Tumor virus is best predictor of throat cancer survival

COLUMBUS, Ohio – The presence of human papilloma virus, the virus that causes cervical cancer, in tumors is the most important predictor of survival for people diagnosed with oropharyngeal cancer (cancer of the back of the mouth), according to a new study led by a researcher at the Ohio State University Comprehensive Cancer Center-Arthur G. James Cancer Hospital and Richard J. Solove Research Institute (OSUCCC-James). Published online June 7 in the New England Journal of Medicine with a related editorial, this is the first study large enough to show that the presence of human papilloma virus (HPV) in tumors accounts for better response to therapy, rather than other favorable factors that may be present, such as young age and small tumors.

The second leading predictor of survival is lifetime smoking history, followed by cancer stage.

The findings suggest that the HPV status of a patient's tumor and their smoking history may be used in the future, in addition to cancer stage, to determine the aggressiveness of a patient's therapy.

"Previous studies indicated a relationship exited between the presence or absence of HPV in oropharyngeal tumors and patient survival, but they couldn't determine if other favorable factors present in these patients were responsible for their better outcome," says study leader Dr. Maura Gillison, a medical oncologist and head and neck cancer specialist at the OSUCCC-James.

"These findings close the door on these questions and will allow the field to move forward with clinical trials designed to determine how we should use molecular and behavioral factors to personalize therapy for patients."

Gillison emphasized that there is insufficient data at this time to indicate how a specific patient's cancer therapy should be tailored based on these factors.

Gillison and her colleagues analyzed the tumors and outcomes of 323 patients with stage III or IV oropharyngeal cancer who were part of a Radiation Therapy Oncology Group clinical trial. Of these patients, 206 had HPV-positive tumors and 117 had HPV-negative tumors.

At three years after treatment, 82 percent of patients with HPV-positive tumors were still alive, compared with 57 percent of patients with HPV-negative tumors. Rates of cancer relapse at three years for the groups were 43 percent and 74 percent, respectively.

The investigators determined that HPV presence in tumors accounted for most of the difference in therapy response and survival between patients with HPV-positive and HPV-negative tumors, while factors such as younger age, white race, better energy level, absence of anemia and smaller tumors were responsible for only about 10 percent of the difference.

Smoking history emerged as the second most important independent predictor of survival and cancer relapse for patients with oropharyngeal cancer. The risk of cancer relapse or death increased by one percent for each additional pack year of tobacco smoking (one pack year is equivalent to smoking one pack a day for a year).

The investigators found that at three-years, about 93 percent of patients with HPV-positive tumors who were never or light- smokers were alive, as compared to about 70 percent of patients with HPV-positive tumors who were smokers and about 46 percent of patients with HPV-negative tumors who were smokers.

"The two risk factors that place an individual at risk for oropharyngeal cancer are also the most important factors determining patient survival. This is probably because these factors determine the genetic profile of these cancers and how they respond to treatment," Gillison says.

Gillison and her colleagues have since conducted a follow-up study to further investigate the influence of tobacco smoking on oropharyngeal cancer. She reported these findings June 7 at the 2010 annual meeting of American Society of Clinical Oncology.

Funding from the National Cancer Institute and the National Institute of Dental and Craniofacial Research supported this research.

Calvin Klein Cologne Attracts Wild Cats and Other Animals

By Jennifer Viegas

Designers often advertise that their perfume and cologne products drive sniffers wild. But I think even Calvin Klein himself might be surprised to learn that his cologne, Obsession for Men, attracts jaguars, pumas and other wildlife, according to the Wildlife Conservation Society.

The WCS has just admitted that its researchers have been using the popular cologne to draw animals in front of remote cameras set up in the wilderness. The cameras are triggered by an infra-red beam, permitting candid shots of animals as they come by to investigate.

One place where this technique is now being used is at the Maya Biosphere Reserve, one of the largest protected areas in Central America. Animal experts there are trying to estimate populations of elusive jaguars.

Pat Thomas, General Curator of the Wildlife Conservation Society's Bronx Zoo, came up with the unusual cologne-attractant technique. He settled on Calvin Klein Obsession for Men after discovering that the zoo's tigers, snow leopards and cheetahs were drawn to it more than any other commercially produced scent.

The big cats rubbed, sniffed, pawed, and otherwise thoroughly enjoyed the designer cologne.

"Calvin Klein Obsession for Men clearly passes the sniff test among the WCS Bronx Zoo's big cat population," said Thomas. "More importantly, this work is a great example of how The Wildlife Conservation Society's Living Institutions and Global Conservation Programs work together to save wildlife and wild places."

As predicted, the cologne is doing a great job at attracting jaguars to the hidden camera setup.

The images show individual jaguars lingering around a cloth treated with the cologne and repeatedly sniffing it. One pair of jaguars even shows some very rarely seen mating behavior, so the smell seems to turn these animals on.

"Jaguars are highly elusive creatures and for years WCS researchers struggled to develop more effective methods for estimating how many jaguars were in the forest, hidden amongst the ancient Maya temples," said Roan McNab, WCS Guatemala Country Director. "Now, due to the fact that jaguars love Obsession for Men, WCS field conservationists are getting more precise estimates of jaguar populations."

Based on the photos released by the WCS, the cologne also attracts pumas, ocelots, tapirs, peccaries and coatis.

To see candid camera video of the jaguars Download Jaguar&ObsessionInYaloch09

The Earth and moon formed later than previously thought

The Earth and Moon were created as the result of a giant collision between two planets the size of Mars and Venus. Until now it was thought to have happened when the solar system was 30 million years old or approx. 4,537 million years ago. But new research from the Niels Bohr Institute shows that the Earth and Moon must have formed much later – perhaps up to 150 million years after the formation of the solar system. The research results have been published in the scientific journal, Earth and Planetary Science Letters.

"We have determined the ages of the Earth and the Moon using tungsten isotopes, which can reveal whether the iron cores and their stone surfaces have been mixed together during the collision", explains Tais W. Dahl, who did the research as his thesis project in geophysics at the Niels Bohr Institute at the University of Copenhagen in collaboration with professor David J. Stevenson from the California Institute of Technology (Caltech).

Turbulent collisions

The planets in the solar system were created by collisions between small dwarf planets orbiting the newborn sun. In the collisions the small planets melted together and formed larger and larger planets. The Earth and Moon are the result of a gigantic collision between two planets the size of Mars and Venus. The two planets collided at a time when both had a core of metal (iron) and a surrounding mantle of silicates (rock). But when did it happen and how did it happen? The collision took place in less than 24 hours and the temperature of the Earth was so high (7000º C), that both rock and metal must have melted in the turbulent collision. But were the stone mass and iron mass also mixed together?

Until recently it was believed that the rock and iron mixed completely during the planet formation and so the conclusion was that the Moon was formed when the solar system was 30 million years old or approximately 4,537 million years ago. But new research shows something completely different.

Dating with radioactive elements

The age of the Earth and Moon can be dated by examining the presence of certain elements in the Earth's mantle. Hafnium-182 is a radioactive substance, which decays and is converted into the isotope tungsten-182. The two elements have markedly different chemical properties and while the tungsten isotopes prefer to bond with metal, hafnium prefers to bond to silicates, i.e. rock.

It takes 50-60 million years for all hafnium to decay and be converted into tungsten, and during the Moon forming collision nearly all the metal sank into the Earth's core. But did all the tungsten go into the core?

"We have studied to what degree metal and rock mix together during the planet forming collisions. Using dynamic model calculations of the turbulent mixing of the liquid rock and iron masses we have found that tungsten isotopes from the Earth's early formation remain in the rocky mantle", explains Tais W. Dahl, Niels Bohr Institute at the University of Copenhagen. The new studies imply that the moon forming collision occurred after all of the hafnium had decayed completely into tungsten.

"Our results show that metal core and rock are unable to emulsify in these collisions between planets that are greater than 10 kilometres in diameter and therefore that most of the Earth's iron core (80-99 %) did not remove tungsten from the rocky material in the mantle during formation", explains Tais W. Dahl.

The result of the research means that the Earth and the Moon must have been formed much later than previously thought – that is to say not 30 million years after the formation of the solar system 4,567 million years ago but perhaps up to 150 million years after the formation of the solar system.

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Why does feeling low hurt?

Depressed mood increases the perception of pain

When it comes to pain, the two competing schools of thought are that it's either "all in your head" or "all in your body". A new study led by University of Oxford researchers indicates that, instead, pain is an amalgam of the two. Depression and pain often co-occur, but the underlying mechanistic reasons for this have largely been unknown. To examine the interaction between depression and pain, Dr. Chantal Berna and colleagues used brain imaging to see how healthy volunteers responded to pain while feeling low.

Their findings revealed that inducing depressed mood disrupted a portion of the participants' neurocircuitry that regulates emotion, causing an enhanced perception of pain. In other words, as explained by Dr. Berna, "when the healthy people were made sad by negative thoughts and depressing music, we found that their brains processed pain more emotionally, which lead to them finding the pain more unpleasant."

The authors speculate that being in a sad state of mind and feeling low disables one's ability to regulate the negative emotion associated with feeling pain. Pain, then, has a greater impact. Rather than merely being a consequence of having pain, depressed mood may drive pain and cause it to feel worse.

"Our research suggests depressed mood leads to maladaptive changes in brain function associated with pain, and that depressed mood itself could be a target for treatment by medicines or psychotherapy in this context," commented Dr. Berna. Thus, the next step in this line of research will be to examine this mechanism in individuals who suffer from chronic pain, as these individuals also commonly experience depression. The ultimate goal, of course, is to develop more effective treatments. This is good news for the millions of individuals around the world who suffer from chronic pain and depression.

Notes to Editors *The article is "Induction of Depressed Mood Disrupts Emotion Regulation Neurocircuitry and Enhances Pain Unpleasantness" by Chantal Berna, Siri Leknes, Emily A. Holmes, Robert R. Edwards, Guy M. Goodwin, and Irene Tracey. Berna, Leknes, and Tracey are affiliated with The Centre for Functional Magnetic Resonance Imaging of the Brain, Department of Clinical Neurology and Nuffield Department of Anaesthetics, University of Oxford, Oxford, United Kingdom. Berna, Holmes, and Goodwin are from the Department of Psychiatry, University of Oxford, Warneford Hospital, Oxford, United Kingdom. Edwards is affiliated with the Department of Anesthesiology, Harvard Medical School, Brigham & Women's Hospital, Chestnut Hill, Massachusetts. The article appeared in Biological Psychiatry, Volume 67, Issue 11 (June 1, 2010), published by Elsevier.*

Gut bacteria may contribute to autism

* 13:56 07 June 2010 **by Linda Geddes**

Children with autism appear to have a characteristic chemical signature in their urine which might form the basis of an early diagnostic test for the condition.

The finding also adds weight the hypothesis that substances released by gut bacteria are contributing to the onset of the condition.

Autism has previously been linked to metabolic abnormalities and gastrointestinal problems such as gut pain and diarrhoea. Several studies have also hinted at changes in gut bacteria in the faeces of children with autism.

To investigate whether signs of these metabolic changes might be detectable in children's urine, Jeremy Nicholson and colleagues at Imperial College London investigated 39 children with autism, 28 of their non-autistic siblings and 34 unrelated children.

Chemical fingerprint

Using nuclear magnetic resonance (NMR) spectroscopy to analyse the children's urine, they found that each of these groups had a distinct chemical fingerprint, with clear and significant differences between children with autism and unrelated controls.

"The signature that comes up is related to gut bacteria," says Nicholson. It is not yet clear whether the bacteria's metabolic products contribute to the development of autism, but it is a possibility worth investigating, he adds. A large proportion of autistic children have severe gastrointestinal problems that tend to appear at about the same time as the behavioural symptoms.

"It adds another link to the gut bacterial involvement in the onset of disorder," says Glenn Gibson of the University of Reading, UK, who has previously identified abnormally high levels of clostridium bacteria in children with autism.

One possibility is that the gut bacteria in children with autism are producing toxins that might interfere with brain development. One of the compounds identified in the urine of autistic children was N-methyl-nicotinamide (NMND), which has also been implicated in Parkinson's disease.

Inducing autism

Meanwhile, Derrick MacFabe of the University of Western Ontario in London, Canada, and his colleagues have found that short-chain fatty acids produced by clostridium bacteria can induce reversible autism-like behavioural and biochemical changes in rats.

"Nicholson's study did find some biomarkers of gut clostridial populations that we think contribute to autistic symptoms," says MacFabe, who presented his findings at a meeting of the International Society for Autism Research in Philadelphia, Pennsylvania, last month. Nicholson emphasises that further research is needed to confirm whether bacteria really are contributing to autism, and if so, how. He also stresses that his findings in no way support claims of a link between vaccines and autism.

Even if bacteria are not actually contributing to the observed metabolic changes, they could still be put to use. "There is probably the basis of a test for autism based on a urinary metabolic profile," says Nicholson.

Early treatment

Autism is currently diagnosed using a series of behavioural tests, and while children can show symptoms of the condition when as young as 5 months old, a clear diagnosis is not usually possible until they are age 2 or 3 years. This is problematic, because there is growing evidence that the earlier behavioural therapies for autism are started, the better the chances of children being able to lead relatively normal lives.

"If you could identify kids who were at risk much earlier by a chemical test rather than by observing the manifestation of full-blown behaviour, we could get them into therapy much earlier," says Nicholson.

The next step is to confirm the results in a much larger group of age-matched children, as well as following high-risk children from birth in order to identify whether there are markers that precede the development of autistic symptoms. *Journal reference: Journal of Proteome Research, DOI:10.1021/pr901188e*

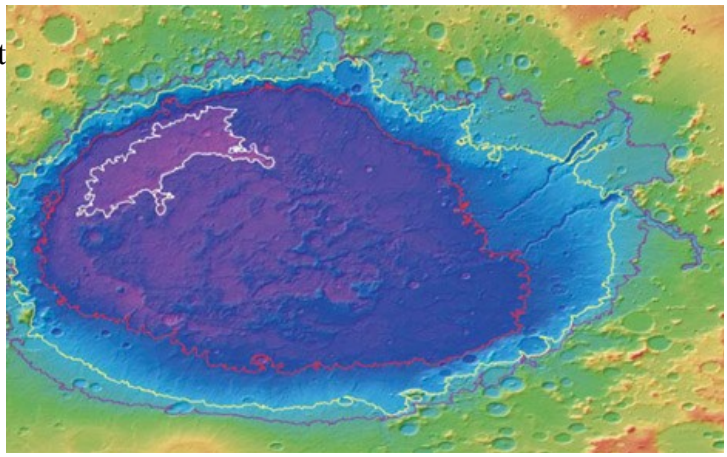
Huge seas 'once existed on Mars'

US scientists have found further evidence that huge seas existed long ago on Mars.

A geological mapping project found sedimentary deposits in a region called Hellas Planitia which suggest a large sea once stood there.

The 2,000 km-wide, 8km-deep Hellas basin is a giant impact crater - the largest such structure on Mars. The researchers say their data support a lake between 4.5 and 3.5 billion years ago. Some scientists believe that conditions on Mars were more favourable for the evolution of life at this time than they were on Earth.

"This mapping makes geologic interpretations consistent with previous studies, and constrains the timing of these putative lakes to the early-middle Noachian period on Mars," said Dr Leslie Bleamaster, research scientist at the Planetary Science Institute in Tucson.



Hellas basin (Nasa) The Hellas basin is the largest impact structure on Mars

The researchers say that fine-layered outcrops around the eastern rim of Hellas are likely to be sedimentary deposits. They were formed through the erosion and transport of rock and soil from the Martian highlands into a standing body of water. The results support earlier studies of the western part of the Hellas basin.

Further study of the region could provide clues about where this water went and to how the Martian climate changed over geological history. The mapping effort used data from a number of instruments aboard Nasa spacecraft, including the Viking orbiter, Mars Global Surveyor and Mars Odyssey.

Drug that helps metastatic colon cancer of no benefit in less advanced tumors

Mayo Clinic researchers say results are unexpected and puzzling

CHICAGO - To the surprise of researchers at Mayo Clinic who led a national clinical trial, a targeted therapy that provides benefit to patients with metastatic colon cancer has failed to help patients with less advanced, stage III cancer. In fact, patients who used the agent, cetuximab, with chemotherapy had outcomes slightly inferior to patients treated with chemotherapy alone.

The phase III North Central Cancer Treatment Group (http://ncctg.mayo.edu/) (NCCTG) study, sponsored by the National Cancer Institute (http://www.nci.nih.gov/) (NCI), was closed to patient accrual in November 2009 after a planned interim analysis demonstrating a lack of benefit from the addition of cetuximab to standard chemotherapy. The first public discussion of the study and results presented in a press conference June 6 at the annual meeting of the American Society of Clinical Oncology (http://www.asco.org/) (ASCO).

