nature (vol 460, p 364)*

Primitive asteroids in the main asteroid belt may have formed far from the sun

Many of the objects found today in the asteroid belt located between the orbits of Mars and Jupiter may have formed in the outermost reaches of the solar system, according to an international team of astronomers led by scientists from Southwest Research Institute (SwRI).

The team used numerical simulations to show that some comet-like objects residing in a disk outside the original orbit of the planets were scattered across the solar system and into the outer asteroid belt during a violent phase of planetary evolution.

Usually, the solar system is considered a place of relative permanence, with changes occurring gradually over hundreds of millions to billions of years. New models of planet formation indicate however, that at specific times, the architecture of the solar system experienced dramatic upheaval.



The animation simulates the first 1.2 billion years of solar system history. The orbits of the 4 giant planets are shown as color ellipses. The green dots show small comet-like objects. A small fraction of these objects become trapped in the asteroid belt when the orbits of the planets become unstable. In the simulation shown, this instability occurs at 880 million years. **Southwest Research Institute**

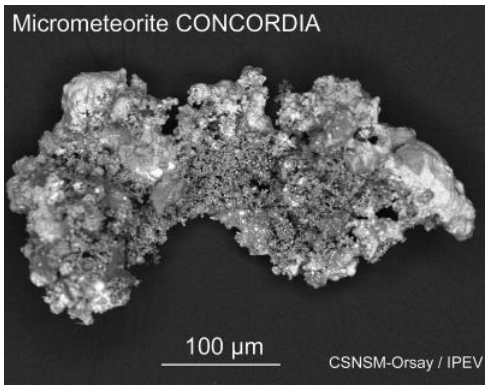
In particular, it now seems probable that approximately 3.9 billion years ago, the giant planets of our solar system -- Jupiter, Saturn, Uranus and Neptune -- rearranged themselves in a tumultuous spasm. "This last major event of planet formation appears to have affected nearly every nook and cranny of the solar system," says lead author Dr. Hal Levison of SwRI.

Key evidence for this event was first identified in the samples returned from the Moon by the Apollo astronauts. They tell us about an ancient cataclysmic bombardment where large asteroids and comets rained down on the Moon.

Scientists now recognize that this event was not limited solely to the Moon; it also affected the Earth and many other solar system bodies. "The existence of life on Earth, as well as the conditions that made our world habitable for us, are strongly linked to what happened at this distant time," states Dr. David Nesvorny of SwRI. The same dynamical conditions that devastated the planets also led to the capture of some would-be impactors in the asteroid belt. "In the classic movie 'Casablanca,' everybody comes to Rick's. Apparently throughout the solar system, the cool hangout for small objects is the asteroid belt," says Dr. William Bottke of SwRI.

Once in the asteroid belt, the embedded comet-like objects began to beat up both themselves and the asteroids. "Our model shows that comets are relatively easy to break up when hit by something, at least when compared to typical asteroids. It is unavoidable that some of the debris went on to land on asteroids, the Moon and the Earth. In fact, some of the leftovers may still be arriving today," says Dr. Alessandro Morbidelli of the Observatoire de la Cote d'Azur in Nice, France.

The team believes the surprising similarities between some micrometeorites landing on Earth and comet samples returned by NASA's Stardust mission are no accident. "There has been lots of debate about the nature of micrometeorites reaching the Earth," says Dr. Matthieu Gounelle of the Museum National d'Histoire Naturelle in Paris. "Some believe they are asteroidal, while others argue they are cometary. Our work suggests that in a sense, both camps may be right."



"Some of the meteorites that once resided in the asteroid belt show signs they were hit by 3.5 to 3.9 billion years ago. Our model allows us to make the case they were hit by captured comets or perhaps their fragments," adds Dr. Kleomenis Tsiganis of Aristotle University of Thessaloniki, Greece. "If so, they are telling us the same intriguing story as the lunar samples, namely that the solar system apparently went berserk and reconfigured itself about 4 billion years ago."

Researchers collected this micrometeorite in the vicinity of CONCORDIA station in central Antarctica (Dome C, 73°S, 123°E).: **CSNSM-Orsay-CNRS / IPEV**

Overall, the main asteroid belt contains a surprising diversity of objects ranging from primitive ice/rock mixtures to igneous rocks. The standard model used to explain this assumes that most asteroids formed in place from a primordial disk that experienced radical chemical changes within this zone. This model shows, however, that the observed diversity of the asteroid belt is not a direct reflection of the intrinsic compositional variation of the proto-planetary disk. These results fundamentally change our view of the asteroid belt.

Additional tests of this model will come from studies of meteorites, the asteroid belt, planet formation and the Moon. "The Moon and the asteroid belt may be the best and most accessible places in the solar system to understand this critical part of solar system history," says Levison. "We believe key evidence from these cold airless bodies may help us unlock the biggest 'cold case' of all time."

"The Contamination of the Asteroid Belt by Primordial Trans-Neptunian Objects," by Hal Levison, William Bottke, Alessandro Morbidelli, Matthieu Gounelle, David Nesvorny and Kleomenis Tsiganis, was published in the July 16 issue of Nature.

Funding for this research was provided by NASA's Outer Planets Research and Origins of Solar Systems programs. Additional support was provided by NASA's Lunar Science Institute.

Osteoporosis drug may save lives by strengthening immune system

DURHAM, N.C. -- An osteoporosis drug proven to save lives after hip fractures may do so by strengthening the body's immune system, according to geriatrics researchers at Duke University Medical Center.

In 2007, Duke researchers reported a 28 percent reduction in death among patients who received zoledronic acid (Reclast) within 90 days of surgery for a hip fracture. Zoledronic acid is a yearly intravenous injection of bisphosphonate that inhibits the progression of bone loss. The researchers also reported that the 2,111 people who participated in the study were 35 percent less likely to suffer another fracture.

"The findings marked the first time an osteoporosis medication was shown to have an effect on mortality, but they didn't tell us why the mortality rate was lower," says Cathleen Colon-Emeric, MD, an associate professor of medicine at Duke. "People assumed it was due to a reduction in secondary fractures. We wanted to know if that was the reason or were other conditions being affected by the medication."

In the current study, now online in the Journal of Bone and Mineral Research, Colon-Emeric and her colleagues report that the reduction in additional broken bones accounts for only eight percent of the mortality benefit. "Even after adjusting for secondary fractures and other risk factors, we found the risk of mortality was still 23 percent lower in the zoledronic acid-treated participants. That suggests the drug must work in other ways."

The link between osteoporosis and an increased risk of death has been observed for some time. Up to 25 percent of the 345,000 Americans hospitalized annually with hip fractures die within a year of their fracture. Typically, most patients die from cardiovascular problems like heart attacks, arrhythmias and strokes, infections such as pneumonia, and cancer.

"People who received the drug experienced common complications at the same rate as those who didn't," says Colon-Emeric. But the people in the zoledronic acid group were better able to survive these events. "In particular, people with certain cardiac problems such as arrhythmias and pneumonias were much less likely to die from those conditions."

