http://www.eurekalert.org/pub_releases/2011-08/ghri-cih080611.php

Common irregular heartbeat raises risk of dementia

Group Health study finds higher dementia risk with atrial fibrillation

SEATTLE, WA—The most common kind of chronically irregular heartbeat, known as atrial fibrillation, is associated with a greater risk of dementia, including Alzheimer's disease. This discovery by scientists at Group Health Research Institute and their collaborators was published online in advance of print on August 1 in the Journal of the American Geriatrics Society.

"Both atrial fibrillation and dementia increase with age," said Sascha Dublin, MD, PhD, a Group Health Research Institute assistant investigator who led the research. "Before our prospective cohort study, we knew that atrial fibrillation can cause stroke, which can lead to dementia. Now we've learned that atrial fibrillation may increase dementia risk in other, more subtle ways as well."

The results of Dr. Dublin's study suggest a relationship between atrial fibrillation and dementia beyond the connection through stroke. The people in the study had a mean age of 74 years when the study began. None had dementia or a history of stroke. At the beginning of the study, 4.3 percent had atrial fibrillation, and an additional 12.2 percent developed it during the study. In the course of the study, 18.8 percent developed some type of dementia. People with atrial fibrillation were more likely to have other cardiovascular risk factors and disease than were those without the condition. So the researchers looked to see if atrial fibrillation increased dementia risk more than just through its association with other kinds of heart disease.

Participants were followed for an average of seven years. Over this time, those with atrial fibrillation had a 40 percent to 50 percent higher risk of developing dementia of any type, including probable Alzheimer's disease, compared to those without atrial fibrillation. This was true even for people who did not also have a stroke during the follow-up period.

The research was part of Adult Changes in Thought (ACT), an ongoing joint project of the Group Health and University of Washington studying risk factors for dementia in older adults. Started in 1994 ACT is led by Dr. Dublin's co-author Group Health Vice President for Research and Group Health Research Institute Executive Director Eric B. Larson, MD, MPH. ACT focuses on finding ways to delay or prevent dementia, including Alzheimer's disease, and declines in memory and thinking. It aims to deepen understanding of how the body—especially the brain—ages. ACT participants are members of Group Health Cooperative, a nonprofit health care system in the U.S. Pacific Northwest.

Dr. Dublin's study, which ran from 1994 to 2008, followed 3,045 people. The researchers relied on Group Health's advanced electronic data systems to determine whether participants had atrial fibrillation. The cognitive function of all study participants was evaluated every two years with tests and interviews as part of ACT. Patients whose ACT tests indicated possible dementia had additional tests including physical, neurological, and psychological exams, and many also had brain scans. A panel of experts determined the correct diagnosis for patients with cognitive problems.

Atrial fibrillation affects 3 million Americans. Dr. Dublin says that some ways it might increase dementia risk are:

* weakening the heart's pumping ability, leading to less oxygen going to the brain;
* increasing the chance of tiny blood clots going to the brain, causing small, clinically undetected strokes;
* a combination of these plus other factors that contribute to dementia such as inflammation.

Dr. Dublin said an important next step is studying whether any treatments for atrial fibrillation reduce the risk of developing dementia. The researchers also hope their results reach primary care providers, who are often the main doctors caring for people with atrial fibrillation, dementia, or both.

"Right now, we think we are protecting our patients' brains as long as they don't have a stroke, but tiny insults over time can add up," said Dr. Dublin, who is a primary care physician at Group Health. "This paper is a wakeup call, telling us that we need to learn more about how to protect brain function, while continuing to give patients with atrial fibrillation the best possible care."

The National Institute on Aging, part of the National Institutes of Health, funded this study.

Drs. Dublin and Larson's co-authors are Melissa L. Anderson, MS, Group Health Research Institute senior biostatistician; Sebastien J. Haneuse, PhD, who recently moved from Group Health Research Institute to Harvard School of Public Health; Group Health Research Institute Affiliate Investigators Susan R. Heckbert, MD, PhD, Paul K. Crane, MD, MPH, Linda Teri, PhD, and Susan M. McCurry, PhD, all of the University of Washington; Wayne McCormick, MD, MPH, of the University of Washington; James D. Bowen, MD, of the University of Washington and Swedish Neuroscience Institute; and John C. S. Breitner, MD, MPH, who recently moved from the University of Washington and Veterans Affairs Puget Sound Health Care System to McGill University, in Montreal, Quebec.

http://www.eurekalert.org/pub_releases/2011-08/uoh-wai080811.php

Walking around is the simplest way to shorten hospital stay

A new study from the University of Haifa has found that walking around the ward during hospitalization significantly reduces the length of the older patient's stay.

"Given the over-occupancy of many hospitals, this finding can be of great importance," the researchers stated.

Walking around the ward during hospitalization reduces the length of geriatric patients' stay in internal wards. This has been shown in a new study by Dr. Efrat Shadmi and Dr. Anna Zisberg of the University of Haifa's Department of Nursing, funded by the Israeli Science Foundation and published in the journal Archives of Internal Medicine.