In theory, many of the patients enrolled on this trial should have benefited from the addition of cetuximab to chemotherapy, says Steven Alberts, M.D., the Mayo Clinic oncologist (http://www.mayoclinic.org/oncology/) who led the clinical trial. All of the patients enrolled in the trial had colon cancers that had spread to the nearby lymph nodes (stage III), but not beyond. To be eligible for the trial, patients first had to have the cancer

completely removed with surgery. These patients also had a normal (wild-type) KRAS gene in their tumor, which previously has been shown necessary to have the potential for cetuximab to work.

"The sum of data to date from trials for metastatic colorectal cancer suggested that cetuximab would provide benefit in these stage III patients with KRAS wild-type tumors, and so our findings are unexpected," Dr. Alberts says. "It is difficult to understand how an agent that helps patients with metastatic cancer is not beneficial to those with less advanced disease. At this point we are focusing our efforts on identifying a biological explanation for these findings."

With more than 3,000 patients enrolled, the study was one of the largest clinical trials yet performed testing cetuximab with the commonly-used FOLFOX chemotherapy following surgery in patients with colon cancer. It was expected that adding cetuximab to FOLFOX would provide benefit in stage III colon cancer patients, and would lead to Food and Drug Administration (FDA)-approval for that indication, Dr. Alberts says. Currently, the FDA has only approved cetuximab for metastatic colon cancer and the drug should continue to be used in that setting as clinically appropriate, he says.

"Based on what we found, any use of cetuximab in stage III colon cancer is not supported by the results of our trial," he says. According to Dr. Alberts, this is the only U.S. study to have looked at the use of chemotherapy and cetuximab in phase III colon cancer patients, but a European clinical trial is ongoing and first results are expected next year.

The randomized clinical trial had multiple arms, but the most important were the two that tested FOLFOX with or without cetuximab. This particular analysis looked only at the 1,864 enrolled patients with a normal KRAS gene (909 treated with chemotherapy alone and 955 patients treated with both drugs).

Based on a statistical analysis of the outcomes at the three-year mark post-treatment, the researchers found that those treated with chemotherapy alone (without cetuximab) had a 76 percent disease-free survival (alive and with no disease recurrence), compared to 72 percent in patients who used both therapies. Overall survival in all patients to date is also better in patients who did not receive cetuximab. However, as this study was stopped early after a median follow-up of approximately two years, "Follow-up in regard to survival is short at this point," Dr. Alberts says.

The researchers also concluded that while there were no differences in toxicity between treatment groups in patients younger than 70, there was increased toxicity as well as greater differences in outcomes in patients aged 70 and older. The researchers have theories as to why stage III colon cancer patients did not benefit from cetuximab, but no evidence yet. Two favored ideas are that cetuximab could be switching on, or increasing activity, in molecular pathways that promote cellular growth, or could be creating resistance to the effects of chemotherapy.

"The most critical question from this trial is why a difference exists between patients with stage III disease, where cetuximab is not of benefit, and patients with metastatic cancer, where cetuximab does provide benefit," Dr. Alberts says. "We aim to find out."

In addition to a grant from NCI, the study received support from Bristol-Myers Squibb, ImClone Systems, Sanofi-Aventis, and Pfizer. The researchers declare no other potential conflict of interest.

Simple eye test measures damage from multiple sclerosis, UT Southwestern researchers find

DALLAS - A quick, painless eye measurement shows promise as a way to diagnose multiple sclerosis in its very early stages, and to track the effectiveness of treatments, researchers from UT Southwestern Medical Center have found in a multicenter study.

"This technique has the potential to provide a powerful and reliable assessment strategy to measure structural changes in the central nervous system, both for diagnostic purposes and in clinical trials to monitor whether potential treatments can prevent deterioration or restore nerve function," said Dr. Elliot Frohman, professor of neurology and ophthalmology, director of the Multiple Sclerosis Clinical Center at UT Southwestern and co-senior author of the study, which appears in the June issue of Annals of Neurology.

The technique, called optical coherence tomography (OCT), reliably measures thinning of the retina in people with multiple sclerosis, the researchers found.

"An ophthalmologist might someday be able to use OCT to identify retinal thinning during a routine eye exam and consider MS as a prime diagnosis," Dr. Frohman said. "However, this prospect is a long way off."

The retina, which lines the back of the eye, detects light and sends visual information to the brain via the optic nerve. Retinal thinning can occur as a result of multiple sclerosis, but this study, Dr. Frohman said, is the first to track such thinning over time in a single group of patients. The Neurology study involved 299 patients with MS who were tracked for six months to 4.5 years.

The researchers found that the retinas thinned significantly with time, and patients often concurrently lost visual sharpness. Overall, the study indicated that OCT is reliable, easy to use and sensitive to changes over time. It could also be used with current clinical measures, the researchers said.

Because the retina is easily visible through the pupil, it provides a convenient route for assessing nerve damage, compared with other parts of the body. As a result, retinal measurement might be able to pick up signs of multiple sclerosis before a person develops other symptoms, Dr. Frohman said.

OCT machines already are available. Patients look into a device similar to those that measure vision for corrective lenses. Near-infrared light, which is invisible to the eye, penetrates the retina and provides information on its thickness. The measurement takes a few seconds for each eye.

In addition to the OCT testing, patients in the latest study looked at eye charts so the researchers could test their vision. Control subjects came from the patients' families and clinics' staff.

Future studies are needed to ascertain whether OCT can characterize the effectiveness of treatments, Dr. Frohman said.

Other UT Southwestern researchers in neurology involved in the study were Gina Remington, clinical research coordinator; Amy Conger, neuro-ophthalmic imaging specialist; and Teresa Frohman, clinic research manager.

The research was a joint project with the University of Pennsylvania School of Medicine and Johns Hopkins University School of Medicine. Researchers from the University of Alabama, Birmingham, also participated.

The study was funded by the National Multiple Sclerosis Society, the National Institutes of Health, DAD's Foundation and the McNeill Foundation.

New evidence for a neuronal link between insulin-related diseases and schizophrenia

By Katherine Harmon

diabetes psychiatric schizophrenia insulin dopamineWhen the body does not properly manage insulin levels, diabetes and other metabolic disorders are familiar outcomes. That hormonal imbalance, however, has also been linked to a higher risk for psychiatric disorders, such as schizophrenia. And a new study has uncovered a potential pathway by which this metabolic hormone can upset the balance of a key neurotransmitter.

"We know that people with diabetes have an increased incidence of mood and other psychiatric disorders," Kevin Niswender, an endocrinologist at Vanderbilt University Medical Center and coauthor of the study, said in a prepared statement. Previous researchers, including Aurelio Galli, a neurobiologist at Vanderbilt, had found that insulin was affecting more than blood sugar levels.

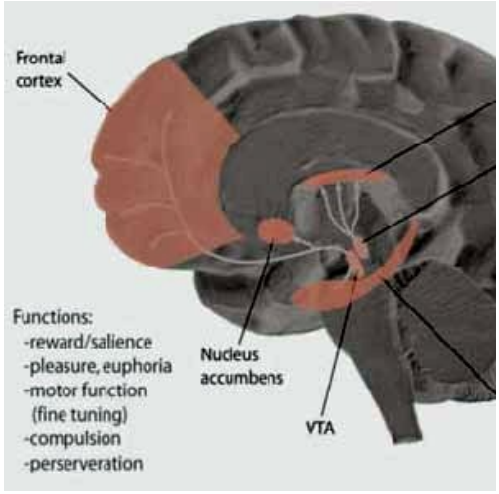


***Image of dopamine pathways in the brain courtesy of* Wikimedia Commons/NIDA**

"Something goes wrong in the brain because insulin isn't signaling the way that it normally does," Galli, a coauthor of the new paper, published online June 8 in the journal PLoS Biology, said in a prepared statement. Although schizophrenia is a complex disease that is thought to have a variety of individual genetic and epigenetic causes, these researchers and others have proposed that a common thread is too little dopamine, a neurotransmitter that is involved in movement, reward and motivation.

But just how, molecularly, insulin and dopamine dysfunctions might be linked has yet to be settled.

In the new study, the researchers focused on the protein kinase Akt, which plays a role in cell signaling and has been linked to schizophrenia as well as to diabetes and obesity. It is controlled by hormones, neurotransmitter receptors and growth factors. To study Akt's possible role in schizophrenia and its relation to insulin dysregulation, the researchers created a line of transgenic mice that had poor Akt signaling, leading to schizophrenia-like behaviors.

The researchers also found that the modified mice had increased transportation of another neurotransmitter norepinephrine (which also acts as a stress hormone) and a deficit of dopamine in their brains, a condition known as hypodopaminergia.

"We believe the excess [norepinephrine transporters are] sucking away all of the dopamine and converting it to norepinephrine, creating this situation of hypodopaminergia in the cortex," Galli said.

And when the researchers inhibited the norepinephrine transporter in these Akt knock-out mice, "we could reverse the cortical hypodopaminergia and behavioral deficits," they noted in the study.

"Taken together, this work supports the potential for targeting both Akt and the norepinephrine transporter for treating dopamine-related mood disorders," the researchers concluded. They also point out clinical trials are already testing norepinephrine transporter blockers for their effectiveness in restoring dopamine balance in schizophrenics.

The molecular pathway might also shed some light on behavioral issues associated with diabetes and other insulin-related metabolic disorders. "We thought that those co-morbidities might explain why some patients have trouble taking care of their diabetes," Niswender said.

Study links long sleep duration to an increased risk of metabolic syndrome in older adults

With more than 29,000 participants, the study is the largest investigation of sleep and metabolic syndrome

WESTCHESTER, IL – Long sleep duration is associated with an elevated prevalence of metabolic syndrome in older adults, according to a research abstract that will be presented Tuesday, June 8, 2010, in San Antonio, Texas, at SLEEP 2010, the 24th annual meeting of the Associated Professional Sleep Societies LLC.

Results indicate that participants who reported a habitual daily sleep duration of eight hours or more including naps were 15 percent more likely to have metabolic syndrome (odds ratio = 1.15). This relationship remained unchanged after full adjustment for potential confounders such as demographics, lifestyle and sleep habits, and metabolic markers. Removing participants with potential ill health from the analysis slightly attenuated the observed association (OR = 1.13). Although participants who reported a short sleep duration of less than six hours were 14 percent more likely to have metabolic syndrome in the initial analysis(OR = 1.14), this association disappeared after controlling for potential confounders (OR = 0.98).

"The most surprising aspect of our study was that long sleep - and not short sleep - was related to the presence of the metabolic syndrome," said lead author Teresa Arora, research scientist at the University of Birmingham School of Medicine in Birmingham, U.K.

The study involved 29,310 people in Guangzhou, China, making it the largest study to assess the relationship between sleep duration and the presence of metabolic syndrome. Participants were 50 years of age or older. Total sleep duration was reported by questionnaire.

The authors cautioned that the cross-sectional nature of the study did not allow for a determination of causality. However, Arora pointed out that the secondary analysis of healthy elders makes the results particularly intriguing. "Long sleep duration is unlikely to be the consequence of ill health in our population sample as we re-ran the analysis in a smaller subset of 'healthy' elders, and the findings remained," she said. "Our follow-up data will allow us to make causal inferences."

Confirming that long sleep duration causes an increased risk of metabolic syndrome would have important public health implications, Arora added. "We can recommend that long sleepers reduce the amount of overall sleep they achieve, which may in turn have beneficial effects on their health," she said. "Programs can be developed to modify sleep in an attempt to reduce the health burden on elderly populations, who are already at higher risk of disease."

According to the National Heart, Lung, and Blood Institute, metabolic syndrome is a group of obesity-related risk factors that increases your risk of heart disease, diabetes and stroke. A person with at least three of these five risk factors is considered to have metabolic syndrome: excess abdominal fat, high triglycerides, low HDL cholesterol, high blood pressure and high blood sugar.

A U.K. study published in the February issue of the journal Sleep suggested that healthy older adults without sleep disorders can expect to have a reduced "sleep need" and to be less sleepy during the day than healthy young adults. In the May issue of Sleep a study of 15,638 older Chinese adults reported that their weighted average daily sleep time was about 7.5 hours including naps.

The SLEEP 2010 abstract supplement is available for download on the website of the journal Sleep at http://www.journalsleep.org/ViewAbstractSupplement.aspx.

The Claim: Keep Stitches Dry and Covered Up After Surgery

By ANAHAD O'CONNOR

THE FACTS The instructions for caring for fresh stitches are fairly universal: keep the sutures clean and dry and avoid getting them wet for at least 48 hours. Doing so, the thinking goes, sharply reduces the rate of infection and improves healing. But when temperatures and humidity are high, following doctor's orders can be a nuisance.

Studies have indicated that the 48-hour rule makes little difference. Washing a sutured wound with soap and water - or simply getting it wet, period - in the first 12 hours did not pose a problem. But most of the studies have been small and unpersuasive. So in 2006, Australian researchers carried out a large, randomized study involving more than 800 patients undergoing minor surgery at several clinical centers. Some were assigned to keep their stitched wounds dry and covered for at least 48 hours (the control group), and others were told to keep the dressing on to absorb immediate bleeding, then remove it and bathe as normal in the first 12 hours. None of the patients were on antibiotics.

Ultimately, the study, published in the British medical journal BMJ, found no major difference in outcome. The infection rates were 8.9 percent in the control group and 8.4 percent in the "wet" group, indicating "that patients can uncover and occasionally wet stitches in the first 48 hours after minor skin excisions without increasing the incidence of wound infection."

THE BOTTOM LINE Research suggests that after minor surgery, a sutured wound is not more likely to become infected if it gets wet.

Under 50? Silent duo could put you at risk for a big stroke

Silent or covert strokes in young adults with first-ever ischemic stroke are associated with recurrent stroke

Quebec City – Being young doesn't mean you are immune to a stroke. You may feel healthy; you may be 18 or a vigorous 50. And yet you could be more vulnerable than you know. That could be because of the role played by silent risk factors in stroke. Now, as a result of research from Hopital Notre Dame in Montreal, two silent factors − leukoaraiosis and silent brain infarcts − are not so silent any more.

Lead investigator, neurologist Dr. Alexandre Poppe, suggests that patients aged 18 to 50 who present with stroke should have brain MRIs to identify those who have experienced silent strokes, in an effort to prevent further damage.

Silent brain infarcts (SBI) are tiny strokes which can be seen on brain imaging but are asymptomatic; the patient is completely unaware of their occurrence, but this does not mean they are not causing damage.

Research tells us that these conditions are common in older adults with acute ischemic stroke and predict recurrent stroke and cognitive decline. Their presence can help neurologists assess the risk of future stroke-related disease and emphasize prevention. Now, in a world-first study, Dr. Poppe has shifted the focus from elderly patients to a much younger, under-investigated age group: 18 to 50 year-olds.

Dr. Poppe and his co-investigators studied 168 stroke patients in this younger age group, all of whom underwent MRI after a first stroke. They were followed for an average of 27 months. Over that time, stroke recurred in 11 per cent.

Those with silent or covert strokes identified on their MRI were three times more likely to experience a recurrent stroke than patients without these covert lesions.

"This study tells us that when younger people come in with a first stroke, they may already have signs of pre-existing damage in their brains," says Dr. Poppe. "We should pay particular attention to those who do, because they are at higher risk of having a second stroke, and prevention efforts need to be greatly emphasized."

"All young people with stroke should be scanned preferably using MRI," he says. "Doing a CT scan alone is often insufficient to pick up the brain changes caused by covert brain infarcts; with an MRI you can actually tell how old the lesions are. You can see if they occurred before the stroke."

Next, Dr. Poppe plans to determine if the location, size, and number of silent lesions have an impact not only on recurrent stroke but also cognitive decline.

"While MRI is helpful in young patients who've already had a stroke, at this stage it is too early to recommend screening for all 18-50 year-olds with a family history of stroke," he says.

"Stroke in the young is under-appreciated," says Canadian Stroke Network spokesperson Dr. Antoine Hakim. "Ten per cent of stroke patients are under 50."

He notes that with risk factors in all age groups increasing, it's especially important for younger adults to manage their stroke risk factors.

"Younger people may be becoming more vulnerable to stroke because of the larger number of Canadians indulging in unhealthy eating and living habits," says Dr. Hakim. "This may be accelerating the impact of risk factors, especially high blood pressure, which are now converging and have the potential to erase the progress we've made in treating heart disease and stroke over the last 50 years."

The recent Heart and Stroke Foundation report card on Canadians' health noted that young people are beginning their adult lives with multiple risk factors for heart disease and stroke.

"Over the past 15 years, Canada has seen significant increases in overweight and obesity, and diabetes mellitus," says Heart and Stroke Foundation spokesperson Dr. Michael Hill. "There are more than 250,000 young Canadians in their 20s and 30s with high blood pressure – the number one risk factor for stroke."