Patients who lived in a nursing home before their broken hip, or who had high levels of cognitive impairment did not receive a mortality benefit from the drug.

It still remains unclear what role zoledronic acid plays. "We know it affects the immune system and inflammation, and both of those are important in fighting infection and cardiovascular disease," Colon-Emeric says. "It may be that the drug is changing the body's ability to fight off and recover from those illnesses." That idea will require confirmation in new studies.

Other investigators participating in this study include: Kenneth W. Lyles, MD and Carl F. Pieper, DrPH, Duke University Medical Center; Steven Boonen, MD, PhD, Katholieke Universiteit Leuven, Belgium; Pierre Delmas, MD, PhD, Claude Bernard University, Lyon, France; Jay Magaziner, PhD, University of Maryland; Peter Mesenbrink, PhD, of Novartis Pharmaceuticals, NJ; and Erik F. Eriksen, MD, DMsc, Novartis Pharma AG, Switzerland.

Baking soda: For cooking, cleaning and kidney health?

Sodium bicarbonate appears to slow progression of chronic kidney disease

A daily dose of sodium bicarbonate - baking soda, already used for baking, cleaning, acid indigestion, sunburn, and more - slows the decline of kidney function in some patients with advanced chronic kidney disease (CKD), reports an upcoming study in the Journal of the American Society of Nephrology (JASN). "This cheap and simple strategy also improves patients' nutritional status, and has the potential of translating into significant economic, quality of life, and clinical outcome benefits," comments Magdi Yaqoob, MD (Royal London Hospital).

The study included 134 patients with advanced CKD and low bicarbonate levels, also called metabolic acidosis. One group received a small daily dose of sodium bicarbonate in tablet form, in addition to their usual care. For this group, the rate of decline in kidney function was greatly reduced - about two-thirds slower than in patients. "In fact, in patients taking sodium bicarbonate, the rate of decline in kidney function was similar to the normal age-related decline," says Yaqoob.

Rapid progression of kidney disease occurred in just nine percent of patients taking sodium bicarbonate, compared to 45 percent of the other group. Patients taking sodium bicarbonate were also less likely to develop end-stage renal disease (ESRD) requiring dialysis.

Patients taking sodium bicarbonate also had improvement in several measures of nutrition. Although their sodium levels went up, this didn't lead to any problems with increased blood pressure.

Low bicarbonate levels are common in patients with CKD and can lead to a wide range of other problems. "This is the first randomized controlled study of its kind," says Yaqoob. "A simple remedy like sodium bicarbonate (baking soda), when used appropriately, can be very effective."

The researchers note some important limitations of their study - there was no placebo group and the researchers were aware of which patients were receiving sodium bicarbonate. "Our results will need validation in a multicenter study," says Yaqoob.

Other authors were Ione de Brito-Ashurst, RD, Mira Varaganum, PhD, and Martin J. Raftery, MD (William Harvey Research Institute and Barts and the London NHS Trust, London). The authors reported no financial disclosures.

The study entitled, Bicarbonate Supplementation Slows Progression of CKD and Improves Nutritional Status will appear online at http://jasn.asnjournals.org/ on July 16, 2009, doi 10.1681/ASN.2008111205.

Genetic source of muscular dystrophy neutralized

Researchers at the University of Rochester Medical Center have found a way to block the genetic flaw at the heart of a common form of muscular dystrophy. The results of the study, which were published today in the journal Science, could pave the way for new therapies that essentially reverse the symptoms of the disease.

The researchers used a synthetic molecule to break up deposits of toxic genetic material and re-establish the cellular activity that is disrupted by the disease. Because scientists believe that potentially all of the symptoms of myotonic dystrophy – the most common form of muscular dystrophy in adults – flow from this single genetic flaw, neutralizing it could potentially restore muscle function in people with the disease.

"This study establishes a proof of concept that could be followed to develop a successful treatment for myotonic dystrophy," said neurologist Charles Thornton, M.D., the senior author of the study and co-director of the University of Rochester Medical Center's Wellstone Muscular Dystrophy Cooperative Research Center. "It also demonstrates the potential to reverse established symptoms of the disease after they have developed, as opposed to simply preventing them from getting worse."

Myotonic dystrophy is a degenerative disease characterized by progressive muscle wasting and weakness. People with myotonic dystrophy have prolonged muscle tensing (myotonia) and are not able to relax certain muscles after use. The condition is particularly severe in the hand muscles and can cause a person's grip to lock making it difficult to perform rapid, repeated movements. Currently there is no medication to halt the progression of the disease.

Toxic RNA Holds Proteins Hostage

Although the genetic flaw that causes myotonic dystrophy was discovered in 1992, researchers studied the defect for many years before they had a clear understanding of the molecular events that ultimately produce the symptoms of the disease. Over time it became apparent that a central player in myotonic dystrophy was RNA, a versatile molecule that is very similar to DNA. RNA serves a vital function by relaying the genetic information from the nucleus – the protected area of the cell that houses DNA –out to the main body of the cell, where the instructions are used to build proteins. Every gene produces its own RNA, usually in multiple copies, and every RNA is a genetic blueprint of its parent gene.

The surprising aspect of myotonic dystrophy was that the genetic defect leads to production of a toxic RNA – the first example in human genetics in which RNA was cast in the role of molecular perpetrator. The errant RNA has a toxic effect because it grabs onto and holds hostage certain proteins, preventing them from carrying out their normal functions. For example, the capture of a protein called "muscleblind" causes the locking grip phenomenon that is a hallmark of the disease, a sign of faulty electrical control in muscle cells. Over time, the toxic RNA is produced in abundance and the captive proteins accumulate in deposits – or inclusions – that are visible in the cell's nucleus.

"An unexpected byproduct of research on myotonic dystrophy was that we were forced to change our ideas about the role of RNA in genetic disease," said Thornton. "Once we adjusted to this new concept, we realized that the prospects for developing treatment might be unusually good. No essential component of muscle is missing, but some important proteins are in the wrong place, stuck on the toxic RNA."

New Tools to Tackle Genetic Flaws

The Rochester team used a synthetic molecule – called an antisense morpholino oligonucleotide – that mimics a segment of the genetic code. In this case the morpholino was specifically designed to bind to the toxic RNA and neutralize its harmful effects by releasing the captured proteins. When injected into the muscle cells of mice with myotonic dystrophy the molecule found its way to the cell nucleus, broke up the deposits of toxic RNA, freed the captive muscleblind proteins, and ultimately improved the function of the muscle cells.

The researchers specifically observed a restoration of proper electrical control in the cells, which is a convenient way to monitor the condition. However, because the hostage proteins play a role in a myriad of other cellular functions, they believe that this treatment will ultimately alleviate other aspects of the disease as well.

"Based on our current understanding we would predict that by releasing the proteins held hostage, many of the symptoms of the disease may potentially be corrected by this approach," said URMC neurologist Thurman Wheeler, M.D., co-author of the study.