The study surveyed 485 participants aged 70 and up, who were hospitalized for at least two days in the internal wards of a hospital in Israel. The participants' physical condition was examined by means of questionnaires and those who were confined to a bed or immobile were excluded from the study. Those who were not restricted in mobility were asked about their physical activity during the course of their hospitalization, and based on their answers were divided into two study groups: those who remained in bed or seated next to it and those who walked around their rooms and the ward.

The study found that all of the patients who walked around shortened their hospital stay by an average day and a half compared with those who did not exercise physical mobility. The study also found that those who walked around the ward on the first day of hospitalization shortened their stay more than the others. The researchers stated that they found this to be relevant regardless of the patients' health status.

According to the researchers, older patients might mistakenly believe that when they are hospitalized they must stay in bed. Studies of older adults have shown, however, that the opposite is true. "The muscle's reserve capacity' can decompose quite quickly in older people. If they shift from a mode of mobility – even if it was minimal – to a state of almost complete immobility, and even for just a few short days of hospitalization, they could very quickly lose their muscle 'reserves', resulting in more difficulties functioning and other complications. This study, along with other new studies in the area, shows that walking really does pay off," the researchers stated.

They also noted that the study results show that simple intervention to encourage walking in the geriatric internal wards ought to be seriously considered, so as to shorten the length of the geriatric patient's hospital stay. "Given the over-occupancy of many hospitals, this finding can be of great importance," they concluded.

http://news.discovery.com/space/earth-moon-life-tilt-110808.html

Earth May Not Have Needed Moon for Life

New simulations show that, even without a moon, the tilt of Earth's axis would be stable enough to support life.

By Irene Klotz | Mon Aug 8, 2011 08:38 AM ET

Scientists have long believed that without the moon's stabilizing gravitational influence, variations in Earth's tilt would have caused climate change too dynamic for complex life to evolve. Not so, concludes a new study that has implications for understanding conditions for life elsewhere in the solar system. The study sprang from the ongoing Kepler Telescope mission to find Earth-like planets circling in habitable zones around other stars in the Milky Way. "We were wondering 'Do we really have to find a moon or not?' around potentially habitable worlds, planetary scientist Jason Barnes, with the University of Idaho, told Discovery News.

Previous studies showed that without the steadying gravitational influence of a large moon, Earth's tilt would shift by as much as about 85 degrees every 100,000 years or so, alternatively freezing and baking the planet's poles. Scientists believe a stable climate spanning about 500,000 years was necessary for complex life to blossom on Earth. A new computational analysis, however, shows that a moonless Earth would still have swings in its tilt but the influence of Jupiter and other factors would limit the variations to about 10 degrees in either direction. Earth's rotational tilt varies between 0.5 and 1 degree about every 100,000 years.

"Plus- or minus-10 would certainly be noticeable and may be a problem, but I don't think it would prevent life from coming about," Barnes said.

"It's a very intriguing result. It's provocative," Richard Vondrak, lead scientist with NASA's ongoing Lunar Reconnaissance Orbiter mission, told Discovery News. "On the moon we can find important evidence and clues of what happened to not only to the moon, but also to the Earth-moon system over the last 4.5 billion years," said Vondrak, a planetary scientist with NASA's Goddard Space Flight Center in Greenbelt, Md.

The study also showed that if Earth revolved around the sun in the opposite direction, called a retrograde orbit, it wouldn't need a moon at all to have a climate about as stable as it has today. Likewise, a Jupiter about half the distance to Earth as its present location would have had a similar steadying hand, Barnes added.

The findings are causing extrasolar planet hunters to revise their thinking on what constitutes a habitable planet. "We think that at least 80 or 90 percent of planets out there statistically won't even require a moon" to have a stable climate, Barnes said.

Location is key. In our own solar system, Mars shows evidence of extreme climate change, the result, scientists believe, of a rotational tilt that flips between zero and 60 degrees over time. A big moon likely could have helped stabilize Mars' orbit, but the planet has just two small moons, most likely captured asteroids, that don't have much gravitational muscle.

Other factors impacting a planet's climate and suitability for life include its star's age, composition and the size and location of sibling planets in the system. "It's a very complex problem for sure and we're not anywhere near solving it, but we're making positive steps as we slowly evaluate each of these conditions and discover what constraints they really place on whether life can exist there," Barnes said.

The research appears in this month's Astrobiology magazine and is pending publication in the journal Icarus.

http://www.nasa.gov/topics/earth/features/tsunami-bergs.html

Tohoku Tsunami Created Icebergs In Antarctica

A NASA scientist and her colleagues were able to observe for the first time the power of an earthquake and tsunami to break off large icebergs a hemisphere away.