Dr. Hill recommends that adults of all ages pay attention to stroke risk factors, including high blood pressure, diabetes, smoking, obesity, high blood cholesterol, physical inactivity, stress and excess alcohol consumption.

Canadians can find out their risk of heart disease and stroke by going to the Heart and Stroke Foundation's website (heartandstroke.ca) and taking the Heart&Stroke Risk AssessmentTM, a personalized risk profile and a customized action plan for healthy living that includes tips, tools, and recipes.

East African human ancestors lived in hot environments, says Caltech-led team

Geochemical findings could help explain facets of early human evolution, including the development of bipedalism

PASADENA, Calif. - East Africa's Turkana Basin has been a hot savanna region for at least the past 4 million years - including the period of time during which early hominids evolved in this area - says a team of researchers led by scientists at the California Institute of Technology (Caltech). These findings may shed light on the evolutionary pressures that led humans to walk upright, lose most of our body hair, develop a more slender physique, and sweat more copiously than other animals.

Their findings - which were based on measurements of the spatial distribution and concentrations of isotopes in carbonate ions - are being reported this week in the early online edition of the Proceedings of the National Academy of Sciences (PNAS).

"When you measure the temperature of the ground, you learn a lot about the environment above it," says John Eiler, Robert P. Sharp Professor of Geology and professor of geochemistry at Caltech. In fact, he says, soil temperature tells you not just about air temperature, but about whether there were trees and plants to shade the soil, keeping temperatures cooler during the hottest part of the day.

Today, northern Kenya - where the Turkana Basin is found - is among the warmest areas on earth. It has little canopy forest, leaving the ground exposed to sunlight. "The question is, was the ground here ever cooler than it is today?" asks Eiler. "And if it was, why? Was it because the air was cooler, or because of more forest shading?"

To find out, the team examined the spatial organization (or "clumping") of rare, naturally occurring isotopes of carbon and oxygen - specifically, carbon-13 and oxygen-18 - in the form of carbonate ions that are constituents of minerals found in buried soils from northern Kenya. The clumping of these isotopes, Eiler and his colleagues have demonstrated in previous papers, is dependent on temperature: Hot temperatures lead to less clumping; cold temperatures, more.

"These carbonates are a common constituent of these soils," Eiler explains. "If you have the ability to measure their isotopes, then you have a ground-temperature thermometer."

When the researchers applied that thermometer to various layers of buried soils from East Africa, they found what Eiler says was "such a straightforward answer, it wasn't obvious how we could talk ourselves out of the conclusion we reached."

That conclusion? "The Turkana Basin region - one of the key places where hominid fossils documenting human evolution are found - has been a really hot place for a really long time," says Benjamin Passey, formerly a postdoctoral scholar at Caltech. Passey, who led the work on this project, is now at Johns Hopkins University.

But why does it matter how hot Africa was millions of years ago? "This is the area where we find the occurrence of some of the earliest hominid species," notes Eiler. "It tells us that this environment, though harsh, was a place where our ancestors could thrive. It tells us that they were probably originally marginal species that lived in difficult-to-survive environments."

The findings also shed some light - and heat - on a longstanding debate over the origin of bipedalism in early humans.

"For a long time, anthropologists have hypothesized that bipedalism and other unique human traits would be advantageous to life in hot savanna environments," says Passey. "For example, by standing upright, we intercept less direct sunlight than if we were on all fours, and in hot, open environments, the ground and near-surface air can be appreciably hotter than the air a few feet above the ground. So, by standing upright, we are avoiding a high-temperature environment."

Of course, Passey adds, this strategy would only be of significant use if the environment in question is indeed a high-temperature one. "In cooler environments, these traits do not really have a thermal advantage," he notes. These considerations led to the team's interest in figuring out just how hot it was in the part of the world where bipedalism is most likely to have first gained a toehold.

Eiler cautions that the team's findings are simply assessments of the area's temperature over time, and have nothing to say about "the importance of ambient temperature in shaping human evolution." But, he notes, they are "consistent with the notion that the heat in the area would have been a selective pressure that could have made bipedalism advantageous."

In addition to Eiler and Passey, the coauthors on the PNAS paper, "High-temperature environments of human evolution in East Africa based on bond ordering in paleosol carbonates," were former Caltech postdoc Naomi Levin, now at Johns Hopkins University; and Thure Cerling and Francis Brown from the University of Utah. Their work was supported by grants from the Camille & Henry Dreyfus Foundation and the National Science Foundation.

Why Humans Have No Fur - Explained

The cradle of human evolution in East Africa has been scorching hot for a long time, favoring fur-free, upright humans, new research finds.

By Emily Sohn

Heat might explain why we lost our fur and now strike an upright and slim (in theory, anyway) pose.

If our ancestors lived somewhere really hot, the theory goes, it would have made sense for us to lose body hair, start sweating more, become slender and even walk upright -- to create distance between our bodies and the hot ground.

A new study supports the theory that heat helped drive human evolution, by showing that a key cradle of human evolution in East Africa has indeed been really hot for at least 4 million years.

"That's something that's been hard to get at," said Ben Passey, a geochemist at Johns Hopkins University in Baltimore. "It's nice to say that these things would be advantageous to living in hot, open environments. But was it actually hot and open?"

To find out, Passey and colleagues analyzed dated soil samples from the Turkana Basin, a well-studied region in Kenya and Ethiopia that contains lots of fossils from our human and pre-human ancestors. In particular, they looked at weighted carbon and oxygen atoms, called isotopes.

As temperatures drop, a rare isotope of carbon called carbon-13 tends to clump together with a rare isotope of oxygen called oxygen-18 within soil. Through a fairly simple relationship, the more clumping the scientists see between these isotopes, the colder they are able to say a sample is.

Their results, published in the Proceedings of the National Academy of Sciences, showed that dirt in the Turkana Basin has remained above about 85 degrees Fahrenheit with spikes above 95 degrees F over the past 4 million years. Since soil absorbs heat from the air, that means that the region has been really hot for a really long time.

Today, air temperatures in the Basin regularly exceed 100 degrees Fahrenheit, Passey said, with nighttime temperatures in the 70s. Temperatures average in the mid-80s all year-round. The landscape is sparse with grasses, shrubs and bushes.

"The heat is sort of unrelenting," he said. "You think, God, it could not have been as hot when humans were evolving here. It must have been much a nicer, lush place. Our results say no, it was still hot."

Because the researchers could look only at soil temperature, Passey added, it's possible that the air was even warmer millions of years ago than it is today, but with more vegetation and more shade that could have cooled the soil a little bit.

"There is no question that the results are fascinating," said Harvard anthropologist Daniel Lieberman, who studies how and why the human body looks the way it does.

For one thing, at some point we developed a unique ability to regulate our body temperatures while running, which might have helped people catch prey in hot and dry conditions. "The hotter it is, the more humans have an advantage over other mammals, especially when running."

"No one knows for sure when we became proficient at sweating and when we lost our fur," he added. "But this paper provides strong evidence that the climatic conditions that would have favored such adaptations intensely were present for a long time."

Occupational Hazard: Playing the Fool

By MICHAEL W. KAHN, M.D.

Micheal Sloan

“Can you tolerate being bamboozled by your patients from time to time?”

I posed that question to a group of first-year psychiatric residents during a discussion of the perils of prescribing “feel good” medicines - anti-anxiety drugs like Xanax, in this case - to patients who might be lying to obtain them.

Xanax is one of a class of drugs known colloquially as “benzos” (benzodiazepines) that are safe and highly effective - but can also be addictive and have potential for abuse. The residents, in full “do no harm” mode, were focused on not fostering anyone’s drug habit.

“Everyone in the emergency room lies to get benzos,” one trainee remarked, as I recall.

Another said, “I’d give them one pill” to last until they could call their primary care provider, and a third said, “They need psychotherapy rather than medication.”

In short, the prevailing attitude was one of “they’ll have to pry that pill from my cold dead hands.” It made me wonder whether these budding psychiatrists might be working too hard to avoid being hoodwinked.

I think we underemphasize the prevalence of certain normal errors inherent in medical practice. Surgeons are fooled when they open an acutely painful abdomen only to find a normal appendix: in the days before CT

scans, it was said that if that didn't happen once in a while, you weren't operating often enough. When in doubt, it was safer (and wiser) to operate than to risk a rupture and peritonitis, even if the diagnosis was "wrong." Here was an error that wasn't an error, but rather a predictable side effect of balancing known risks with imperfect information.

I suggest that we apply a similar principle to the prescribing of narcotic painkillers and anti-anxiety drugs. Let's assume that it's impossible not to be fooled at least some of the time - that when assessing patients' sincerity, we should expect a certain rate of false positives.

Thus, when confronting patients who demand Xanax or morphine, doctors should worry less about defending their self-esteem and their lie-detection skills (after all, the most talented sociopaths are the ones most skilled at convincing others of their honesty) and more about what treatment is best for the patient.

Doing so doesn't exempt the physician from exercising proper skepticism and clinical shrewdness. I still vividly remember being fooled by a patient who couldn't have seemed more earnest about his need for narcotics to treat the pain from an accident that we were finally able to determine had never happened.

But even in that case, the cost was modest: two days of unnecessary narcotics for the patient and a survivable blow to my own estimation of my clinical acumen.

"It is better to suffer wrong than to do it," Samuel Johnson wrote, "and happier to be sometimes cheated than not to trust." Knowing that false positives are inevitable gives a statistical perspective to that wisdom - and frees the doctor from having to interrogate the patient like a criminal suspect.

To put it another way, I'd rather be taken for a sucker once in a while than know that my suspicion had denied someone legitimate help.

Furthermore, excessive suspicion compromises empathy and compassion. It is draining to approach patients as possible adversaries who must be bested.

The truth of Johnson's comment was recently brought home to me by the serene calm of a resident who was treating a particularly complicated narcotic-dependent patient. "I just give her what she asks for," the resident said. "She's been on narcotics for years, and if I give her what she wants she doesn't demand more. And then we can get on with her care."

Was this resident a naïve wishful thinker caving in to a demanding substance abuser rather than confronting her dependency? Maybe - but it didn't really seem so, and when we all met the patient together it was clear that both she and her medical team had bigger things to worry about - among them her pancreatitis, kidney failure and unstable diabetes.

Since "first, do no harm" remains a guiding principle of care, let's remember that the harm of missing a chance to help often greatly exceeds the harm of prescribing under a false pretext. Our system of justice is based on the idea that we should let the guilty go free rather than punish the innocent. Could our prescribing habits benefit from the same philosophy? ***Dr. Michael W. Kahn is a psychiatrist in Boston.***

Archaeologists given the rune around

A new study of rune stones from Viking times shows that many of the carvings are meaningless

After studying about a thousand inscriptions on ancient rune stones scattered around Scandinavia, a researcher from Uppsala University in Sweden has come to the conclusion that many of the carvings are gibberish. The researcher claims that the Vikings who carved them couldn't write and the people who saw them couldn't read.

"What was important was showing that you could write," explained researcher Marco Bianchi, who is an expert in Nordic languages. "What you wrote wasn't so important since no-one could read it anyway."

Most of the rune stones examined by Bianchi are found in the remote north of Sweden, but he has also discovered rune stones with meaningless signs that look like writing in Denmark.

The runic alphabets are a set of related alphabets using letters known as runes to write various Germanic languages prior to the adoption of the Latin alphabet. Most of the rune stones in Scandinavia were carved in the late Viking period around the year 1,000 AD.

Snakes in mystery global decline

By Richard Black Environment correspondent, BBC News

Snakes may be declining across the world, according to a global study.

Researchers examined records for 17 snake populations covering eight species over the last few decades, and found most had declined markedly. For reasons that are not entirely clear, some populations shrank in number abruptly around 1998.

Writing in the journal Biology Letters, the researchers describe the findings as "alarming" but say much more work is needed to understand the causes.

"This is the first time that data has been analysed in this way, and what we've shown is that in different parts of the world we seem to have this steep decline in a short period," said project leader Chris Reading.
"It surprised us when we realised what we were looking at," he told BBC News. "And we don't have a clue what it was about that period of time (around 1998)."

Dr Reading's team at the UK's Centre for Ecology and Hydrology ran the study with institutions in Australia, France, Italy and Nigeria.

Data deficiencies

The main problem for anyone wanting to conduct a global survey such as this is simply lack of data. Monitoring snake populations means marking the individuals in some way - typically by snipping a pattern into their scales, or implanting a microchip.

Field seasons can last for many months, and have to be repeated annually.

The researchers believe they amassed most, if not all, long-term datasets for this study - although "long-term" in this context means going back more than one decade, in some cases more than two.

Nevertheless, within this relatively short timeframe, eight of the 17 populations were seen to fall markedly in size - some by more than 90% - with only one showing any sign of a rise.

Species in decline include the asp and the smooth snake from Europe, the Gabon viper and rhinoceros viper of West Africa, and the royal python.

Populations shrank even in protected areas, suggesting that the progressive loss of habitat for wild animals being seen all over the world is not the only cause. Similar steep declines observed in frogs and newts in an earlier period were eventually found to be caused by the fungal disease chytridiomycosis.
The year when many of the snake declines began - 1998 - raises the question of whether climatic factors might be involved, as very strong El Nino conditions contributed to making it the hottest year recorded in modern times. Dr Reading's research group suggests many causes might be involved, and is appealing to other researchers to come forward with any more long-term datasets that might broaden the picture.

"The purpose of this paper was to say 'this is what we've found', and to say to other herpetologists 'now go and look at your own data'," he said.

"But I think that with so many populations in different places showing decline, it's more than co-incidence."

Healthy diet could slow or reverse early effects of Alzheimer's disease

Patients in the early to moderate stages of Alzheimer's Disease could have their cognitive impairment slowed or even reversed by switching to a healthier diet, according to researchers at Temple University.
In a previous study [http://www.temple.edu/newsroom/2009_2010/12/stories/alzheimers.htm], researchers led by Domenico Praticò, an associate professor of pharmacology in Temple's School of Medicine, demonstrated that a diet rich in methionine could increase the risk of developing Alzheimer's Disease. Methionine is an amino acid typically found in red meats, fish, beans, eggs, garlic, lentils, onions, yogurt and seeds.

"The question we asked now as a follow-up is if, for whatever reason, you had made bad choices in your diet, is there a chance you can slow down or even reverse the disease or is it too late - that there is nothing you could do," said Praticò.

As in the previous study, the researchers fed one group of mice a diet high in methionine and another group a regular, healthy diet. After five months, they split the group receiving the methionine-rich diet into two, with one group continuing the amino-heavy diet while the second switched to the healthy diet for an additional two months.

"At the end of the study, when we looked at these mice, what we found - very surprisingly - was that switching to a more healthy diet reversed the cognitive impairment that had built up over the first three months of eating the methionine-rich diet," said Praticò. "This improvement was associated with less amyloid plaques - another sign of the disease - in their brains.

Pratico said that the cognitive impairment that had been observed in the mice after three months on the methionine-rich diet was completely reversed after two months on the healthier diet, and they were now able to function normally. "We believe this finding shows that, even if you suffer from the early effects of MCI or Alzheimer's, switching to a healthier diet that is lower in methionine could be helpful in that memory capacity could be improved," he said.

Pratico stressed that this was not a drug therapy for curing MCI or Alzheimer's, but that it did demonstrate that a lifestyle change such as diet can improve some of the impairments that have already occurred in the brain.

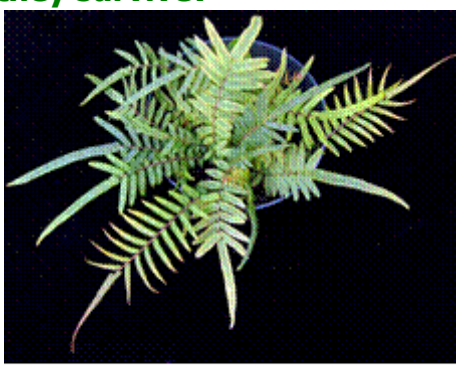
"What it tells us is that the brain has this plasticity to reverse a lot of the bad things that have occurred; the ability to recoup a lot of things such as memory that were apparently lost, but obviously not totally lost," he said. Pratico also emphasized that the researchers believe that in addition to switching to a healthy diet, patients diagnosed with MCI or Alzheimer's also need a regiment of physical as well as mental exercises.

"This combination won't cure you, but we believe, as we saw in this study, that it will be able to slow down or even possibly reverse the effects on the cognitive impairment," he said.

The study, "Normalization of hyperhomocysteinemia improves cognitive deficits and ameliorates brain amyloidosis of a transgenic mouse model of Alzheimer's disease," is being published in the Journal of the Federation of American Societies for Experimental Biology (http://www.fasebj.org/). It was funded by a grant from the National Institutes of Health.