These genetic tools are relatively new and have provided researchers with a heretofore unprecedented ways to precisely target and manipulate genetic activity. "The current textbooks for medical students do not have chapters on antisense oligonucleotides, but this will change in the near future," said Thornton. "As compared to conventional drugs that work on proteins, antisense oligonucleotides work on RNA. They have been around for 20 years, but only recently is their full potential being realized. They provide great flexibility and they can be developed rapidly."

The authors are quick to point out that major hurdles must be overcome before this compound can be tested in humans. Specifically, a better delivery system must be developed to get this or a similar compound to where it needs to go in the body, and the potential side effects must be carefully analyzed. However, having established a general concept of what a treatment for myotonic dystrophy may look like, researchers believe that the next steps in developing an effective drug should go faster. Other authors on the study include University of Rochester Medical Center scientists Krysztof Sobczak, Ph.D., Robert J. Osborne, Ph.D., and Xiaoyan Lin, Ph.D., John Lueck, Ph.D., Robert Dirksen, Ph.D. Funding for the study was provided by the National Institute of Arthritis and Musculoskeletal and Skin Diseases, the National Institute of Neurological Disorders and Stroke, the Muscular Dystrophy Association, and the Run America Foundation.

Artistic tendencies linked to 'schizophrenia gene'

* 15:58 16 July 2009 **by Ewen Callaway**

We're all familiar with the stereotype of the tortured artist. Salvador Dali's various disorders and Sylvia Plath's depression spring to mind. Now new research seems to show why: a genetic mutation linked to psychosis and schizophrenia also influences creativity.

The finding could help to explain why mutations that increase a person's risk of developing mental illnesses such as schizophrenia and bipolar syndrome have been preserved, even preferred, during human evolution, says Szabolcs Kéri, a researcher at Semmelweis University in Budapest, Hungary, who carried out the study.

Kéri examined a gene involved in brain development called neuregulin 1, which previous studies have linked to a slightly increased risk of schizophrenia. Moreover, a single DNA letter mutation that affects how much of the neuregulin 1 protein is made in the brain has been linked to psychosis, poor memory and sensitivity to criticism.

About 50 per cent of healthy Europeans have one copy of this mutation, while 15 per cent possess two copies.

Creative thinking

To determine how these variations affect creativity, Kéri genotyped 200 adults who responded to adverts seeking creative and accomplished volunteers. He also gave the volunteers two tests of creative thinking, and devised an objective score of their creative achievements, such as filing a patent or writing a book.

People with two copies of the neuregulin 1 mutation – about 12 per cent of the study participants – tended to score notably higher on these measures of creativity, compared with other volunteers with one or no copy of the mutation. Those with one copy were also judged to be more creative, on average, than volunteers without the mutation. All told, the mutation explained between 3 and 8 per cent of the differences in creativity, Kéri says.

Exactly how neuregulin 1 affects creativity isn't clear. Volunteers with two copies of the mutation were no more likely than others to possess so-called schizotypal traits, such as paranoia, odd speech patterns and inappropriate emotions. This would suggest that the mutation's connection to mental illness does not entirely explain its link to creativity, Kéri says.

Dampening the brain

Rather, Kéri speculates that the mutation dampens a brain region that reins in mood and behaviour, called the prefrontal cortex. This change could unleash creative potential in some people and psychotic delusions in others.

Intelligence could be one factor that determines whether the neuregulin 1 mutation boosts creativity or contributes to psychosis. Kéri's volunteers tended to be smarter than average. In contrast, another study of families with a history of schizophrenia found that the same mutation was associated with lower intelligence and psychotic symptoms.

"My clinical experience is that high-IQ people with psychosis have more intellectual capacity to deal with psychotic experiences," Kéri says. "It's not enough to experience those feelings, you have to communicate them."

Intelligence's influence

Jeremy Hall, a geneticist at the University of Edinburgh in the UK who uncovered the link between the neuregulin 1 mutation and psychosis, agrees that the gene's effects are probably influenced by cognitive factors such as intelligence.

This doesn't mean that psychosis and creativity are the same, though. "There's always been this slightly romantic idea that madness and genius are the flipside to the same coin. How much is that true? Madness is often madness and doesn't have as much genetic association with intelligence," Hall says.

Bernard Crespi, a behavioural geneticist at Simon Fraser University in Burnaby, British Columbia, Canada, is holding his applause for now. "This is a very interesting study with remarkably strong results, though it must be replicated in an independent population before the results can be accepted with confidence," he says.

Journal reference: Psychological Science (DOI: 10.1111/j.1467-9280.2009.02398.x)

Primate archaeology, proposal of a new research field

Published in Nature by an international group of researchers

The use of tools by hominins - the primate group which includes humans (Homo) and chimpanzees and bonobos (Pan) - has been extensively researched by archaeologists and primatologists, both of who manifest the relevance of tool-use in understanding technology and the origins of human behaviour. However, recent research has highlighted the need to include other species such as gorillas and orangutans, as well as other extinct primate groups prior to hominins, in order to situate, for the first time in history, the full evolution of human behaviour within a greater biological context. With this aim, an international group of researchers from different universities, among them Universitat Autònoma de Barcelona (UAB), proposes to create a new interdisciplinary field called primate archaeology. The new field is described in detail in Nature.

The proposal of this new discipline, published in Nature, is the result of a meeting which took place in Cambridge in October 2008 with the objective of tackling a central theme in Palaeoanthropology: the origin and meaning of the first stone tools found at the archaeological sites studied until now. Researchers defending the need for this new field come from the University of Cambridge, University College London, Rutgers University, New Jersey, Kenyon College and Miami University, Ohio, Kyoto University, University of Calgary, the Centre International de la Réchérche Scientifique, Istituto di Scienze e Tecnologie della Cognizione de Roma, and Universitat Autònoma de Barcelona (UAB), with the collaboration of Rafael Mora, professor of the Department of Prehistory.

Researchers argue that since long there has been a separation between archaeology - with a focus on the study of "Homo" - and primatology, which has impeded applying a holistic perspective required to obtain more knowledge on the cognitive evolution of the order Primates and a better understanding of the biological, environmental and social contexts of their behaviour. With primate archaeology, researchers propose to combine methodologies used in both areas of knowledge, compare data obtained in the studies on tool-use carried out until now, both by humans and non-humans, and establish a new field which is needed for a variety of researches already taking place.

Thus, answers to the following questions could be found: how many extinct primate groups "invented" tool-use; what circumstances allowed or prevented such discoveries; how has continuous and repetitive use of tools influenced the anatomy of non-human primates; or why is it that only hominins have continued to use tools up until the present date? Researchers will also be able to delve deeper and learn about the precise moment in human evolution that tools began to be used, as well as which species made them and what uses these tools were given.

A necessary field

The need to establish a new field has grown in recent years due to the results obtained in researches carried out in the second half of the 20th century, which shed new light upon the possibility that a common ancestor of chimpanzees and humans - before the lineage split between humans and chimpanzees some 5-7 million years ago - already manufactured stone tools; or the use orangutans, capuchins and macaques presently give to rudimentary tools.

On the one hand, stone artefacts found in prehistoric Oldowan sites which date back 2.6 to 1.6 million years demonstrate much planning depth, optimal spatial coordination and manual dexterity carried out by a genus older than the earliest member of our genus (Homo habilis). This leads researchers to believe that it is likely that earlier, currently unrecognised, tool manufacturing occurred in more ancestral hominins (Australopithecus africanus, A. afarensis and A. garhi). On occasions more than 70 flakes struck from a single cobble were found

at these sites, which were obtained during the manufacturing of tools used mainly to cut meat, as well as hammers and anvils similar to the pounding tools used today by chimpanzees.
On the other hand, the relevant brain size and manipulative abilities of these hominins and modern great apes has led to speculation that the capacities of modern great apes may either directly or through analogy provide an insight into those of extinct hominins. Non-human primates take stones and plant material and modify them in order to obtain food or interact socially, and this challenges the conventional idea of hominins as the only creators found at archaeological sites. Although free-living primate populations have not been seen deliberately manufacturing tools, recent studies with animals in captivity (orangutans, bonobos and capuchins) have demonstrated that they can be taught to reduce stones or obtain sharp stone edges through different techniques.

Researchers conclude that primate archaeology will not only improve the integral development of both archaeology and primatology, but it will also favour the incorporation of other disciplines such as comparative anatomy or evolutionary ecology. All in all, this will help researchers obtain new and valuable data on the cognitive evolution of both human and non-human primates.

Reference article: "Primate archaeology". Michael Haslam, Adriana Hernandez-Aguilar, Victoria Ling, Susana Carvalho, Ignacio de la Torre, April DeStefano, Andrew Du, Bruce Hardy, Jack Harris, Linda Marchant, Tetsuro Matsuzawa, William McGrew, Julio Mercader, Rafael Mora, Michael Petraglia, Hélène Roche, Elisabetta Visalberghi, Rebecca Warren. Nature, Vol. 460 (pp. 339-344), 16 July 2009.

Neanderthals Were Few and Poised for Extinction

By Jeanna Bryner, Senior Writer

Neanderthals are of course extinct. But there never were very many of them, new research concludes.

In fact, new genetic evidence from the remains of six Neanderthals (Homo neanderthalensis) suggests the population hovered at an average of 1,500 females of reproductive age in Europe between 38,000 and 70,000 years ago, with the maximum estimate of 3,500 such female Neanderthals.

"It seems they never really took off in Eurasia in the way modern humans did later," said study researcher Adrian Briggs of the Max-Planck Institute for Evolutionary Anthropology in Germany.

The research, which will be published in the July 17 issue of the journal Science, suggests the small population size of our ancestral cousins may have been a factor in their demise.

"Because there never really were millions of them, they probably were more susceptible to some event that made them go extinct, which to me, suspiciously coincides with the emergence of modern humans," Briggs told LiveScience.

Ian Tattersall, curator of anthropology at the American Museum of Natural History in New York, who was not involved in the current research, said the study "does support notions that toward the end of last ice age, the Neanderthal population was declining as a result of harsh circumstances." He added, "I don't believe Neanderthals would've gone extinct if it wasn't for this new element, the Homo sapiens competing for the same resources."

Savvy Neanderthals

The Neanderthals inhabited the plains of Europe and parts of Asia as far back as 230,000 years ago. They disappeared from the fossil record more than 20,000 years ago, a few thousand years after modern humans appeared on the scene. Figuring out why Neanderthals died out and what they were like when alive have kept plenty of scientists busy.
Rather than the dumb cavemen characters starring in Geico car insurance ads, accumulating archaeological and genetic evidence shows Neanderthals were pretty sophisticated. They apparently hunted with blades and spear tips rivaling those of modern humans, ate marine mammals like seals and dolphins and sported brains that grew like ours. Their bodies likely looked similar to ours, and some Neanderthals showed off red locks on their heads.

Sparse population

Now, Briggs and his colleagues have used a new method that targets the genetic material of interest, analyzing so-called mitochondrial DNA from the fossils of six Neanderthals, who lived between 38,000 and 70,000 years ago. That genetic material comes from females and so can be used to trace maternal lineages.

To get a sense of the genetic diversity, and ultimately population size, the team compared the Neanderthal sequences with one another. Then, the researchers looked at such genetic information from 50 living humans from around the world, asking, "how different are their genes from one another?"

(Diversity of genes can provide indirect evidence for the number of breeding individuals, because with more people mating more genes are thrown into the mix, and vice versa.)

The Neanderthals had about three times less genetic diversity than the modern humans. Briggs suggests the entire population could be roughly estimated by doubling the number of females, which they set at no higher than 3,500.

In addition, the sequenced genetic material from the Neanderthals did not support any interbreeding among Neanderthals and modern humans. However, with such a small Neanderthal population, even if interbreeding occurred, the few Neanderthal genes thrown into the mix could've been sort of diluted out over time, Briggs said.

The entire Neanderthal genome is expected to be reported later this year and could shed more light on the interbreeding question, he added.

Cystic fibrosis treatments may have unseen long-term benefits

Cystic fibrosis medicines that help to break down mucus in the lungs may carry an unexpected long-term benefit, a study suggests

Cystic fibrosis medicines that help to break down mucus in the lungs may carry an unexpected long-term benefit, a study suggests.

The treatments not only help breathing in the short term - they may also make lung infections develop to be less harmful in the long run, research from the University of Edinburgh shows.

Scientists studied how bacteria which infect the lungs of cystic fibrosis patients gather nutrients from their surroundings. The work builds on the knowledge that most bacteria co-operate to scavenge what they need from their environment, but some bacteria do not actively hunt, instead stealing nutrients from neighbouring bacteria.

Scientists found that in a viscous environment, similar to thick mucus, the co-operating type of bacteria is most common. However, in a more liquid environment - similar to mucus having been broken down by medicine - the number of thieving bacteria increases, eventually outnumbering the scavenging type. In this environment, because the thieving bacteria are less adept at obtaining food, the bacterial growth slows down.

The results suggest that liquefying lung mucus would be expected to limit the impact of infection in cystic fibrosis.

Dr Rolf Kuemmerli, formerly a researcher at the University of Edinburgh, who led the study, said: "Treating cystic fibrosis patients with drugs that clear their lungs delivers short-term relief for the patient, but may have long-term health benefits too. We hope that our findings will underline the need for treatments that target mucus in the lungs."

Dr Freya Harrison of the University of Bath, who took part in the study, added: "Bacterial infections develop over time, and understanding how medical treatments affect this could be very important for managing long-term infections such as those found in cystic fibrosis."

Cystic fibrosis is an inherited condition that affects more than 8,000 people in the UK, according to the Cystic Fibrosis Trust. Thick mucus can clog the internal organs, especially the lungs and digestive system, making it hard to breathe and digest food.

The study, carried out by researchers at the Universities of Edinburgh, Oxford and Bath, was published in Proceedings of the Royal Society B. Work was supported by the Royal Society and the Leverhulme Trust.