Arsenic hyperaccumulating ferns: How do they survive?

Insights into arsenic transport and tolerance



Researchers discover mechanisms of arsenic hyperaccumulation and tolerance using the fern *Pteris vittata*.

Arsenic is toxic to most forms of life, and occurs naturally in soil and ground water in many regions of the world. Chronic exposure to arsenic has been linked to lung, bladder and kidney cancer, and thus there are strict limits on allowable levels or arsenic in drinking water. Chemically similar to phosphorus, arsenic forms arsenate (AsO_4^{3-}), which closely resembles phosphate (PO_4^{3-}). Arsenate interferes with many phosphate-requiring metabolic reactions, including synthesis of adenosine triphosphate (ATP), a ubiquitous and essential source of cellular energy. Thus, exposure to even low levels of arsenic can be extremely toxic.

In well-aerated soils, arsenic exists mainly as arsenate, which is taken up by plant roots using a phosphate transporter protein. Plant tissues rapidly reduce arsenate to arsenite (AsO_3^{3-}), which is transported to the aerial portions of the plant. In aquatic environments or water-logged soils, arsenic exists primarily as arsenite. Whereas rice grains can accumulate up to 60 μg/g arsenic, the fern Pteris vittata (see figure) can hyperaccumulate arsenic to levels 1000-fold greater than this. A team of researchers led by David Salt and Jo Ann Banks of Purdue University have recently isolated a gene encoding an arsenite transporter protein. This transporter allows these ferns to sequester arsenic in the vacuole, a cellular storage compartment isolated from the cytoplasm by the vacuolar membrane.

In research recently published in The Plant Cell and performed primarily by graduate student Emily Indriolo (now a researcher at the University of Toronto), these scientists describe how they used an arsenic-sensitive strain of yeast to isolate and characterize a gene encoding the P. vittata arsenite transporter. Yeast cells are arsenic-resistant because their plasma membrane contains an arsenite effluxer protein that is encoded by the Arsenical Compound Resistance 3 (ACR3) gene. The researchers introduced a library of P. vittata genes into an arsenic-sensitive acr3 mutant yeast strain and isolated a gene that restored arsenic resistance to this mutant. The protein encoded by this gene was then demonstrated to be very similar to the ACR3 protein of yeast in both structure and function.

Sequence analysis showed that this fern protein contains 10 putative transmembrane domains, suggesting a cellular membrane location. Using an antibody that specifically recognizes the ACR3 protein, they showed that ACR3 is found in the membranes of vacuoles, but not in the plasma membrane or in endoplasmic reticulum membranes. This suggests a mechanism for arsenic tolerance in P. vittata tissues: arsenite that enters the cell is transported by ACR3 into the vacuolar compartment, where it is spatially isolated from the cell cytoplasm, the site of many of the cell's arsenic-sensitive metabolic reactions.

Furthermore, the researchers showed that ACR3 gene expression in P. vittata is induced more than 30-fold in the presence of arsenite. To verify that ACR3 is required for arsenic tolerance, the ACR3 gene was silenced using an inhibitory mRNA. In these silenced plants, ACR3 expression was not induced by arsenite, and arsenic significantly reduced the growth rate of these ACR3-deficient plants relative to unsilenced plants.

Sequence analysis showed that, although this gene is found in a wide range of organisms including bacteria, fungi, mosses and gymnosperms, it is absent in angiosperms. By studying the occurrence and function of ACR3 in various plants, including hyperaccumulating and nonaccumulating ferns, the authors hope to provide additional insights into mechanisms of arsenic transport, tolerance, and accumulation. In addition to potential benefits for human health, this research will hopefully lead to strategies for phytoremediation of arsenic-contaminated soil and water.

This work was supported by the National Science Foundation and the National Institutes of Health with technical assistance from the Purdue Ionomics Center. The research paper cited in this report is available at the following link: http://www.plantcell.org/cgi/content/abstract/tpc.109.069773v1

Teen automobile crash rates are higher when school starts earlier

Early school start times promote sleep deprivation and daytime sleepiness, which can reduce the alertness of teen drivers

WESTCHESTER, IL – Earlier school start times are associated with increased teenage car crash rates, according to a research abstract that will be presented Wednesday, June 9, 2010, in San Antonio, Texas, at SLEEP 2010, the 24th annual meeting of the Associated Professional Sleep Societies LLC.

Results indicate that in 2008 the teen crash rate was about 41 percent higher in Virginia Beach, Va., where high school classes began at 7:20 a.m., than in adjacent Chesapeake, Va., where classes started more than an hour later at 8:40 a.m. There were 65.4 automobile crashes for every 1,000 teen drivers in Virginia Beach, and 46.2 crashes for every 1,000 teen drivers in Chesapeake.

"We were concerned that Virginia Beach teens might be sleep restricted due to their early rise times and that this could eventuate in an increased crash rate," said lead author Robert Vorona, MD, associate professor of internal medicine at Eastern Virginia Medical School in Norfolk, Va. "The study supported our hypothesis, but it is important to note that this is an association study and does not prove cause and effect."

The study involved data provided by the Virginia Department of Motor Vehicles. In Virginia Beach there were 12,916 drivers between 16 and 18 years of age in 2008, and these teen drivers were involved in 850 crashes. In Chesapeake there were 8,459 teen drivers and 394 automobile accidents. The researchers report that the two adjoining cities have similar demographics, including racial composition and per-capita income.

Further analysis by time of day found that in both cities the afternoon crash rates were higher than the morning crash rates. In Virginia Beach, where the school day ended around 2 p.m., the afternoon crash rate peaked from 2 p.m. to 6 p.m. In Chesapeake, where school dismissed at about 3:40 p.m., the afternoon crash rate peaked from 4 p.m. to 5 p.m. Six-hour analysis indicated that the overall afternoon crash rate per 1,000 teen drivers in Virginia Beach (35.2) was higher than in Chesapeake (20.6).

According to Vorona, delaying high school start times may promote driver alertness by reducing the severity of chronic sleep restriction, which is a common problem during adolescence. This idea is supported by a study in the December 2008 issue of the Journal of Clinical Sleep Medicine, which reported that the teen crash rate dropped by 16.5 percent in one county that delayed high school start times by an hour.

"We believe that high schools should take a close look at having later start times to align with circadian rhythms in teens and to allow for longer sleep times," said Vorona. "Too many teens in this country obtain insufficient sleep. A burgeoning literature suggests that this may lead to problematic consequences including mood disorders, academic difficulties and behavioral issues."

The study was supported by the Eastern Virginia School of Medicine Division of Sleep Medicine.

Global biodiversity estimate revised down

* 09:00 09 June 2010 **by Wendy Zuckerman**

Talk about overestimation. Only 5.5 million species may share our planet, a much smaller number than the older, often quoted estimate of more than 30 million.

Most vertebrates and plants and many microorganisms have been documented. Much of the uncertainty in such global estimates lies with arthropods, a phylum that includes insects and spiders.

Beetle mania

The global figure of over 30 million species was suggested in 1982 by Terry Erwin at the Smithsonian Institution in Washington DC. He counted 163 species of beetle exclusive to one tropical tree species in Panama, Luehea seemannii, then multiplied by the number of tree species globally. He also scaled up the result to take into account the fact that beetles make up around 40 per cent of arthropod species.

Erwin later called for others to improve on his estimate. Now Andrew Hamilton of the University of Melbourne, Australia, has obtained a revised estimate using observations of 434 beetle species on 56 tree species in Papua New Guinea.

He concludes with 90 per cent certainty that there are between 2.5 and 3.7 million arthropod species. The probability of Erwin's estimate of 30 million species being correct is less than 0.001 per cent, he says.

Grand total

So what does that mean for the total number of species? If you add to the arthropods the known vertebrates (approximately 50,000 species), plants (400,000) and other organisms such as fungi and algae (1.3 million), "the magic number is 5.5 million", says Hamilton.

Neither he nor Erwin included bacteria, however, as they are hard to separate into species.

Erwin's figure of 30 million was unrealistic, says Corey Bradshaw at the University of Adelaide, Australia. But he adds that Hamilton's extrapolation of global arthropod numbers using data from just one country underestimates the role that local geographical and ecological factors play in driving biodiversity.

Conservative count

According to Bradshaw, the new prediction is probably too conservative. The true number probably lies somewhere in between the two estimates, he says.

Hamilton's estimate is "perfectly reasonable", says Margie Mayfield of the University of Queensland, Australia. "You can think of these results as a very well informed vote" on how many species there are on Earth, she says.

Journal reference: American Naturalist, DOI: 10.1086/652998

Letter to UC Faculty on Nature Publishing Group Subscription Increases

Author: **Ellen Meltzer**

A letter dated June 4, 2010 describing exorbitant subscription increases on the part of the Nature Publishing Group (NPG) for the 67 journals UC licenses (including Nature) was distributed to UC faculty by campus librarians with the support of faculty library committees, including the systemwide University Committee on Library and Scholarly Communication (UCOLASC). The letter is an informational update about the UC Libraries' pricing challenges with NPG and the likelihood that the libraries will have to cancel some or all NPG titles in light of the University's current budget challenges. The letter also describes a potential boycott that some faculty are proposing if the dispute cannot be satisfactorily resolved. The boycott would encourage faculty to:

* Decline to peer review manuscripts for journals from the Nature Publishing Group.
* Resign from Nature Publishing Group editorial and advisory boards.
* Cease to submit papers to the Nature Publishing Group.
* Refrain from advertising any open or new UC positions in Nature Publishing Group journals.
* Talk widely about Nature Publishing Group pricing tactics and business strategies with colleagues outside UC, and encourage sympathy actions such as those listed above.

An article appearing in The Chronicle of Higher Education (June 8, 2010), U. of California Tries Just Saying No to Rising Journal Costs, includes interviews with Laine Farley, Executive Director, CDL; Rich Schneider, UCSF, the chair of UCOLASC; and Keith Yamamoto, also of UCSF (referenced in the letter).

UC will be taking the pulse of faculty response to this issue in hopes that it will prompt a more productive dialogue with Nature Publishing Group.

New evidence that drinking coffee may reduce the risk of diabetes

Scientists are reporting new evidence that drinking coffee may help prevent diabetes and that caffeine may be the ingredient largely responsible for this effect. Their findings, among the first animal studies to demonstrate this apparent link, appear in ACS' bi-weekly Journal of Agricultural and Food Chemistry.

Fumihiko Horio and colleagues note that past studies have suggested that regular coffee drinking may reduce the risk of type 2 diabetes. The disease affects millions in the United States and is on the rise worldwide. However, little of that evidence comes from studies on lab animals used to do research that cannot be done in humans.

The scientists fed either water or coffee to a group of laboratory mice commonly used to study diabetes. Coffee consumption prevented the development of high-blood sugar and also improved insulin sensitivity in the mice, thereby reducing the risk of diabetes. Coffee also caused a cascade of other beneficial changes in the fatty liver and inflammatory adipocytokines related to a reduced diabetes risk. Additional lab studies showed that caffeine may be "one of the most effective anti-diabetic compounds in coffee," the scientists say.

"Coffee and Caffeine Ameliorate Hyperglycemia, Fatty Liver, and Inflammatory Adipocytokine Expression in Spontaneously Diabetic KK-Ay Mice"

Researchers report new autism genes discovered

University of Illinois at Chicago researchers are part of an international consortium working with Autism Speaks, the world's largest autism science and advocacy organization, which today reports new autism genetic discoveries.

The results, from the second phase of the collaborative Autism Genome Project, are published in the June 10 issue of the journal Nature.

Autism is a complex neurobiological disorder that inhibits a person's ability to communicate and develop social relationships, and is often accompanied by behavioral challenges. Autism spectrum disorders are diagnosed in one in 110 children in the U.S., affecting four times as many boys as girls.

The new report shows that individuals with autism tend to carry more sub-microscopic insertions and deletions called copy-number variants (CNV) in their genome than nonautistic people do. Some of these CNV appeared to be inherited, while others are considered new because they are found only in affected offspring and not in the parents.

Taken together, more of the CNVs disrupt genes previously reported to be implicated in intellectual disability without autism or in autism than expected by chance.

The findings are based on analysis of high-density genotyping data collected from 1,000 individuals with autism spectrum disorder (ASD) and 1,300 without ASD.

The new study also identified new autism susceptibility genes including SHANK2, SYNGAP1, DLGAP2 and the X-linked DDX53–PTCHD1 locus. Some of these genes belong to synapse-related pathways, while others are involved in cellular proliferation, projection and motility, and intracellular signaling, functional targets that may lead to the development of new treatment approaches.

These findings further support an emerging consensus within the scientific community that autism is caused in part by many "rare variants" or genetic changes found in less than 1 percent of the population.

While each of these variants may only account for a small fraction of the cases, collectively they are starting to account for a greater percentage of individuals in the autism community, as well as providing insights into possible common pathogenic mechanisms.

The overlap between autism susceptibility genes and genes previously implicated in intellectual disabilities further supports the hypothesis that at least some genetic risk factors are shared by different psychiatric developmental disabilities. The identification of these biological pathways points to new avenues of scientific investigation, as well as potential targets for the development of novel treatments, according to the authors.

"These results are another step on the long path to sufficiently understanding autism to further develop treatments for the core symptoms of autism," says Dr. Edwin Cook, UIC professor of psychiatry.

"At the Autism Center of Excellence at UIC, we continue to work to understand the genetics, neurobiology, and treatment of autism," he said.

Polyphenols in red wine and green tea halt prostate cancer growth

New report in the FASEB Journal suggests that disrupting a particular cellular signaling pathway could stop or slow the initiation, promotion, and progression of prostate cancer

In what could lead to a major advance in the treatment of prostate cancer, scientists now know exactly why polyphenols in red wine and green tea inhibit cancer growth. This new discovery, published online in The FASEB Journal (http://www.fasebj.org), explains how antioxidants in red wine and green tea produce a combined effect to disrupt an important cell signaling pathway necessary for prostate cancer growth. This finding is important because it may lead to the development of drugs that could stop or slow cancer progression, or improve current treatments.

"Not only does SphK1/S1P signaling pathway play a role in prostate cancer, but it also plays a role in other cancers, such as colon cancer, breast cancer, and gastric cancers," said Gerald Weissmann, MD, editor-in-chief of The FASEB Journal. "Even if future studies show that drinking red wine and green tea isn't as effective in humans as we hope, knowing that the compounds in those drinks disrupts this pathway is an important step toward developing drugs that hit the same target."

Scientists conducted in vitro experiments which showed that the inhibition of the sphingosine kinase-1/sphingosine 1-phosphate (SphK1/S1P) pathway was essential for green tea and wine polyphenols to kill prostate cancer cells. Next, mice genetically altered to develop a human prostate cancer tumor were either treated or not treated with green tea and wine polyphenols. The treated mice showed reduced tumor growth as a result of the inhibited SphK1/S1P pathway. To mimic the preventive effects of polyphenols, another experiment used three groups of mice given drinking water, drinking water with a green tea compound known as EGCg, or drinking water with a different green tea compound, polyphenon E. Human prostate cancer cells were implanted in the mice and results showed a dramatic decrease in tumor size in the mice drinking the EGCg or polyphenon E mixtures.

"The profound impact that the antioxidants in red wine and green tea have on our bodies is more than anyone would have dreamt just 25 years ago," Weissmann added. "As long as they are taken in moderation, all signs show that red wine and green tea may be ranked among the most potent 'health foods' we know."

Improving recovery from spinal cord injury

Once damaged, nerves in the spinal cord normally cannot grow back and the only drug approved for treating these injuries does not enable nerve regrowth. Publishing online this week in the Early Edition of the Proceedings of the National Academy of Sciences, researchers at the Johns Hopkins University School of Medicine show that treating injured rat spinal cords with an enzyme, sialidase, improves nerve regrowth, motor recovery and nervous system function.

"This is the first functional study showing behavioral improvement below a spinal cord injury by the delivery of sialidase," says Ronald Schnaar, Ph.D., a professor of pharmacology and molecular sciences at Johns Hopkins. "Sialidase has properties that are appealing from the human drug development point of view."

Sialidase is a bacterial enzyme that removes specific chemical groups found on the surface of nerve cells. The chemical groups normally function to stablize the cells, but also act to prevent nerve regeneration.

The team built upon earlier research where they disovered that sialidase treatment improved the growth of nerves into a graft. "We wanted to take this further and look at the animal model most relevant to human spinal cord injury," says Schnaar. "Typically, in motor vehicle accidents for example, vertebra shift and pinch the spinal cord, severing the long spinal nerve axons like you would if you pinched a piece of wet spaghetti." So they treated rats after a spinal cord impact injury by injecting sialidase directly to the injury site.