Caltech, JPL scientists say that microbial mats built 3.4-billion-year-old stromatolites

Findings may provide insight into the origins of life on Earth, and even the search for life on Mars

PASADENA, Calif. - Stromatolites are dome- or column-like sedimentary rock structures that are formed in shallow water, layer by layer, over long periods of geologic time. Now, researchers from the California Institute of Technology (Caltech) and the Jet Propulsion Laboratory (JPL) have provided evidence that some of the most ancient stromatolites on our planet were built with the help of communities of equally ancient microorganisms, a finding that "adds unexpected depth to our understanding of the earliest record of life on Earth," notes JPL astrobiologist Abigail Allwood, a visitor in geology at Caltech.

Their research, published in a recent issue of the Proceedings of the National Academy of Sciences (PNAS), might also provide a new avenue for exploration in the search for signs of life on Mars.

This is a rare paleosurface view of what conical stromatolites would have looked like if you snorkeled in the shallows of the reef. **Abigail Allwood**

"Stromatolites grow by accreting sediment in shallow water," says John Grotzinger, the Fletcher Jones Professor of Geology at Caltech. "They get molded into these wave forms and, over time, the waves turn into discrete columns that propagate upward, like little knobs sticking up."

Geologists have long known that the large majority of the relatively young stromatolites they study - those half a billion years old or so - have a biological origin; they're formed with the help of layers of microbes that grow in a thin film on the seafloor.

How? The microbes' surface is coated in a mucilaginous substance to which sediment particles rolling past get stuck. "It has a strong flypaper effect," says Grotzinger. In addition, the microbes sprout a tangle of filaments that almost seem to grab the particles as they move along.

"The end result," says Grotzinger, "is that wherever the mat is, sediment gets trapped."

Thus it has become accepted that a dark band in a young stromatolite is indicative of organic material, he adds. "It's matter left behind where there once was a mat."

But when you look back 3.45 billion years, to the early Archean period of geologic history, things aren't quite so simple.

"Because stromatolites from this period of time have been around longer, more geologic processing has happened," Grotzinger says. Pushed deeper toward the center of Earth as time went by, these stromatolites were exposed to increasing, unrelenting heat. This is a problem when it comes to examining the stromatolites' potential biological beginnings, he explains, because heat degrades organic matter. "The hydrocarbons are driven off," he says. "What's left behind is a residue of nothing but carbon."

This is why there has been an ongoing debate among geologists as to whether or not the carbon found in these ancient rocks is diagnostic of life or not.

Proving the existence of life in younger rocks is fairly simple - all you have to do is extract the organic matter, and show that it came from the microorganisms. But there's no such cut-and-dried method for analyzing the older stromatolites. "When the rocks are old and have been heated up and beaten up," says Grotzinger, "all you have to look at is their texture and morphology."

Which is exactly what Allwood and Grotzinger did with samples gathered at the Strelley Pool stromatolite formation in Western Australia. The samples, says Grotzinger, were "incredibly well preserved." Dark lines of what was potentially organic matter were "clearly associated with the lamination, just like we see in younger rocks. That sort of relationship would be hard to explain without a biological mechanism."

"We already knew from our earlier work that we had an assemblage of stromatolites that was most plausibly interpreted as a microbial reef built by Early Archean microorganisms," adds Allwood, "but direct evidence of actual microorganisms was lacking in these ancient, altered rocks. There were no microfossils, no organic material, not even any of the microtextural hallmarks typically associated with microbially mediated sedimentary rocks."

So Allwood set about trying to find other types of evidence to test the biological hypothesis. To do so, she looked at what she calls the "microscale textures and fabrics in the rocks, patterns of textural variation through the stromatolites and - importantly - organic layers that looked like actual fossilized organic remnants of microbial mats within the stromatolites."

What she saw were "discrete, matlike layers of organic material that contoured the stromatolites from edge to edge, following steep slopes and continuing along low areas without thickening." She also found pieces of microbial mat incorporated into storm deposits, which disproved the idea that the organic material had been introduced into the rock more recently, rather than being laid down with the original sediment. "In addition," Allwood notes, "Raman spectroscopy showed that the organics had been 'cooked' to the same burial temperature as the host rock, again indicating the organics are not young contaminants."

Allwood says she, Grotzinger, and their team have collected enough evidence that it's no longer any "great leap" to accept these stromatolites as biological in origin. "I think the more we dig at these stromatolites, the more evidence we'll find of Early Archean life and the nature of Earth's early ecosystems," she says.

That's no small feat, since it's been difficult to prove that life existed at all that far back in the geologic record. "Recently there has been increasing but still indirect evidence suggesting life existed back then, but direct evidence of microorganisms, at the microscale, remained elusive due to poor preservation of the rocks," Allwood notes. "I think most people probably thought that these Early Archean rocks were too poorly preserved to yield such information."

The implications of the findings don't stop at life on Earth.

This is a close-up, cross-section view of the interior of a domical stromatolite. The black layers are the "cooked" organic remains of Early Archean microbial mats. **Abigail Allwood**

"One of my motivations for understanding stromatolites," Allwood says, "is the knowledge that if microbial communities once flourished on Mars, of all the traces they might leave in the rock record for us to discover, stromatolite and microbial reefs are arguably the most easily preserved and readily detected. Moreover, they're particularly likely to form in evaporative, mineral-precipitating settings such as those that have been identified on Mars. But to be able to interpret stromatolitic structures, we need a much more detailed understanding of how they form."

The other authors on the paper, "Controls on development and diversity of Early Archean stromatolites," are Mark Anderson, Max Coleman, and Isik Kanik from JPL; Andrew Knoll, the Fisher Professor of Natural History at Harvard University; and Ian Burch from the University of New South Wales in Australia.

Our Metallic Reflection: Considering Future Human-Android Interactions

Everyday human interaction is not what you would call perfect, so what if there was a third party added to the mix - like a metallic version of us? In a new article in Perspectives on Psychological Science, psychologist Neal J. Roese and computer scientist Eyal Amir from the University of Illinois at Urbana-Champaign investigate what human-android interactions may be like 50 years into the future.

With knowledge of present day technology, the scientists predict that within 50 years androids will be able to speak in human-like voices, identify spoken words with precision, answer questions from a body of textual information, walk and run in a human-like motion, display realistic facial expressions, and detect others' emotions through visual processing.

However, even with these advances, it will be more than 50 years before we see the human-acting and organic-looking androids of sci-fi movies. By 2060, it is predicted that androids will still be unable to detect aspects of natural language, and be incapable of forming conclusions from visual sensory input (specifically, seeing but not understanding). The most difficult development in artificial intelligence (AI) is trying to program the "Theory of Mind," or the effortless human ability to process other people's speech, actions, underlying motives, and emotional state.

Roese and Amir predict that by 2060 androids will be used for menial jobs, such as toll collectors, where the presence of a non-human is practical, but not frightening. A major worker shift from people to androids, similar to the shift to machines in factories, is expected to occur.

The psychological challenges of human-android interaction involve the absence of basic human functions such as blinking, body language, eye contact, and the coordination of personal space in an android, which could potentially make people uneasy when interacting with them.

But would people be more or less comfortable interacting with androids if they were ever indistinguishable from humans? Would stereotypes towards non-humans occur? Being unable to gauge who is human and who is not might cause confusion and fear in the public, even though we are the ones creating androids for our own benefit.

Roese and Amir conclude that the psychological impacts of human-android interaction must be considered in the present to shape android development in the future.

Why winning athletes are getting bigger

DURHAM, N.C. -- While watching swimmers line up during the 2008 Olympic Games in Beijing, former Olympic swimmer and NBC Sports commentator Rowdy Gaines quipped that swimmers keep getting bigger, with the shortest one in the current race towering over the average spectator.

What may have been seen as an off-hand remark turns out to illustrate a trend in human development -- elite athletes are getting bigger and bigger.

What Gaines did not know was that a new theory by Duke University engineers has indeed showed that not only have Olympic swimmers and sprinters gotten bigger and faster over the past 100 years, but they have grown at a much faster rate than the normal population.

Furthermore, the researchers said, this pattern of growth can be predicted by the constructal theory, a Duke-inspired theory of design in nature that explains such diverse phenomena as river basin formation and the capillary structure of tree branches and roots. (www.constructal.org).

In a new analysis, Jordan Charles, an engineering student who graduated this spring, collected the heights and weights of the fastest swimmers (100 meters) and sprinters (100 meters) for world record winners since 1900. He then correlated the size growth of these athletes with their winning times.

"The trends revealed by our analysis suggest that speed records will continue to be dominated by heavier and taller athletes," said Charles, who worked with senior author Adrian Bejan, engineering professor who came up with the constructal theory 13 years ago. The results of their analysis were published online in the Journal of Experimental Biology. "We believe that this is due to the constructal rules of animal locomotion and not the contemporary increase in the average size of humans."

Specifically, while the average human has gained about 1.9 inches in height since 1900, Charles' research showed that the fastest swimmers have grown 4.5 inches and the swiftest runners have grown 6.4 inches.

The theoretical rules of animal locomotion generally state that larger animals should move faster than smaller animals. In his constructal theory, Bejan linked all three forms of animal locomotion -- running, swimming and flying. Bejan argues that the three forms of locomotion involve two basic forces: lifting weight vertically and overcoming drag horizontally. Therefore, they can be described by the same mathematical formulas. (http://www.pratt.duke.edu/news/?id=1692)

Using these insights, the researchers can predict running speeds during the Greek or Roman empires, for example. In those days, obviously, time was not kept.

"In antiquity, body weights were roughly 70 percent less than they are today," Charles said. "Using our theory, a 100-meter dash that is won in 13 seconds would have taken about 14 seconds back then."

Charles, a varsity breaststroke swimmer during his time at Duke, said this new way of looking at locomotion and size validates a particular practice in swim training, though for a different reason. Swimmers are urged by their coaches to raise their body as far as they can out of the water with each stroke as a means of increasing their speed.

"It was thought that the swimmer would experience less friction drag in the air than in the water," Charles said. "However, when the body is higher above the water, it falls faster and more forward when it hits the water. The larger wave that occurs is faster and propels the body forward. A larger swimmer would get a heightened effect. Right advice, wrong reason."

In an almost whimsical corollary, the authors suggest that if athletes of all sizes are to compete in these kinds of events, weight classes might be needed.

"In the future, the fastest athletes can be predicted to be heavier and taller," Bejan said. "If the winners' podium is to include athletes of all sizes, then speed competitions might have to be divided into weight categories. Larger athletes lift, push and punch harder than smaller athletes, and this led to the establishment of weight classes in certain sports, like boxing, wrestling or weight-lifting.

Large epidemiologic study supports brain power of fish in older people

Experts estimate that over 24 million people worldwide suffer from dementia, and many of these people live in low- and middle-income countries. Recently, there has been growing interest in whether dietary factors, particularly oily fish and meat, might influence the onset and/or severity of dementia. Oily fish are rich in omega-3 long-chain polyunsaturated fatty acids, which some studies suggest are positively related to cognitive function in later life. Conversely, there is a suggestion from some studies that increased meat consumption may be related to cognitive decline. To examine this, a group of international researchers studied older people in 7 middle- to low-income countries. You can read the results of their study in the August 2009 issue of the American Journal of Clinical Nutrition.

Data from 14,960 participants (≥65 y of age) living in China, India, Cuba, the Dominican Republic, Venezuela, Mexico, and Peru were analyzed. Dietary habits were assessed by using standard, culturally appropriate face-to-face interviews, and dementia was diagnosed by using validated culturally and educationally fair criteria.

In each of the study countries, except India, there was an inverse association between fish consumption and dementia prevalence. These data extend to low- and middle-income countries previous conclusions from industrialized countries that increased fish consumption is associated with lower dementia prevalence in later life. The authors propose that this relation is not due to poor overall nutritional status in those with dementia, because meat consumption tended to be higher in this group. The relation between meat consumption and dementia remains unclear.

To access full text of the study visit: http://www.nutrition.org/media/publications/ajcnAug709.pdf

Airport travelators actually slow passengers down

* 18 July 2009 **by MacGregor Campbell**

YOUR flight leaves in 10 minutes and you've only just made it through security. As you run to your gate you come to a corridor with a moving walkway. Should you hop on?

Maybe not. People on travelators actually tend to slow their pace, making time-savings minimal, and a new study helps to explain why.

Manoj Srinivasan, a locomotion researcher at Princeton University, created two mathematical models of how people travel on such walkways (Chaos, DOI: 10.1063/1.3141428). In the first, he assumed people walk in a way that minimises the energy they expend, a standard theory in locomotion research. In the second, he assumed people walk in a way that best makes sense of the signals relayed from their eyes and legs.

Srinivasan's models predict that when a person steps onto a moving walkway, they slow their foot speed by about half the speed of the walkway. This suggests that our desires to conserve energy and to resolve the conflict between visual cues and leg muscle signals - your eyes tell you that you are going faster than your legs are taking you - slow us down so that our total speed is only slightly greater than it would have been on regular ground.

This may save energy, but even under ideal conditions of no congestion and no baggage a walkway only makes a small difference in travel time - about 11 seconds for a 100-metre stretch.

Even with no congestion and no baggage, a walkway only makes a small difference in travel time

The findings help to explain earlier work by Seth Young, now at Ohio State University, who observed travellers at San Francisco and Cleveland airports slowing down on moving walkways, though not as drastically as Srinivasan's model suggests (Transportation Research Record, DOI: 10.3141/1674-03).