Rats with lower-back impact injury - severe enough to lose hind-limb function - were injected with sialidase directly over the spinal cord immediately following injury. The researchers then implanted into each rat a small pump that delivered a steady stream of sialidase directly to the injury over the course of two weeks, hoping that bathing the injured nerves in the enzyme would help their recovery and promote regrowth. They then let the rats recover for another three weeks before assessing the degree of recovery.

Using a well-established, 21-point scale where zero represents paralysis and 21 is normal function, the team of researchers assesed treated and untreated rats for a range of functions including whether they could lift their feet off the ground and whether they had coordinated leg movements. The initial injury rendered all rats to score below four, and all rats, treated or not, recovered somewhat by the end of two weeks. By the end of five weeks after injury most untreated rats scored 12 or less, while most treated rats scored better than 15. "The difference in coordination control was most remarkable," says Schnaar.

In addition to motor control, spinal cord injury can cause other nervous system problems, including losing the ability to control blood pressure and heart rate. To see if sialidase treatment improved nerve connections enough to remedy these problems, the team measured the nerve circuits that control blood pressure in treated and untreated rats. They found that treated animals improved blood pressure control. "We interpret this as improved communication in the spinal cord," says Schnaar.

Finally, the team looked at the nerve ends under a microscope and found that indeed, treated nerves showed an increased number of "sprouted" nerve ends, which according to Schnaar, provided anatomical evidence to add to the functional evidence that "something is going on."

"The positive is that we have shown functional recovery in a relevant animal model of spinal cord injury," says Schnaar. "That being said, we haven't done full toxicity studies on these rats, which definitely needs to be done before we think about taking the long road into using this as a drug in people; efficacy in animals also doesn't necessarily translate to humans."

This study was funded by the National Institutes of Health and by the PhRMA Foundation.

Authors on the paper are Andrea Mountney, Matthew R. Zahner, Ileana Lorenzini, Martin Oudega, Lawrence P. Schramm and Ronald L. Schnaar, all of Johns Hopkins. Martin Oudega also was a member of the International Center for Spinal Cord Injury at the Hugo W. Moser Research Institute of Kennedy Krieger.

Popular Cancer Drug Can Cause Kidney Damage

Bevacizumab Increases the Risk of Severe Urinary Protein Loss by More than 4-Fold

Washington, DC - The widely used cancer drug bevacizumab may cause severe loss of protein from the kidney into the urine that can lead to significant kidney damage and can compromise the efficacy of cancer treatment, according to a study appearing in an upcoming issue of the Journal of the American Society of Nephrology (JASN). The results suggest that physicians should monitor patients' kidney health when prescribing this angiogenesis inhibitor.

While research indicates that treatment with the chemotherapy drug bevacizumab can lead to urinary protein leakage (proteinuria) and kidney damage, the overall risk associated with the drug and patient risk factors are unknown. Bevacizumab blocks a protein called vascular endothelial growth factor, thus inhibiting the production of new blood vessels around tumors.

Shenhong Wu MD, PhD (Stony Brook University Cancer Center), Xiaolei Zhu, MD, PhD (Kidney Doctors PLLC), and their colleagues conducted a review of published randomized, controlled clinical trials to assess the overall risk for severe proteinuria in patients taking bevacizumab. The researchers analyzed data from 16 studies comprising 12,268 patients with a variety of tumors.

Severe proteinuria occurred in 2.2% of patients taking bevacizumab. Compared with patients taking chemotherapy alone, patients taking bevacizumab combined with chemotherapy had a 4.79-fold increased risk of developing severe proteinuria and a 7.78-fold increased risk of developing nephrotic syndrome. (Nephrotic syndrome is a group of symptoms including protein in the urine, low blood protein levels, high cholesterol levels, high triglyceride levels, and swelling.)

Patients taking higher dosages of bevacizumab had the greatest risk of developing proteinuria. Also, when the investigators looked at differences by cancer type, they found that patients with kidney cancer had the highest risk of developing proteinuria (10.2% incidence).

These results indicate that it is particularly important to monitor the effects of bevacizumab in patients who have kidney cancer or who are receiving high doses of the drug. Future studies should investigate how to reduce bevacizumab's kidney-related effects, and physicians should be prepared to treat these potential side effects. Study co-authors include Christi Kim, MD and Lea Baer, MD (Stony Brook University Cancer Center).

***Disclosures**: Dr. Wu received honoraria from Onyx Pharmaceuticals, Novartis, and Wyeth, and has been a speaker for Onyx, Pfizer Inc, and Novartis. The other authors reported no financial disclosures. The article, entitled "Bevacizumab Increases Risk for Severe Proteinuria in Cancer Patients," will appear online at http://jasn.asnjournals.org/ on June 10, 2010, doi 10.1681/ASN.2010020167.*

Inexpensive drug to stop sight loss shown to be effective

An inexpensive, but unlicensed drug to help prevent severe sight loss in older people has been shown to be safe and effective, finds a study published on bmj.com today.

Bevacizumab (Avastin) is licensed as a treatment for bowel cancer, but it is widely used "off label" as a considerably cheaper alternative to the approved drug ranibizumab (Lucentis) to prevent wet age related macular degeneration (AMD) and several large trials comparing the two drugs are now underway.

Although ranibizumab was not included in this study (it was not licensed for use when the trial began) the researchers support its immediate implementation in healthcare systems whose budgetary limitations prevent patients' access to ranibizumab. In the majority of countries in the world, where either no treatment or inferior therapies are available to patients with wet AMD, the appropriate use of bevacizumab, a highly cost effective intervention, would have an immediate impact in reducing incident blindness from this condition, they say.

Wet AMD is the leading cause of visual loss in people over the age of 50 in Europe and North America. Visual loss is a result of progressive loss of light sensitive cells at the back of the eye due to damage from abnormal, leaking blood vessels. Sufferers do not go blind, but find it virtually impossible to read, drive, or do tasks requiring fine, sharp, central vision.

In 2006, researchers based at three UK eye centres, set out to test whether bevacizumab is an effective and safe treatment for wet AMD compared with standard NHS care available at the time. A total of 131 patients aged at least 50 years with wet AMD were randomised to either bevacizumab injections at six week intervals or standard care (one of three different treatments available on the NHS at the start of the study). Visual acuity was measured at the start of the study (baseline) and then monitored over one year (54 weeks).

At one year, 32% of patients in the bevacizumab group gained 15 or more letters from baseline visual acuity compared with 3% in the standard care group. In addition, the proportion of patients who lost fewer than 15 letters of visual acuity from baseline was significantly greater among those receiving bevacizumab treatment (91%) compared with 67% in the standard care group.

Average visual acuity increased by seven letters in the bevacizumab group with a median of seven injections compared with a decrease of 9.7 letters in the standard care group, and the initial improvement at week 18 was sustained to week 54. Bevacizumab treatment was associated with a low rate of serious adverse events.

These results show that bevacizumab injections given at six weekly intervals for wet AMD is superior to the standard care available at the start of the trial, say the authors. This trial provides level-one evidence for the use of bevacizumab injections for the treatment of wet AMD, they conclude.

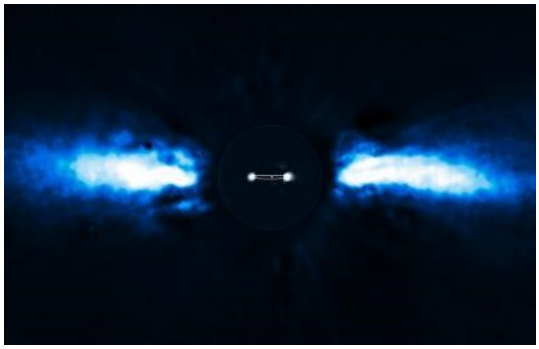
In an accompanying editorial, Professor Usha Chakravarthy from the Royal Victoria Hospital in Belfast says that, although this trial fills a gap in the evidence base and shows robustly that bevacizumab is better than previously employed treatments, it does not tell us whether the drug is as effective as ranibizumab. And she warns that "the off label use of bevacizumab should not be encouraged until the large randomised trials comparing it with ranibizumab report their findings."

Exoplanet caught on the move

Only 12 million years old, or less than three-thousandths of the age of the Sun, Beta Pictoris is 75% more massive than our parent star. It is located about 60 light-years away towards the constellation of Pictor (the Painter) and is one of the best-known examples of a star surrounded by a dusty debris disc [1]. Earlier observations showed a warp of the disc, a secondary inclined disc and comets falling onto the star. "Those were indirect, but tell-tale signs that strongly suggested the presence of a massive planet, and our new observations now definitively prove this," says team leader Anne-Marie Lagrange. "Because the star is so young, our results prove that giant planets can form in discs in time-spans as short as a few million years."

Recent observations have shown that discs around young stars disperse within a few million years, and that giant planet formation must occur faster than previously thought. Beta Pictoris is now clear proof that this is indeed possible.

The team used the NAOS-CONICA instrument (or NACO [2]), mounted on one of the 8.2-metre Unit Telescopes of ESO's Very Large Telescope (VLT), to study the immediate surroundings of Beta Pictoris in 2003, 2008 and 2009. In 2003 a faint source inside the disc was seen (eso0842), but it was not possible to exclude the remote possibility that it was a background star. In new images taken in 2008 and spring 2009 the source had disappeared! The most recent observations, taken during autumn 2009, revealed the object on the other side of the disc after a period of hiding either behind or in front of the star (in which case it is hidden in the glare of the star). This confirmed that the source indeed was an exoplanet and that it was orbiting its host star. It also provided insights into the size of its orbit around the star.



Images are available for approximately ten exoplanets, and the planet around Beta Pictoris (designated "Beta Pictoris b") has the smallest orbit known so far. It is located at a distance between 8 and 15 times the Earth-Sun separation - or 8-15 Astronomical Units - which is about the distance of Saturn from the Sun. "The short period of the planet will allow us to record the full orbit within maybe 15-20 years, and further studies of Beta Pictoris b will provide invaluable insights into the physics and chemistry of a young giant planet's atmosphere," says student researcher Mickael Bonnefoy.

The above composite shows the reflected light on the dust disc in the outer part, as observed in 1996 with the ADONIS instrument on ESO's 3.6-metre telescope. In the central part, the observations of the planet obtained in 2003 and autumn 2009 with NACO are shown. The possible orbit of the planet is also indicated, albeit with the inclination angle exaggerated. **ESO/A.-M. Lagrange**

The planet has a mass of about nine Jupiter masses and the right mass and location to explain the observed warp in the inner parts of the disc. This discovery therefore bears some similarity to the prediction of the existence of Neptune by astronomers Adams and Le Verrier in the 19th century, based on observations of the orbit of Uranus. "Together with the planets found around the young, massive stars Fomalhaut and HR8799, the existence of Beta Pictoris b suggests that super-Jupiters could be frequent byproducts of planet formation around more massive stars," explains Gael Chauvin, a member of the team.

Such planets disturb the discs around their stars, creating structures that should be readily observable with the Atacama Large Millimeter/submillimeter Array (ALMA), the revolutionary telescope being built by ESO together with international partners.

A few other planetary candidates have been imaged, but they are all located further from their host star than Beta Pictoris b. If located in the Solar System, they all would lie close to or beyond the orbit of the furthest planet, Neptune. The formation processes of these distant planets are likely to be quite different from those in our Solar System and in Beta Pictoris. "The recent direct images of exoplanets - many made by the VLT- illustrate the diversity of planetary systems," says Lagrange. "Among those, Beta Pictoris b is the most promising case of a planet that could have formed in the same way as the giant planets in our Solar System."

This research was presented in a paper to appear this week in the journal Science ("A Giant Planet Imaged in the disk of the Young Star Beta Pictoris," by A.-M. Lagrange et al.).

Many Solar System Comets May Have Been Swiped from Other Stars

The cloud of comets in the outer solar system could include a significant contribution from the sun's former stellar neighbors

By John Matson

Comets are usually thought of as icy, dusty emissaries from the deepest reaches of the solar system. But according to a new simulation, many of them could have originated somewhere even more exotic - in budding planetary systems around other stars.

Like most stars, the sun may very well have been created in a tightly nestled birth cluster, a stellar nursery with tens, hundreds or possibly even thousands of stars. During millions of years of intimate infancy, the newborn stars could have exchanged vast numbers of comets from the fringes of their disks, each of them winding up with an ensemble of hand-me-downs from their stellar siblings.

According to a study published online June 10 in Science, the capture mechanism could account for a substantial share of the comets in the Oort Cloud, a spherical flock of hundreds of billions of comets, and perhaps more, at the solar system's outer fringes. The new model could resolve the confoundingly large

population of the Oort Cloud, says Harold Levison, a planetary scientist at the Southwest Research Institute in Boulder, Colo.

Prevailing theories of solar system formation, Levison explains, hold that early in the system's history there were plenty of icy objects left over from planetary formation. But interactions with the newly formed giant planets ejected many of those comets into interstellar space, flung others out into what would become the Oort Cloud, and knocked some into elongated, somewhat shorter orbits in what is known as the scattered disk.

Scattered disk objects occupy elliptical orbits and may venture as close to the sun as Neptune's orbit before looping back out to the deeper reaches of the solar system. Models of planetesimal dispersion predict that roughly 10 times as many objects wound up in the Oort Cloud as in the scattered disk, according to the new study.

But observations do not match theory: By one estimate, the Oort Cloud contains some 700 times as many comets as the scattered disk. So Levison's group set out to test a theory floating around since at least 1990 - that star-mingling billions of years ago could provide an additional cometary reservoir to populate the Oort Cloud.

In the new simulation the researchers modeled interactions among 30 to 300 stars packed in a birth cluster just a few light-years across. They followed the evolution of the clusters until the stars dispersed, which took anywhere from 10 million to 52 million years. Two mechanisms for comet exchange emerged: In the more common scenario gravitational effects among neighboring stars stripped off comets that then roamed free through the cluster before hitching a ride with another star when the cluster dispersed. The other, less common, mechanism showed that comets could migrate directly and in large numbers from one star to another during a close approach.

A rough estimation, assuming that our solar system is fairly typical, finds that one third to two thirds of the Oort Cloud comets originated in the grip of another star. A more dramatic assessment, which the researchers assert is more realistic, comes from a simple comparison of observation and theory. The capture process, Levison says, "is the only mechanism that we can think of that could contribute to the Oort Cloud" beyond the standard prediction, which appears to provide a woefully insufficient number of comets. If the capture of comets from other stars indeed provided the rest, that would mean that more than 90 percent of the Oort Cloud sprang from extrasolar beginnings.

But estimates for the cloud's size are indirect, based on observations of the few comets that venture inward to the planetary region; the cloud itself is too distant to be directly observed. And population estimates for the scattered disk also vary. The contents of both reservoirs "are uncertain by an order of magnitude," says Paul Weissman, a senior research scientist at the NASA Jet Propulsion Laboratory in Pasadena, Calif. "So I'm not entirely certain that they have a problem that needs solving."

Faced with these uncertainties, Levison and his group did choose somewhat conservatively from the available population estimates, picking a relatively small figure for the size of the Oort Cloud and a large one for the scattered disk. "I think the numbers are pretty solid," Levison says.

Weissman takes issue, however, with the larger estimate of the contribution of extrasolar comets to the Oort Cloud. "The claim that over 90 percent of the cloud is captured is a substantial overstatement," Weissman says. "What they have shown is that the number can be about one third to two thirds." Even that, he notes, is "a very important result."

Regardless of whether the sun nabbed one third, two thirds or nearly all of its comets from its birth-cluster companions, why should it have made out so well? In the standard model of solar system formation, Levison notes, the bulk of the comets originally available in the sun's disk wound up not in the Oort Cloud but flung clear into interstellar space. The capture mechanism provides a way that some of those losses could have been offset. "It's not that our sun got more at the expense of somebody else," Levison says. "Everybody gets more."

Why Patients Aren't Getting the Shingles Vaccine

By PAULINE W. CHEN, M.D.

Four years ago at age 78, R., a retired professional known as much for her small-town Minnesotan resilience as her commitment to public service, developed a fleeting rash over her left chest. The rash, which turned out to be shingles, or herpes zoster, was hardly noticeable.

But the complications were unforgettable.

For close to a year afterward, R. wrestled with the searing and relentless pain in the area where the rash had been. “It was ghastly, the worst possible pain anyone could have,” R. said recently, recalling the sleepless nights and fruitless search for relief. “I’ve had babies and that hurts a lot, but at least it goes away. This pain never let up. I felt like I was losing my mind for just a few minutes of peace.”

Shingles and its painful complication, called postherpetic neuralgia, result from reactivation of the chicken pox virus, which remains in the body after a childhood bout and is usually dormant in the adult. Up to a third of

all adults who have had chicken pox will eventually develop one or both of these conditions, becoming debilitated for anywhere from a week to several years. That percentage translates into about one million Americans affected each year, with older adults, whose immune systems are less robust, being most vulnerable. Once the rash and its uncomfortable sequel appear, treatment options are limited at best and carry their own set of complications.

While the search for relief costs Americans over $500 million each year, the worst news until recently has been that shingles could happen to any one of us. There were no preventive measures available.

But in 2006, the Food and Drug Administration approved a new vaccine against shingles. Clinical trials on the vaccine revealed that it could, with relatively few side effects, reduce the risk of developing shingles by more than half and the risk of post-herpetic neuralgia by over two-thirds. In 2008, a national panel of experts on immunizations at the Centers for Disease Control and Prevention went on to recommend the vaccine to all adults age 60 and older.

At the time, the shingles vaccine seemed to embody the best of medicine, both old school and new. Its advent was contemporary medicine's elegant response to a once intractable, age-old problem. It didn't necessarily put an end to the spread of disease, in this case chicken pox; but it dramatically reduced the burden of illness for the affected individual. And, most notably, its utter simplicity was a metaphoric shot-in-the-arm for old-fashioned doctoring values. Among the increasingly complex and convoluted suggestions for health care reform that were brewing at that moment, here was a powerful intervention that relied on only three things: a needle, a syringe and a patient-doctor relationship rooted in promoting wellness.

Not.

In the two years since the vaccine became available, fewer than 10 percent of all eligible patients have received it. Despite the best intentions of patients and doctors (and no shortage of needles and syringes), the shingles vaccine has failed to take hold, in large part because of the most modern of obstacles. What should have been a widely successful and simple wellness intervention between doctors and their patients became a 21st century Rube Goldberg-esque nightmare.

Last month in The Annals of Internal Medicine, researchers from the University of Colorado in Denver and the C.D.C. surveyed almost 600 primary care physicians and found that fewer than half strongly recommended the shingles vaccine. Doctors were not worried about safety - a report in the same issue of the journal confirmed that the vaccine has few side effects; rather, they were concerned about patient cost.

Although only one dose is required, the vaccination costs $160 to $195 per dose, 10 times more than other commonly prescribed adult vaccines; and insurance carriers vary in the amount they will cover. Thus, while the overwhelming majority of doctors in the study did not hesitate to strongly recommend immunizations against influenza and pneumonia, they could not do the same with the shingles vaccine.

"It's just a shot, not a pap smear or a colonoscopy," said Dr. Laura P. Hurley, lead author and assistant professor of medicine at the University of Colorado in Denver. "But the fact is that it is an expensive burden for all patients, even those with private insurance and Medicare because it is not always fully reimbursed."

Moreover, many private insurers require patients to pay out of pocket first and apply for reimbursement afterward. And because the shingles vaccine is the only vaccine more commonly given to seniors that has been treated as a prescription drug, eligible Medicare patients must also first pay out of pocket then submit the necessary paperwork in order to receive the vaccine in their doctor's office. It's a complicated reimbursement process that stands in stark contrast to the automatic, seamless and fully covered one that Medicare has for flu and pneumonia vaccines.

Despite this payment maze, some physicians have tried to stock and administer the vaccine in their offices; many, however, eventually stop because they can no longer afford to provide the immunizations. "If you have one out of 10 people who doesn't pay for the vaccine, your office loses money," said Dr. Allan Crimm, the managing partner of Ninth Street Internal Medicine, a primary care practice in Philadelphia. Over time, Dr. Crimm's practice lost thousands of dollars on the shingles vaccine. "It's indicative of how there are perverse incentives that make it difficult to accomplish what everybody agrees should happen."

Even bypassing direct reimbursement is fraught with complications for doctors and patients. A third of the physicians surveyed in the University of Colorado study resorted to "brown bagging," a term more frequently used to describe insurers who have patients carry chemotherapy drugs from a cheaper supplier to their oncologists' offices. In the case of the shingles vaccine, the study doctors began writing prescriptions for patients to pick up the vaccine at the pharmacy and then return to have it administered in their offices. However, the shingles vaccine must be frozen until a few minutes before administration, and a transit time greater than 30 minutes between office and pharmacy can diminish the vaccine's effectiveness.

Dr. Crimm and the physicians in his office finally resorted to what another third of the physicians in the study did: they gave patients prescriptions to have the vaccine administered at pharmacies that offered immunization clinics. But when faced with the added hassles of taking additional time off from work and making a separate trip to the pharmacy, not all patients followed through. “Probably about 60 percent of our patients finally did get the vaccine at the pharmacy,” Dr. Crimm estimated. “This is as opposed to 98 percent of our patients getting the pneumonia and influenza vaccines, immunizations where they just have to go down the hall because we stock it, roll up their sleeves then walk out the door.”

With all of these barriers, it comes as no surprise that in the end only 2 percent to 7 percent of patients are immunized against shingles. “There’s just so much that primary care practices must take care of with chronic diseases like obesity and diabetes and heart disease,” Dr. Hurley noted. “If a treatment isn’t easy to administer, then sometimes it just falls to the bottom of the list of things for people to do. Shingles vaccination has become a disparity issue,” Dr. Hurley added. “It’s great that this vaccine was developed and could potentially prevent a very severe disease. But we have to have a reimbursement process that coincides with these interventions. Just making these vaccines doesn’t mean that they will have a public health impact.”

Diabetes May Double Cancer Risk in Women

TAU study confirms early detection tests are key for diabetic women

Type 2 adult-onset diabetes causes insulin-like hormones to circulate through the body. A new study finds this has a surprisingly positive effect on reducing the rate of prostate cancer in men, but is bad news for women: Type 2 diabetes may double the risk of female genital and other cancers.

The new study, led by Dr. Gabriel Chodick and Dr. Varda Shalev of Tel Aviv University's Department of Epidemiology and Preventive Medicine at the Sackler Faculty of Medicine, is not the first to report such a risk. But it's one of the largest to confirm these findings, and it's the first to determine the statistical differences in cancer risks for men and women.

Recently published in the journal Cancer Causes & Control, the Tel Aviv University study looked at 16,721 diabetics, differentiating between men and women and defining the relative cancer risks for each group. When the study began in 2000, none of the subjects had a history of cancer. Over the following eight years, the researchers documented 1,639 cases of different cancers among people with diabetes, and compared them to occurrences of the same cancers in the healthy non-diabetic population - a sample of 83,874 people.

Good news for men, bad news for women

"For men, this study is good news," says Dr. Chodick. The study demonstrates that diabetes actually appears to have a preventative effect on conditions like prostate cancer, reducing the risk of cancers associated with insulin-like hormones by a whopping 47 percent. But the opposite is true for women, he continues. "The interaction of diabetes and female hormones appears to exaggerate the risk, and make certain organs like the uterus and ovaries more receptive to certain kinds of cancer."

While the news is something for female diabetics and their practitioners to take into consideration, there's no cause for panic, Dr. Chodick notes. Although colon and ovarian cancers are serious, their overall risk in women is generally quite low. However, he stresses that physicians should take the research into account when assessing the long-term health histories of their patients.

An ounce of prevention still the best medicine

Dr. Chodick encourages diabetic women to be screened for colon cancer earlier and more often than those in the general population. As the occurrence of diabetes in America rises, primarily brought on by an unhealthy lifestyle, such screenings can save lives.

Type 2 diabetes, which is characterized by high blood glucose and an insulin deficiency, normally occurs in adulthood, and it can often be managed with a healthy diet, exercise, and oral medications. It affects more than 10% of all women in America over the age of 20, according to the American Diabetes Association.

Of course, wherever possible, the best approach is to avoid diabetes entirely, says Dr. Chodick, with the help of a high-fiber, low-carbohydrate diet combined with exercise.

New model is proposed to explain absence of organic compounds on surface of Mars

New Rochelle - The ongoing search for evidence of past or present life on Mars includes efforts to identify organic compounds such as proteins in Martian soil, but their absence to date remains a mystery. A new theory to explain what happens to these carbon-based molecules is presented in an article published in Astrobiology, a peer-reviewed journal published by Mary Ann Liebert, Inc. The article is available free online at www.liebertpub.com/ast.

"There may be no 'safe haven' for these organic molecules on Mars," conclude Ilya Shkrob, Sergey Chemerisov, and Timothy Marin, from Argonne National Laboratory and Benedictine University, in Illinois, in

their article entitled "Photocatalytic Decomposition of Carboxylated Molecules on Light-Exposed Martian Regolith and its Relation to Methane Production on Mars."

Unlike on Earth, where plants and other organisms convert carbon dioxide and water into organic compounds via photosynthesis, the authors propose that the opposite happens on the surface of Mars. The iron oxides that make up Martian soil and give the planet its distinctive red color are photocatalysts. They use energy from ultraviolet light absorbed through the thin Martian atmosphere to oxidize carbon-containing organic molecules trapped in soil particles, converting them to carbon dioxide and gases such as methane.

The authors present study data to support this model and to explain why it might not be realistic to rely on the discovery of proteins, amino acids, and other carbon-containing compounds in the upper soil layers of Mars to determine whether life forms are or have been present on the planet.

"This is an interesting result and may be an important step in solving the enduring mystery of organics on Mars," says Christopher P. McKay, Senior Editor of Astrobiology and Research Scientist at NASA Ames Research Center. "We see organics in many places in the solar system but have not been able to detect them on Mars - the planet that we think had the most Earth-like conditions. Why? Could it be our instrument approach has been wrong? Or could it be that there is some chemistry on Mars that is actively destroying organics? This work points toward this latter explanation. Mars may have a self cleaning surface. If so, we may have to dig deeply to find any organic materials."

"The importance of drilling below the Martian surface for rocks and soils that might retain preserved organics is certainly on the minds of future mission scientists," says Sherry L. Cady, PhD, Editor of Astrobiology and Associate Professor in the Department of Geology at Portland State University. "The possible 2018 joint ESA-NASA mission is a case in point."

Freezing 'to death' and living to tell about it: Study reveals how suspended animation protects against lethal hypothermia

Findings in yeast and worms may have implications for extending preservation of human organs for transplantation

SEATTLE - How is it that some people who apparently freeze to death, with no heart rate or respiration for extended periods, can be brought back to life with no long-term negative health consequences? New findings from the laboratory of cell biologist Mark B. Roth, Ph.D., of Fred Hutchinson Cancer Research Center, may help explain the mechanics behind this widely documented phenomenon.

Reporting online ahead of the July 1 print issue of Molecular Biology of the Cell, Roth, a member of the Hutchinson Center's Basic Sciences Division, and colleagues show that two widely divergent model organisms – yeast and nematodes, or garden worms – can survive hypothermia, or potentially lethal cold, if they are first put into a state of suspended animation by means of anoxia, or extreme oxygen deprivation.

Roth and colleagues found that under normal conditions, yeast and nematode embryos cannot survive extreme cold. After 24 hours of exposure to temperatures just above freezing, 99 percent of the creatures expire. In contrast, if the organisms are first deprived of oxygen and thus enter a state of anoxia-induced suspended animation, 66 percent of the yeast and 97 percent of the nematode embryos will survive the cold. Once normal growth conditions are resumed – upon rewarming and reintroduction of oxygen – the organisms will reanimate and go on to live a normal lifespan.

A better understanding of the potentially beneficial, symbiotic relationship between low oxygen and low temperatures may one day lead to the development of improved techniques for extending the shelf life of human organs for transplantation, Roth said.

"We have found that extension of survival limits in the cold is possible if oxygen consumption is first diminished," he said. "Our experiments in yeast and nematodes suggest that organs may last longer outside the body if their oxygen consumption is first reduced before they are made cold."

Roth's laboratory studies the potential clinical benefits of metabolic flexibility – from anoxia-induced reversible suspended animation to metabolic hibernation brought on by exposure to agents such as hydrogen sulfide. The ultimate goal of this work is to find ways to temporarily lower metabolism – like dialing down a dimmer switch on a lamp – as a means to "buy time" for patients in trauma situations, such as victims of heart attack or blood-loss injury, by reducing their need for oxygen until definitive medical care can be given.

Roth first got the idea to study the link between anoxia-induced suspended animation and hypothermia from documented cases in which humans have managed to make complete recoveries after apparently freezing to death. Widely publicized cases include Canadian toddler Erica Nordby, who in the winter of 2001 wandered outside clad only in a diaper. Her heart had stopped beating for two hours and her body temperature had plummeted to 61 degrees Fahrenheit before she was discovered, rewarmed and resuscitated. Another incident that made headlines was that of a Japanese man, Mitsutaka Uchikoshi, who in 2006 fell asleep on a snowy

mountain and was found by rescuers 23 days later with a core body temperature of 71 degrees Fahrenheit. He, too, was resuscitated and made a full recovery.

"There are many examples in the scientific literature of humans who appear frozen to death. They have no heartbeat and are clinically dead. But they can be reanimated. Similarly, the organisms in my lab can be put into a state of reversible suspended animation through oxygen deprivation and other means. They appear dead but are not. We wondered if what was happening with the organisms in my laboratory was also happening in people like the toddler and the Japanese mountain climber. Before they got cold did they somehow manage to decrease their oxygen consumption? Is that what protected them? Our work in nematodes and yeast suggests that this may be the case, and it may bring us a step closer to understanding what happens to people who appear to freeze to death but can be reanimated," Roth said.

The mechanism by which anoxia-induced suspended animation protects against extreme cold has to do with preventing the cascade of events that lead to biological instability and, ultimately, death. For example, suspended animation preserves the integrity of cell-cycle control by preventing an organism's cells from dividing in an error-prone fashion. During suspended animation, the cell cycle is reversibly halted. Upon reanimation, the cycle resumes as normal.

"When an organism is suspended its biological processes cannot do anything wrong," Roth said. "Under conditions of extreme cold, sometimes that is the correct thing to be doing; when you can't do it right, don't do it at all."

The first author of the paper, Kin Chan, Ph.D., formerly a postdoctoral research associate in the Roth lab, is now with the Laboratory of Molecular Genetics in the National Institute of Environmental Health Sciences at the National Institutes of Health. The NIH and the National Science Foundation funded this research.

Virus infection may trigger unusual immune cells to attack nerves in multiple sclerosis

White blood cells with receptors for both virus and nerve proteins may assault nerves after fighting an infection

A virus infection can incite the body to attack its own nerve tissue by activating unusual, disease-fighting cells with receptors for both viral and nerve proteins. The dual-receptor observation suggests a way brain and spinal cord nerve damage might be triggered in susceptible young adults afflicted with multiple sclerosis (MS). University of Washington Department of Immunology scientists Qingyong "John" Ji, Antoine Perchellet, and Joan M. Goverman conducted the study, which was published June 6 in Nature Immunology.

This is thought to be the first study to reveal a mechanism for autoimmune disease that depends on destroyer immune cells expressing dual receptors for a normal protein made by the body and a pathogen.

Multiple sclerosis is one of many autoimmune disorders in which the body's lines of defense become misguided and start damaging normal tissue. In the case of multiple sclerosis, the protective sheath around major nerves -- the myelin -- in the brain and spinal cord disintegrates. Like a frayed electrical cord, the nerves no longer transmit a clear signal.

People with multiple sclerosis might lose their ability to see, walk, or use their arms, depending on which nerves are affected. The symptoms can appear, disappear, and re-appear. The disease is more common in women than in men.

In healthy people, the immune system is kept in check to tolerate the usual proteins and cells in the body, much like an eager watch dog is put on a leash and trained to ignore friends and neighbors, yet still protect the family.

"Autoimmunity is believed to arise from an accidental breakdown in this tolerance of the body's own proteins. This breakdown is triggered by something in the environment, most likely a pathogen," noted Goverman, professor and acting chair of immunology whose research concentrates on the origins of autoimmune disease. Her lab is studying mechanisms that maintain tolerance, as well as the "tripping" mechanisms that defeat it.

In their most recently published study, her research team genetically engineered mice that over-produce a certain type of white blood cell from a group known as killer T cells. The normal function of killer cells is to attack tumor cells or cells infected with viruses or other pathogens. These T cells have receptors that recognize specific proteins that infected cells display to them, much like holding up a target in a window.

The specific killer T cells examined in this study were CD8+ T cells. The Goverman lab engineered mice to over-produce CD8+cells that recognized myelin basic protein, a predominant protein in the myelin sheath that covers nerves. The major question investigated in the study was whether the genetically engineered mice would exhibit a disease that resembled multiple sclerosis.

The researchers infected the mice with a virus that has itself been engineered to produce myelin basic protein. This infection should activate the CD8+T cells to first attack the virally infected cells making myelin

basic protein to eliminate the virus, then kill other cells that make myelin basic protein to wrap around nerves. Killing those cells would destroy the myelin sheath.

As expected, the mice developed a multiple sclerosis-like disease. But the researchers were surprised when viruses lacking the myelin basic protein also triggered the disease.