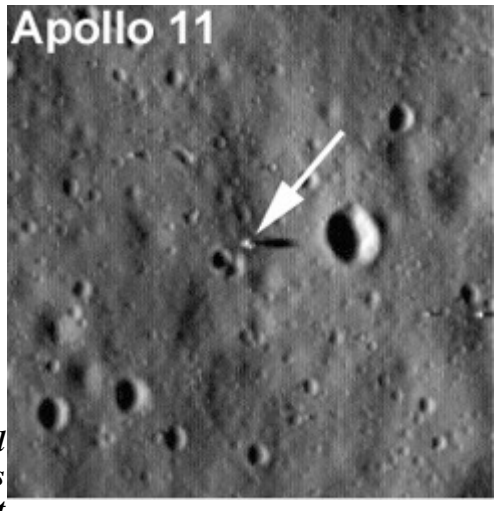
If there is no congestion, people on travelators are marginally faster than on normal ground. However, Young found that the odds that other travellers will block the way are such that on average, it takes longer to get from A to B on a moving walkway.

"Moving walkways are the only form of transportation that actually slow people down," says Young. He believes their main benefit is to reduce walking distance, giving weary travellers a chance to rest.

New NASA Photos Show Apollo Leftovers on the Moon

By The Associated Press

WASHINGTON (AP) -- New NASA photos of the moon show the leftovers from man's exploration 40 years ago. For the first time, photos from space pinpoint equipment left behind from Apollo landings, and even the well-worn tracks made by astronauts on the moon surface. The images are from the Lunar Reconnaissance Orbiter, which was launched last month and now circles the moon in search of future landing sites.



The lunar module Eagle, which was used to carry Apollo 11 astronauts Neil Armstrong and Buzz Aldrin down to the lunar surface on 20 July 1969 is clearly seen in the image on the left. When LRO settles into its final orbit later this year, it will deliver images that are at least twice as sharp as this one. **(Image: NASA/GSFC/ASU)**

The photos were released Friday, in time for the 40th anniversary of the first moon landing on July 20, 1969. A picture of the Apollo 11 site shows the Eagle lunar module used by Neil Armstrong and Buzz Aldrin.

"It was really great to see the hardware sitting on the surface, waiting for us to come back," said Arizona State University scientist Mark Robinson, who runs the camera on the orbiter. "You could actually see the descent module sitting on the surface."

But that's only if you know where to look. NASA helps out by putting a giant arrow on each photo. The lunar landers look to be square white blobs; the Eagle is a fuzzy image near a crater.

NASA landed on the moon six times, but the orbital camera so far has only photographed five of the landing sites. Apollo 12 will be done later. That leaves Apollo 11 and Apollo 14 through 17. Apollo 13 never landed on the moon because of an explosion on board the ship on the way to the moon.

The images for Apollo 14 are the best so far. Taken on Wednesday, they show the path made by astronauts Alan Shepard Jr. and Edgar Mitchell as they went back and forth from the lander to the work site.

Robinson said the route was "a high traffic zone, sort of like when you go in an old building and the carpet is worn down." A similar but lighter path could be seen at the Apollo 17 site.

Also at the Apollo 14 site, a close examination shows a trail made by the cart used to carry tools, Robinson said.

The photos varied in quality based on how high up the satellite was and the angle of the sun. For Apollo 11, the spaceship was taking pictures from 70 miles above. For Apollo 14, it was six miles closer.

In the next couple months, as the lunar satellite starts its mission to map the moon for future landing sites for astronauts, it will get much better photos, Robinson said. The mission is a first step in NASA's effort to return humans to the moon by 2020.

Other robotic probes, including those launched by Japan and India, have looked for signs that man was on the moon, but their cameras weren't strong enough, NASA officials said.

The Lunar Reconnaissance Orbiter was launched with a second spacecraft that was designed to crash into the moon in the fall to try to find buried ice. The total cost of the mission is $583 million.

On the Net: NASA's LRO site: *http://www.nasa.gov/mission(underscore)pages/LRO/main/index.html*

Grand Plans for Moon and Mars, Budget Permitting

By KENNETH CHANG

NASA's program to send astronauts back to the Moon by 2020 is often called "Apollo on steroids."

To detractors, this is a description of disparagement - treading the same path as 40 years ago, only with bigger, costlier rockets.

But the officials at the National Aeronautics and Space Administration say the new missions will be much grander - astronauts living on the Moon for months at a time, driving hundreds of miles across the lunar surface and, for the first time, building an outpost on ground that is not Earth.

"It's not just flags and footsteps," said John Olson, director of the office within NASA's exploration systems mission directorate that integrates the disparate parts of a lunar program. "It's substantially important work."

The technologies and skills, the NASA officials say, are essential before pushing on to Mars, the next major destination. Scientists see several exciting research possibilities on the Moon, like building a radio telescope on the far side, shielded from the noise from Earth, and looking for layers of frost in shadowed craters near the poles, which may preserve hints of the solar system's past.

But with trillion-dollar federal budget deficits and a blue ribbon panel now re-evaluating the United States' human space flight program, there is some question whether the lunar designs that NASA has drawn up over the past five years will be built. The agency could be told to focus on robotic missions, to undertake cheaper alternatives for getting to the Moon or to shift its target to something else, like an asteroid.

If NASA does not go to the Moon, it is not clear anyone else would go, either. Some Chinese and Russian officials have talked about establishing a Moon base sometime around 2025, but neither China nor Russia has made any official pronouncements, and their current rockets are too small for the task.

The nascent private space industry, which has yet to send anyone into orbit, does not seem likely to head to the Moon, either, with no obvious profit windfall to offset the billions of dollars in cost. "The idea that a private investor can put together the funds to develop rockets capable of a lunar mission is extremely speculative, verging on fantasy," said John Logsdon, chairman of space history at the National Air and Space Museum.

What is perhaps more likely is that the Moon program will, like the International Space Station, become a combined effort of multiple nations.

At the first public meeting of the panel reviewing NASA's human spaceflight program, Gen. Anatoly N. Perminov, the head of Roscosmos, the Russian space agency, said by telephone, "Roscosmos supports the necessity of involving technical and scientific potential of other countries for such large-scale projects," including sending astronauts to the Moon and Mars.

NASA has named its next-generation space transportation system the Constellation program. The first two pieces of Constellation - the Ares I rocket with an Orion crew capsule - are to take astronauts to the International Space Station beginning in 2015.

Two additional pieces are needed for the trip to the Moon: the Ares V, a behemoth "heavy lifter" rocket, and the Altair lunar lander, for getting the astronauts down to the Moon's surface.

At first glance, the Ares V looks more or less like the Saturn V from the Apollo era, and the Altair looks like a fashion update - with a rounder, more modern aesthetic - of the lander that carried Neil Armstrong and Buzz Aldrin to the Sea of Tranquility.

"Physics and engineering drive a lot of the designs," Dr. Olson said, explaining the similarities.

Then there are the differences. The Ares V is to be just a tad taller than the Saturn V - 381 feet versus 363 feet. But the Ares V will be able to send about 140,000 pounds on a journey to the Moon, or 40 percent more than the Saturn V.