Additional cross-breeding experiments revealed the existence of two receptors on a few of the CD8+T cells. These cells, engineered specifically to bind to myelin basic protein, also built their own receptors for viruses, and could recognize both. When exposed to cells infected with viruses, they would bind to and destroy them using one receptor. Geared up as if they were beserk, some of these double-agent cells then would head elsewhere to bind their other receptor to cells producing myelin basic protein and ruin the coats on nerve cells.

"These results," the authors noted, "demonstrate a role for dual-receptor cells in autoimmunity." The study also points to why a ubiquitous viral infection could leave most people without any lasting effects, but trigger autoimmunity in genetically predisposed individuals.

The findings open a new perspective on the proposal that multiple sclerosis is virally induced, despite the inability to detect infectious virus in the central nervous system of multiple sclerosis patients. Data from other studies show that CD8+T cells can cross the blood-brain barrier, and also that multiple sclerosis patients have more central nervous system protein-specific CD8+T cells, compared to healthy people.

In the dual-receptor model, the autoimmune activity against nerve protein can continue after the virus is wiped out. Multiple sclerosis patients usually have high levels of antibodies indicating past infectious from several common viruses, but a live virus associated with multiple sclerosis has not been consistently observed. Therefore, to date, no specific virus has been confirmed as a causative agent for multiple sclerosis.

The authors explained that it's possible that multiple viruses could influence susceptibility to multiple sclerosis. The ability of any particular virus to contribute to the disease could depend on an individual's own repertoire of other predisposing genes, exposure to other predisposing environmental factors, and the random chance that T cells had been generated that recognize a myelin protein and a pathogen.

Receptors on T cells are randomly generated during their development. This observation helps explain why multiple sclerosis is partly a matter of chance. Some people with a genetic predisposition and environmental exposure develop the disease, while others with similar genetic predisposition and environmental exposure do not.

It's uncertain how common these dual-receptor T cells are, according to the researchers, although there are reports that up to one-third of human T cells express dual receptors. Goverman and her group plan to test samples from multiple sclerosis patients and see how many have dual-receptor T-cells.

A grant from the National Institutes of Health supported the study.

Dingoes, like wolves, are smarter than pet dogs

June 11, 2010 **by Lin Edwards**

PhysOrg.com - Studies in the past have shown that wolves are smarter than domesticated dogs when it comes to solving spatial problems, and now new research has shown that dingoes also solve the problems well.

The dingo is considered a “pure” prehistoric dog, which was brought to Australia tens of thousands of years ago by the Aborigines. While they have in the past been associated with humans, they have adapted to surviving “wild” in the Australian outback. The dingo lies somewhere between the wolf, its ancient ancestor, and the domestic or pet dog, and has cognitive differences between the two. There has been little research done on dingoes, even though studies would aid in the understanding of the evolution of dogs, and it was unknown whether the dingo was more “wolf-like” or “dog-like”.

Researchers in South Australia have now subjected the Australian dingo (Canis dingo) to the classic “detour task,” which has been used by previous researchers to assess the abilities of wolves (Canis lupus) and domestic dogs (Canis familiaris) to solve non-social, spatial problems.

The detour task involves placing a treat behind a transparent or wire mesh fence. The dog can see the food but cannot get to it directly and has to find its way along the fence and through a door and then double back to get the food. Previous research has shown wolves are adept at solving the problem quickly, while domesticated dogs generally perform poorly and fail to improve significantly even after repeated trials. The wolves were also able to adapt easily when conditions were reversed, but pet dogs also generally fared poorly at this task.

Until now dingoes had not been tested, so lead researcher, PhD student Mr. Bradley Smith of the School of Psychology at the University of South Australia, decided to subject 20 sanctuary-raised dingoes (Canis dingo) to the V-shaped detour task, in which a V-shaped fence is the barrier to the treat (a bowl of food) placed at the intersection point of the V, and the detour doors swung either inward or outward.

The dingoes were randomly assigned to one of four experimental conditions previously used to test dogs and wolves. These were the inward or outward detour (with doors closed), inward detour (with doors open), and

inward detour (with a human demonstrator). Each dingo was tested four times and then given a fifth trial with the conditions reversed.

The results showed the dingoes completed the detour tasks successfully, and they achieved fewer errors and solved the problems more quickly (in around 20 seconds) than domestic dogs tested in previous research. Unlike domesticated dogs in previous studies, the dingoes did not look to humans for help, and only one dingo even looked at the human when solving the problem. This behavior was much more similar to findings with wolves than for pet dogs.

The findings were published in the journal Animal Behaviour. All tests were carried out at the Dingo Discovery Centre in Victoria. More information: References: http://dx.doi.org/10.1016/j.anbehav.2010.04.017 *http://courses.media.mit.edu/2003spring/mas963/sociallearningdogs.pdf* *http://dx.doi.org/10.1006/anbe.2001.1866*

Tiny insect brains capable of huge feats

Insects may have tiny brains the size of a pinhead, but the latest research from the University of Adelaide shows just how clever they really are. For the first time, researchers from the University's Discipline of Physiology have worked out how insects judge the speed of moving objects.

It appears that insect brain cells have additional mechanisms which can calculate how to make a controlled landing on a flower or reach a food source. This ability only works in a natural setting.

In a paper published in the international journal Current Biology, lead author David O'Carroll says insects have well identified brain cells dedicated to analysing visual motion, which are very similar to humans.

"It was previously not understood how a tiny insect brain could use multiple brain pathways to judge motion," Associate Professor O'Carroll says. "We have known for many years that they can estimate the direction of moving objects but until now we have not known how they judge speed like other animals, including humans.

"It appears they take into account different light patterns in nature, such as a foggy morning or a sunny day, and their brain cells adapt accordingly.

"This mechanism in their brain enables them to distinguish moving objects in a wide variety of natural settings. It also highlights the fact that single neurons can exhibit extremely complex behaviour."

Assoc. Prof. O'Carroll co-authored the paper with Paul Barnett, a Physiology PhD student at the University of Adelaide, and Dr Karin Nördstrom, a former Physiology Postdoctoral Fellow at Adelaide who is now based at Uppsala University in Sweden. Their specific research is focused on how the brain makes sense of the world viewed by the eye, using the insect visual system as an important model.

"Insects are ideal for our research because their visual system accounts for as much as 30% of their mass, far more than most other animals," Assoc. Prof. O'Carroll says.

His team is collaborating with industry to develop artificial eyes in robots, mimicking human and insect vision.

World's first plastic antibody works in mice

11:07 11 June 2010 **by Andy Coghlan**

Antibodies made entirely from plastic have saved the lives of mice injected with bee venom – the first time such a strategy has worked in live animals.

Researchers developing the antibodies say it is the first step towards customised antibodies for a host of other medical applications, from treating people who have been poisoned to combating infection.

Natural antibodies are made by the body's immune system to lock onto a specific "antigen". Likewise, the plastic antibodies contain cavities moulded in exactly the right shape to capture target molecules, in this case, melittin – the active agent in bee venom.

Faking it

Kenneth Shea of the University of California at Irvine led the team which made melittin antibodies through a process called molecular imprinting. They used a catalyst to stimulate polymers to form around molecules of bee venom, then dissolved away the venom itself, leaving empty cavities with the exact shape to trap melittin.

Shea injected these tiny plastic nanoparticles into mice 20 seconds after they'd been injected with bee venom, 60 per cent survived whereas all the untreated mice died. The plastic antibodies were then destroyed by the liver. "We conclude that imprinted polymer nanoparticles efficiently capture melittin in the bloodstream," say Shea and his colleagues in the paper.

"We see this as a very significant paper, and the first demonstration in living things of these materials, effectively using them as a drug," says Mike Whitcombe, whose lab at Cranfield University in the UK develops imprinted polymers and runs a database of the polymers available.

Plastic copycats

Philipp Holliger of the Laboratory of Molecular Biology in Cambridge, UK, said that the plastic antibodies do perform some of the functions of natural antibodies – capturing toxins and sending them to the liver for

destruction. "These properties should make them attractive alternatives to antibodies in antidote anti-toxin treatments," he says.

However, Holliger doubts whether they could perform other important functions of natural antibodies, such as priming the body's immune system to fight future infections. Unlike natural antibodies, they are not equipped to communicate with other cells and components of the immune system.

Journal reference: Journal of the American Chemical Society, DOI: 10.1021/ja102148f

Hayabusa asteroid probe faces moment of truth

* 16:10 11 June 2010 **by Wendy Zukerman**

Following a mishap during its rendezvous with an asteroid in 2005, and other technical problems, the Japanese spacecraft Hayabusa was all but given up for dead. Miraculously it has been nursed back to life, and it is now hurtling towards Earth, on track to hurl a small but potentially momentous capsule into the Australian outback on Sunday, 13 June.

No one knows whether Hayabusa managed to grab a dust sample when it landed on the asteroid Itokawa. If it did, this would be a first. Even more significantly, the dust will give us clues as to how the Earth formed, as well as insights into how to prevent it being destroyed by an asteroid impact. Find out more in this New Scientist briefing.

Where is Hayabusa now?

At 0900 Japan time today (11 June) the Japan Aerospace Exploration Agency (JAXA) announced that Hayabusa was 1,142,550 kilometres from home, following its fourth and final "trajectory correction manoeuvre" on Wednesday. This nudged Hayabusa into the position needed for an 18-kilogram capsule - hopefully containing a sample of asteroid soil – to land at the intended target, the Woomera test range in South Australia.

How will the Hayabusa land?

Three hours before re-entry, when the spacecraft is 40,000 kilometres from Earth, Hayabusa will release its sample recovery capsule. The capsule will then enter the atmosphere as a fireball, experiencing temperatures of up to 3000 °C, says Hitoshi Kuninaka, a senior member of the Hayabusa project at JAXA.

At an altitude of 10 kilometres, the capsule will deploy a parachute and an antenna that will be used to track it as it lands at Woomera. Once the landing point is confirmed, the capsule will be collected and airfreighted to Japan. Only when it has reached the Sagamihara Curation Center near Tokyo will we know what, if anything, is inside.

What went wrong on Itokawa?

When Hayabusa landed on the asteroid in 2005, it was supposed to fire two metal pellets onto its surface, pushing up material into the cone-shaped sampling capsule for collection. But just after landing, the fuel thruster leaked, causing the spacecraft to enter a "safe" mode, in which all unnecessary systems were shut down.

Given the malfunction, how could the capsule contain any dust?

Because gravity on Itokawa is extremely weak, Hayabusa is likely to have puffed up a plume of dust when it landed. Kuninaka is hopeful that some of this may have entered the capsule, though he admits there is no evidence this has happened.

Michael Zolensky, curator of stratospheric dust at the Astromaterials Research and Exploration Science directorate at NASA Johnson Space Centre in Houston, Texas, also predicts that there is something interesting inside the capsule. Even microscopic samples could be "sliced up" and analysed to give information about the chemical composition of asteroids, he says.

What will we learn from any samples that have been brought back?

"Asteroids are a threat to civilisation," says Zolensky. Understanding their chemical composition and physical properties could help "mitigate that hazard", he says.

Trevor Ireland, an earth chemist at the Australian National University in Canberra, Australia, who will be analysing the Hayabusa samples, says that combining analyses of samples from Itokawa with knowledge about solar winds and cosmic radiation might reveal details about the early history of our solar system and the planets.

Unlike meteorites and cosmic dust that have fallen onto the Earth's surface, samples from Hayabusa will not be contaminated by terrestrial materials.

Strain of bacteria discovered; could aid in oil spill, other environmental cleanup

Corvallis, Ore. – Researchers have discovered a new strain of bacteria that can produce non-toxic, comparatively inexpensive “rhamnolipids,” and effectively help degrade polycyclic aromatic hydrocarbons, or PAHs – environmental pollutants that are one of the most harmful aspects of oil spills.

Because of its unique characteristics, this new bacterial strain could be of considerable value in the long-term cleanup of the massive Gulf Coast oil spill, scientists say.

More research to further reduce costs and scale up production would be needed before its commercial use, they added.

The findings on this new bacterial strain that degrades the PAHs in oil and other hydrocarbons were just published in a professional journal, Biotechnology Advances, by researchers from Oregon State University and two collaborating universities in China. OSU is filing for a patent on the discovery. "PAHs are a widespread group of toxic, carcinogenic and mutagenic compounds, but also one of the biggest concerns about oil spills," said Xihou Yin, a research assistant professor in the OSU College of Pharmacy.

"Some of the most toxic aspects of oil to fish, wildlife and humans are from PAHs," Yin said. "They can cause cancer, suppress immune system function, cause reproductive problems, nervous system effects and other health issues. This particular strain of bacteria appears to break up and degrade PAHs better than other approaches we have available."

The discovery is strain "NY3" of a common bacteria that has been known of for decades, called Pseudomonas aeruginosa. It was isolated from a site in Shaanxi Province in China, where soils had been contaminated by oil.

P. aeruginosa is widespread in the environment and can cause serious infections, but usually in people with health problems or compromised immune systems. However, some strains also have useful properties, including the ability to produce a group of "biosurfactants" called rhamnolipids.

A "surfactant," technically, is a type of wetting agent that lowers surface tension between liquids – but we recognize surfactants more commonly in such products as dishwashing detergent or shampoo. Biosurfactants are produced by living cells such as bacteria, fungi and yeast, and are generally non-toxic, environmentally benign and biodegradable. By comparison, chemical surfactants, which are usually derived from petroleum, are commonly toxic to health and ecosystems, and resist complete degradation.

Biosurfactants of various types are already used in a wide range of applications, from food processing to productions of paints, cosmetics, household products and pharmaceuticals. But they also have uses in decontamination of water and soils, with abilities to degrade such toxic compounds as heavy metals, carcinogenic pesticides and hydrocarbons.

Although the type of biosurfactant called "rhamnolipids" have been used for many years, the newly discovered strain, NY3, stands out for some important reasons. Researchers said in the new study that it has an "extraordinary capacity" to produce rhamnolipids that could help break down oil, and then degrade some of its most serious toxic compounds, the PAHs.

Rhamnolipids are not toxic to microbial flora, human beings and animals, and they are completely biodegradable. These are compelling advantages over their synthetic chemical counterparts made from petroleum. Even at a very low concentration, rhamnolipids could remarkably increase the mobility, solubility and bioavailability of PAHs, and strain NY3 of P. aeruginosa has a strong capability of then degrading and decontaminating the PAHs.

"The real bottleneck to replacing synthetic chemicals with biosurfactants like rhamnolipid is the high cost of production," Yin said. "Most of the strains of P. aeruginosa now being used have a low yield of rhamnolipid. But strain NY3 has been optimized to produce a very high yield of 12 grams per liter, from initial production levels of 20 milligrams per liter."

By using low-cost sources of carbon or genetic engineering techniques, it may be possible to reduce costs even further and scale up production at very cost-effective levels, researchers said.

The rhamnolipids produced by NY3 strain appear to be stable in a wide range of temperature, pH and salinity conditions, and strain NY3 aggressively and efficiently degrades at least five PAH compounds of concern, the study showed. It's easy to grow and cultivate in many routine laboratory media, and might be available for commercial use in a fairly short time. Further support to develop the technology is going to be sought from the National Science Foundation.

"Compared to their chemically synthesized counterparts, microbial surfactants show great potential for useful activity with less environmental risk," the researchers wrote in their report. "The search for safe and efficient methods to remove environmental pollutants is a major impetus in the search for novel biosurfactant-producing and PAH-degrading microorganisms."

Collaborating on this research were scientists from Xi'an University of Architecture and Technology and Nanjing Agricultural University in China.

The Reproductive Revolution: How Women Are Changing the Planet's Future

The population bomb is being defused. It is being done without draconian measures by big government, without crackdowns on our liberties--by women making their own choices.

By Fred Pearce

Aisha, Miriam and Akhi are three young factory workers in Dhaka, the capital of Bangladesh. They are poorly educated and badly paid. But, like millions of other young women, they relish their freedom from the stultifying conformity of rural life, where women are at the constant beck and call of fathers, brothers and husbands.

There is something else. The three women together have 22 siblings. But Aisha plans three children, Miriam two and Akhi just one. They represent a gender revolution that many see as irrevocably tied to a reproductive revolution. Together, the changes are solving what once seemed the most difficult problem facing the future of humanity: growing population.

Almost without anyone noticing, the population bomb is being defused. It is being done without draconian measures by big government, without crackdowns on our liberties - by women making their own choices.

Family planning experts used to say that women only started having fewer children when they got educated or escaped poverty. Pessimists feared that if rising population prevented the world's poor from advancing, they would get caught in a cycle of poverty and large families. The poverty trap would become a demographic trap.

But the reality is proving very different. Round the world, women today are having half as many children as their mothers did. And often it is the poorest and least educated women who are in the vanguard. Women like Aisha, Miriam and Akhi.

There are holdouts, in parts of the Middle East and rural Africa. But more than 60 countries - containing approaching half of the world's population - already have fertility rates at or below the rate needed to maintain their populations long-term. The club now includes most of the Caribbean islands, Japan, South Korea, China, Thailand, Sri Lanka, Iran, Turkey, Vietnam, Brazil, Algeria, Kazakhstan and Tunisia. Within 20 years, demographic giants like Indonesia, Bangladesh, Mexico and India will in all probability also have below-replacement fertility.

How is this happening? For one thing, more and more women are leading independent working lives, rather than succumbing to a life of child bearing and raising. In many countries, women are staying single through their 20s and beyond. In 1960, two thirds of American women in their early 20s were already married; today the figure is less than a quarter. The habit is spreading fast. As recently as 1980, a UN study found that nuptials were "near universal" across Asia, and half of Asian women were married by the age of 18. No more. In Japan, half of all 30-year-old women today are unmarried. In South Korea, the figure is 40 per cent.

The trend is especially marked in cities. In Bangkok, a fifth of all women are single at 45. Manila, Singapore and Hong Kong are not far behind. And, again largely unremarked, it is women who are heading the urbanization of the planet. Whether sweatshop workers in Dhaka, bar girls in Bangkok, office workers in Shanghai, students in Delhi or maids in Caracas, there are more young women than men in almost every city in the world.

Academics debate whether they are there for the jobs or the men (there are more rich educated men in cities, but more women of all sorts). But whatever the motive, they are there: in the bars, shoe shops, gyms and clubs. With jobs but often no dependants. In Japan, they get called wagamama, or "single parasites." No matter; it's better than changing daipers.

How have they gained their freedom? Some say liberation allowed women to make new choices about their lives. Equally, however, it has been the dramatic improvement in the survival rate of infants that for the first time has freed women from the social obligation for a lifetime producing and rearing babies.

Women are having smaller families and grabbing a new life outside the home because, for the first time in history, they can. In the 20th century, the world largely eradicated the diseases that used to mean most children died before growing up. Mothers no longer need to have five or six children to ensure the next generation. So they do not. Two or three is enough.

Rich or poor, educated or illiterate, socialist or capitalist, Muslim or Catholic, secular or devout, with tough government birth control policies or none, most - most families - tell the same story.

Scottish sociologist John MacInnes at the University of Edinburgh calls this the reproductive revolution. Until the 18th century, half of children died before entering their fertile years, and many more before they completed them. Most women spent almost all their (often rather short) adult lives bearing and rearing children.

The patrician societies that have dominated the world for millennia were designed to ensure women fulfilled this role. The regulation of child production was done "through church and state, the norms surrounding sexual

activity and sex roles, illegitimacy, cohabitation and marriage, family and kinship obligations and property law," says MacInnes.

In much of the world, the drive to maintain fertility institutionalized arranged marriages, often of very young girls, maintained brutal sanctions against female adulterers or girls who would not accept their lot, and ostracized any form of homosexuality.

The reproductive revolution is kicking all this away, because it is simply no longer needed to sustain populations. Feminism is not a new idea. And some individuals have always broken free. But, for most women, the reproductive revolution has "taken it from the realm of utopia to practical possibility," says MacInnes.

The global collapse in fertility rates and rise of feminism is not the slow diffusion of a new idea, or a mechanistic response to aid workers handing out condoms. This is the breaking of a logjam. The logjam of a patriarchy that has suddenly lost its purpose.

British demographer Tim Dyson of the London School of Economics sees the change even in rural India. India still adds about 19 million people to its population each year - a quarter of total global growth. But its fertility is falling fast, now averaging 2.8 children per woman. Dyson remembers that back in the 1960s, sociologists compared women's lives in India and the U.S. "In India they married at 17, had seven kids, the last one at 43 years old, and died typically at 46. In America, women typically married at 18, had two kids quickly and then more than 40 years of life after childbearing. Now, Indian women are grabbing that life too. Sterilization is the main form of contraception in India, and the average age of sterilization is 26 years old."

Where is this taking us? Through most of history, women have had between five and eight live births each. For a while after childhood death rates fell, they continued to do so. That's why world population quadrupled in the 20th century. But today the global average is 2.6 births, half the figure even a generation ago. The figure is falling fast towards the current global replacement rate which, allowing for girls who do not reach adulthood, is 2.3 children per woman.

Smaller families do not immediately cut population growth. There is rising life expectancy to account for, along with a legacy of 20th-century baby boomers who remain fertile. But the trend is clear. And in the past 40 years, the world's population growth rate has fallen from 2.1 per cent to below 1.2 per cent.

Percentages are not the same as absolute numbers, of course. But since 1987, the number of additional people on the planet each year has fallen from 87 million to 78 million. The downward trend will accelerate. Growth may, on current trends, be zero or even negative by mid-century. Some countries are already shrinking. In 2008 there were 26 of them, headed by Russia, Montenegro, Bulgaria, Zimbabwe, Ukraine, Latvia and Swaziland. Others are only sustained by inward migration.

It took around 130 years, from about 1800 to 1927, for the world to get from one billion people to two billion, but only another 33 years to reach three billion, which happened in 1960. Reaching four billion took just 15 years to 1975. The fifth billion came in 12 years, in 1987, as did the next billion, achieved in 1999.

The next billion people will take a little longer than the last, probably 14 years if we reach seven billion as expected in 2013. And getting to eight billion will take another 20 or more years. And we may never reach 9 billion.

Peak population is probably much closer than most people think. Virtually all countries that have brought their fertility rates down from five, to four and three have gone on down below the replacement level. Much of Europe is below 1.5. By 2100, on current fertility trends, Germany could have fewer natives than today’s Berlin, and Italy’s population could crash from 58 million to just 8 million.

Once a negative trend has set in, it may prove very hard to break. As well as having ever fewer potential mothers, societies may get out of the habit of having babies. Children will be rare, exotic and unusual. We can see this already. Only a few years ago, going to a cafe in Italy would see you surrounded by noisy children. Now you will likely only see adults, including many young latte-sipping men and women who would once have been surrounded by kids.

Other repercussions of the baby bust will play out over the coming decades. One of the most controversial is the rising tide of migration. It is created in part by the income differentials round the world, but even more by current record differences in fertility. When women in some countries have more than six children, while others have barely more than one, trading people is an obvious safety valve for both sides. Europe and North America already badly need foreign hands to keep societies and economies functioning. We should stop pretending otherwise.

The other critical change is aging. As fertility falls, the world is becoming older. Our species has never lived in societies where there were more old people than children. But soon we will. This is truly terra incognita.

Some say we will never be able to afford to look after all those old people. But there is, to coin a phrase, a silver lining. The old are human capital, sources of wisdom and experience. We have to harness that capital better. The old will have to work longer - but they will also expect to be valued more.

Take health. All the things I have been talking about have happened because the 20th century eradicated the killer diseases that wiped out most children before they could grow up. But in the 21st century there is a new priority - to help the old stay fitter for longer. So they can have better lives, of course. But also so they can contribute more.

We are coming through the greatest surge of human population numbers in history. It has already changed us profoundly and the end game will change us even more. The reproductive revolution unleashed huge forces of economic activity, social dislocation and liberation - for women in particular. But well before the end of this century, Homo sapiens - the brash, go-getting, hormone-driven, young naked ape of the 20th century - will be older and will likely be more conservative, less innovative, more boring even. But perhaps also wiser, less frenetic, and more caring of each other and the environment. Older, wiser, greener.

The tribal elders may take center stage once more. But this time they will not just be revered, they will be the largest group in society. And in all the probability, they will be dominated by women. Demography is destiny. There is no going back.

"Buddha remains" unveiled in east China temple

NANJING, (Xinhua) -- Chinese Buddhist monks and archaeologists revealed what they believed to be top part of the skull of Sakyamuni, the founder of Buddhism, Saturday morning in east China's Jiangsu Province. The object, taken out for the first time around 9 a.m. from a miniature gold coffin nestled inside a silver one, was part of Buddha's parietal bone, said Master Chuan Yin, president of the Buddhist Association of China, after attending the worshipping ceremony held in Qixia Temple in Nanjing, capital of Jiangsu Province.

The bone, irregular and light brown, looked like a small rock. "It is full of cell-like cavities, just like a honeycomb," said Hua Guorong, deputy head of Nanjing City Museum.

"Our findings conform with the descriptions of the parietal bone in historical records," said Master Xue Cheng, vice president of the association, adding the bone was hugely sacred for Buddhists.

Besides Sakyamuni's remains, ten sacred pieces of remains of other Buddhas were also found in another gold and silver mini-coffin. All the relics had been enshrined at Qixia Temple by 108 eminent Buddhist monks from the Chinese mainland, Macao and Taiwan. The relics would be open to believers at the temple for one month, Hua said.

To ensure the safety of the invaluable treasures, Saturday's activities were conducted under heavy security, as well the indoor temperature was kept stable at 20 degrees Celsius and humidity between 55 to 60 percent, he said.

The parietal bone of Sakyamuni, allegedly recovered from the cremation ash of Sakyamuni, had been stored in a miniature pagoda named the Pagoda of King Asoka unearthed two years ago in an underground shrine built in 1011 under the former Changgan Temple of Nanjing.

The palace was found when archaeologists began excavating the ruins of the Grand Bao'en Temple of Nanjing built in the Ming Dynasty (1368-1644 AD).

In July 2008, archaeologists found a stele in the palace, the inscription on it said the palace preserved a "Seven-Treasure Pagoda of King Asoka" containing gold and silver coffins with Sakyamuni's parietal bone and relics of other Buddhas inside. One month later, an iron case containing a pagoda was unearthed from the palace. In November 2008, archaeologists removed the pagoda from the case and found two mini-coffins.

It is said that 2,500 years ago, Sakyamuni's disciples recovered one parietal bone, four teeth, two collar bones and 84,000 particles of relics from the cremation ash of Sakyamuni, according to Lu Jianfu, a senior official with the association.

Asoka, an Indian emperor (273 BC - 232 BC), allegedly collected all the parts of Sakyamuni's remains, stored them in pagoda-like shrines, and sent them to different parts of the world. The pagoda in Nanjing is believed to be one of tens of thousands of "pagodas of King Asoka" that contain Sakyamuni's remains.

The four-layer, 1.21-m-high and 0.42-m-wide pagoda is allegedly the largest of its kind unearthed in China.

According to Tang Dynasty (618-907) Buddhist records, China had 19 pagodas of King Asoka holding Sakyamuni's relics. To date, it is believed seven of the pagodas have been found in different parts of the country.

Editor: Bi Mingxin

A Decade Later, Genetic Map Yields Few New Cures

By NICHOLAS WADE

Ten years after President Bill Clinton announced that the first draft of the human genome was complete, medicine has yet to see any large part of the promised benefits.

For biologists, the genome has yielded one insightful surprise after another. But the primary goal of the $3 billion Human Genome Project - to ferret out the genetic roots of common diseases like cancer and Alzheimer's and then generate treatments - remains largely elusive. Indeed, after 10 years of effort, geneticists are almost back to square one in knowing where to look for the roots of common disease.

One sign of the genome's limited use for medicine so far was a recent test of genetic predictions for heart disease. A medical team led by Nina P. Paynter of Brigham and Women's Hospital in Boston collected 101 genetic variants that had been statistically linked to heart disease in various genome-scanning studies. But the variants turned out to have no value in forecasting disease among 19,000 women who had been followed for 12 years.

The old-fashioned method of taking a family history was a better guide, Dr. Paynter reported this February in The Journal of the American Medical Association.

In announcing on June 26, 2000, that the first draft of the human genome had been achieved, Mr. Clinton said it would "revolutionize the diagnosis, prevention and treatment of most, if not all, human diseases."

At a news conference, Francis Collins, then the director of the genome agency at the National Institutes of Health, said that genetic diagnosis of diseases would be accomplished in 10 years and that treatments would start to roll out perhaps five years after that.

"Over the longer term, perhaps in another 15 or 20 years," he added, "you will see a complete transformation in therapeutic medicine."

The pharmaceutical industry has spent billions of dollars to reap genomic secrets and is starting to bring several genome-guided drugs to market. While drug companies continue to pour huge amounts of money into genome research, it has become clear that the genetics of most diseases are more complex than anticipated and that it will take many more years before new treatments may be able to transform medicine.

"Genomics is a way to do science, not medicine," said Harold Varmus, president of the Memorial Sloan-Kettering Cancer Center in New York, who in July will become the director of the National Cancer Institute.

The last decade has brought a flood of discoveries of disease-causing mutations in the human genome. But with most diseases, the findings have explained only a small part of the risk of getting the disease. And many of the genetic variants linked to diseases, some scientists have begun to fear, could be statistical illusions.

The Human Genome Project was started in 1989 with the goal of sequencing, or identifying, all three billion chemical units in the human genetic instruction set, finding the genetic roots of disease and then developing treatments. With the sequence in hand, the next step was to identify the genetic variants that increase the risk for common diseases like cancer and diabetes.

It was far too expensive at that time to think of sequencing patients' whole genomes. So the National Institutes of Health embraced the idea for a clever shortcut, that of looking just at sites on the genome where many people have a variant DNA unit. But that shortcut appears to have been less than successful.

The theory behind the shortcut was that since the major diseases are common, so too would be the genetic variants that caused them. Natural selection keeps the human genome free of variants that damage health before children are grown, the theory held, but fails against variants that strike later in life, allowing them to become quite common. In 2002 the National Institutes of Health started a $138 million project called the HapMap to catalog the common variants in European, East Asian and African genomes.

With the catalog in hand, the second stage was to see if any of the variants were more common in the patients with a given disease than in healthy people. These studies required large numbers of patients and cost several million dollars apiece. Nearly 400 of them had been completed by 2009. The upshot is that hundreds of common genetic variants have now been statistically linked with various diseases.

But with most diseases, the common variants have turned out to explain just a fraction of the genetic risk. It now seems more likely that each common disease is mostly caused by large numbers of rare variants, ones too rare to have been cataloged by the HapMap.

Defenders of the HapMap and genome-wide association studies say that the approach made sense because it is only now becoming cheap enough to look for rare variants, and that many common variants do have roles in diseases.

At this point, some 850 sites on the genome, most of them near genes, have been implicated in common diseases, said Eric S. Lander, director of the Broad Institute in Cambridge, Mass., and a leader of the HapMap project. "So I feel strongly that the hypothesis has been vindicated," he said.

But most of the sites linked with diseases are not in genes - the stretches of DNA that tell the cell to make proteins - and have no known biological function, leading some geneticists to suspect that the associations are spurious.

Many of them may “stem from factors other than a true association with disease risk,” wrote Jon McClellan and Mary-Claire King, geneticists at the University of Washington, Seattle, in the April 16 issue of the journal Cell. The new switch among geneticists to seeing rare variants as the major cause of common disease is “a major paradigm shift in human genetics,” they wrote.

The only way to find rare genetic variations is to sequence a person’s whole genome, or at least all of its gene-coding regions. That approach is now becoming feasible because the cost of sequencing has plummeted, from about $500 million for the first human genome completed in 2003 to costs of $5,000 to $10,000 that are expected next year.

But while 10 years of the genome may have produced little for medicine, the story for basic science has been quite different. Research on the genome has transformed biology, producing a steady string of surprises. First was the discovery that the number of human genes is astonishingly small compared with those of lower animals like the laboratory roundworm and fruit fly. The barely visible roundworm needs 20,000 genes that make proteins, the working parts of cells, whereas humans, apparently so much higher on the evolutionary scale, seem to have only 21,000 protein-coding genes.

The slowly emerging explanation is that humans and other animals have much the same set of protein-coding genes, but the human set is regulated in a much more complicated way, through elaborate use of DNA’s companion molecule, RNA.

Little, if any, of this research could have been done without having the human genome sequence available. Every gene and control element can now be mapped to its correct site on the genome, enabling all the working parts of the system to be related to one another.

“Having a common scaffold on which one can put all the information has dramatically accelerated progress,” Dr. Lander said.

The genome sequence has also inspired many powerful new techniques for exploring its meaning. One is chip sequencing, which gives researchers access to the mysterious and essential chromatin, the complex protein machinery that both packages the DNA of the genome and controls access to it.

The data from the HapMap has also enabled population geneticists to reconstruct human population history since the dispersal from Africa some 50,000 years ago. They can pinpoint which genes bear the fingerprints of recent natural selection, which in turn reveals the particular challenges to which the populations on different continents have had to adapt.

As more people have their entire genomes decoded, the roots of genetic disease may eventually be understood, but at this point there is no guarantee that treatments will follow. If each common disease is caused by a host of rare genetic variants, it may not be susceptible to drugs.

“The only intellectually honest answer is that there’s no way to know,” Dr. Lander said. “One can prefer to be an optimist or a pessimist, but the best approach is to be an empiricist.”