The Ares V, unlike the Saturn V, will not carry astronauts as it lifts off. Following the recommendations of panel that investigated the loss of the space shuttle Columbia, the Constellation program puts crew and cargo on separate rockets to improve astronaut safety. While most of the spacecraft hardware - the Altair lander and the Earth departure stage - goes up on the Ares V, a crew of four astronauts will launch in an Orion capsule on top of an Ares I.

In Earth orbit, the Orion capsule will dock with the components sent up by the Ares V, and the combined spacecraft will then head to the Moon.

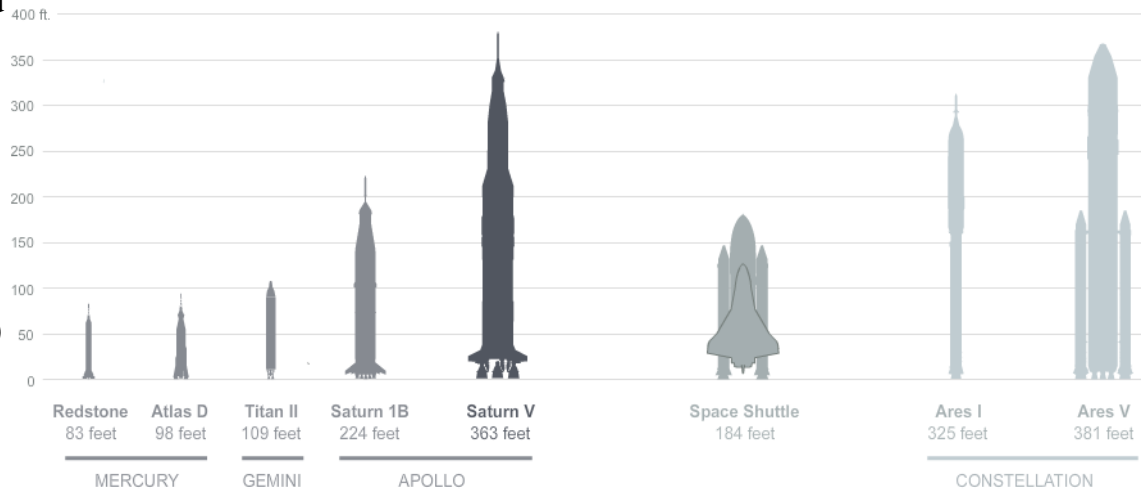
On Apollo 11, Michael Collins had to sit by himself circling the Moon in the command module while his two companions went to the surface in the lander. For the next Moon missions, all four astronauts are to head to the surface, while the Orion capsule, empty, takes care of itself.

That means the Altair lander must be much larger than the Apollo-era lander, both to carry the additional astronauts and supplies and to be able to reach more parts of the Moon. The advances in technology could also

enable cargo versions of the Altair - without astronauts - to bring modular components of an outpost as well as R.V.-size rovers.

The rover concept calls for a fully pressurized cabin in which the astronauts can work in short sleeves. For sorties lasting a week or so, the astronauts would be essentially living out of their car. “Call it a ‘Luna-bago’ of sorts!” Dr. Olson said The spacesuits would actually be stored outside the rover, and the astronauts would be able to jump into them via openings in the back, enabling them to go from inside to outside in 10 minutes.

“It’s a total game changer,” Dr. Olson said.



Relative size of the Apollo Program's Saturn V rocket compared with previous and planned manned vehicles.

But the federal budget proposed by President Obama would not pay for that, certainly not before 2020. After increases in the current year and for fiscal year 2010, Mr. Obama’s proposed spending on human exploration in years 2011 through 2013 was several billion dollars less than what President Bush proposed last year. That essentially cut the money to turn the Altair and the Ares V from paper concepts to detailed designs and real spacecraft.

“No bucks, no Buck Rogers,” Dr. Olson said.

But the hope of many inside and outside NASA is that the Obama administration’s budget levels are just placeholders pending the recommendations of the panel reviewing the agency’s human space program. Its report is expected by the end of August.

The panel is looking at alternatives to Ares I and Ares V, like adapting existing rockets like the Delta IV for NASA’s astronaut needs.

Consultation on 'UK space agency'

By Jonathan Amos Science reporter, BBC News

There is to be a 12-week consultation on whether the UK should have its own dedicated space agency. The Science Minister Lord Drayson is supportive of the idea but wants to hear the views of academics and industrialists. Currently, British space policy is devised by a "partnership" of government departments and research councils operating devolved budgets. The minister says Britain would benefit from a more strategic approach.

"Both in terms of raising the profile of space, which is a fantastic asset in the UK, and in terms of organising ourselves more efficiently, I think that an agency is the way to go," he told the BBC.

Different structure

The House of Commons Select Committee on Science and Technology looked at the issue in 2007 and concluded marginally that an agency was only worth setting up if the UK increased its civil spend on space substantially. At the moment, the government invests some £250m a year, mostly channelled through the European Space Agency (Esa). But Lord Drayson said the benefits were strong even if no extra funds were forthcoming, and urged people to set aside the budgetary issue for the time being.

Whereas Germany, France and Italy have national space agencies that speak with single voices backed up by single budgets, the UK's approach is to devolve space policy decisions to a club of "users" facilitated by a civil service unit called the British National Space Centre (BNSC).

These users are the government departments and research councils that have interests in space science or space-borne services. The arrangement is supposed to ensure that limited space funding chases "need" and "value".

But critics say the inability of this club sometimes to adopt coherent positions on complex programmes means that UK delegations often find themselves marginalised when they go into international negotiations.

Necessary 'clout'

Lord Drayson cited the example of the European GMES (Global Monitoring for Environment and Security) programme. This is a joint endeavour of the European Space Agency (Esa) and the European Commission which seeks to build a coordinated system for Earth observation and monitoring.

Many politicians agreed it was the perfect project for the UK because of its vocal position on climate change.

But Britain went in late to the multi-billion-euro venture and only caught the second opportunity after some last-minute funding was organised by the Treasury. Industry has complained that the confusion over GMES cost UK companies the chance to bid for satellite contracts.

Phil Willis MP, the Liberal Democrat chair of the HoC Science and Technology Committee, said the consultation was "excellent news".

"The principal of a space agency is one which we as a committee supported to give a central focus to space exploration, and particularly the UK's eminent position in terms of robotics," he said.

"My personal view is that it is still worth having [without a budgetary increase], but quite frankly without very significant additional funds, what you have is an organisation in name with very little clout."

Technical centre

This is an important week for UK space. On Wednesday, the European Space Agency will formally open its new technical centre at Harwell, Oxfordshire. The UK is the only major Esa contributor not to have such a showcase facility.

The British government last month also initiated a panel to review space activity in the country.

The Space Innovation and Growth Team (IGT) will attempt to identify key trends and then list the actions industry and government need to take if they want to fully exploit the changes that are coming over the next 20 years.

Lord Drayson already has a third report on his desk that looks at the role Britain could play in the future exploration of the Solar System given its current areas of expertise.

Esa accepted helicopter test pilot "Major Tim" Peake into its astronaut corps in May. Major Tim is the first Briton to make the corps.