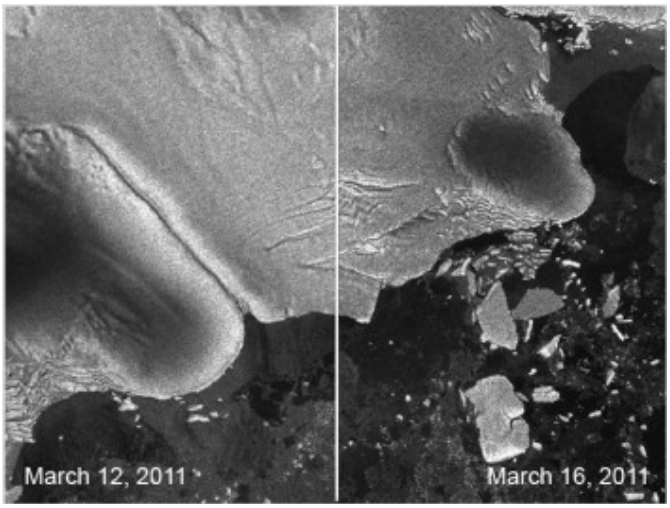
Kelly Brunt, a cryosphere specialist at Goddard Space Flight Center, Greenbelt, Md., and colleagues were able to link the calving of icebergs from the Sulzberger Ice Shelf in Antarctica following the Tohoku Tsunami, which originated with an earthquake off the coast of Japan in March 2011. The finding, detailed in a paper published online today in the Journal of Glaciology, marks the first direct observation of such a connection between tsunamis and icebergs.

The birth of an iceberg can come about in any number of ways. Often, scientists will see the towering, frozen monoliths break into the polar seas and work backwards to figure out the cause.

So when the Tohoku Tsunami was triggered in the Pacific Ocean on March 11 this spring, Brunt and colleagues immediately looked south. All the way south. Using multiple satellite images, Brunt, Emile Okal at Northwestern University and Douglas MacAyeal at University of Chicago were able to observe new icebergs floating off to sea shortly after the sea swell of the tsunami reached Antarctica.

To put the dynamics of this event in perspective: An earthquake off the coast of Japan caused massive waves to explode out from its epicenter. Swells of water swarmed toward an ice shelf in Antarctica, 8,000 miles (13,600 km) away, and about 18 hours after the earthquake occurred, those waves broke off several chunks of ice that together equaled about two times the surface area of Manhattan. According to historical records, this particular piece of ice hadn't budged in at least 46 years before the tsunami came along.

And as all that was happening, scientists were able to watch the Antarctic ice shelves in as close to real-time as satellite imagery allows, and catch a glimpse of a new iceberg floating off into the Ross Sea.



Before (left) and after (right) photos of the Sulzberger Ice Shelf illustrate the calving event associated with the Japan earthquake and resulting tsunami that occurred on March 11, 2011. The icebergs have just begun to separate in the left image. **Credit: European Space Agency/Envisat** **Download/Play video**

"In the past we've had calving events where we've looked for the source. It's a reverse scenario – we see a calving and we go looking for a source," Brunt said. "We knew right away this was one of the biggest events in recent history – we knew there would be enough swell. And this time we had a source."

Scientists first speculated in the 1970s that repeated flexing of an ice shelf – a floating extension of a glacier or ice sheet that sits on land – by waves could cause icebergs to break off. Scientific papers in more recent years have used models and tide gauge measurements in an attempt to quantify the impact of sea swell on ice shelf fronts.

The swell was likely only about a foot high (30 cm) when it reached the Sulzberger shelf. But the consistency of the waves created enough stress to cause the calving. This particular stretch of floating ice shelf is about 260 feet (80 meters) thick, from its exposed surface to its submerged base.

When the earthquake happened, Okal immediately honed in on the vulnerable faces of the Antarctic continent. Using knowledge of iceberg calving and what a NOAA model showed of the tsunami's projected

path across the unobstructed Pacific and Southern oceans, Okal, Brunt and MacAyeal began looking at what is called the Sulzberger Ice Shelf. The Sulzberger shelf faces Sulzberger Bay and New Zealand.

Through a fortuitous break in heavy cloud cover, Brunt spotted what appeared to be a new iceberg in MODerate Imaging Spectroradiometer (MODIS) data.

"I didn't have strong expectations either way whether we'd be able to see something," Brunt said. "The fastest imagery I could get to was from MODIS Rapid Response, but it was pretty cloudy. So I was more pessimistic that it would be too cloudy and we couldn't see anything. Then, there was literally one image where the clouds cleared, and you could see a calving event."

A closer look with synthetic aperture radar data from the European Space Agency satellite, Envisat, which can penetrate clouds, found images of two moderate-sized icebergs – with more, smaller bergs in their wake. The largest iceberg was about four by six miles in surface area – itself about equal to the surface area of one Manhattan. All the ice surface together about equaled two Manhattans. After looking at historical satellite imagery, the group determined the small outcropping of ice had been there since at least 1965, when it was captured by USGS aerial photography.

The proof that seismic activity can cause Antarctic iceberg calving might shed some light on our knowledge of past events, Okal said.

"In September 1868, Chilean naval officers reported an unseasonal presence of large icebergs in the southernmost Pacific Ocean, and it was later speculated that they may have calved during the great Arica earthquake and tsunami a month earlier," Okal said. "We know now that this is a most probable scenario."

MacAyeal said the event is more proof of the interconnectedness of Earth systems.

"This is an example not only of the way in which events are connected across great ranges of oceanic distance, but also how events in one kind of Earth system, i.e., the plate tectonic system, can connect with another kind of seemingly unrelated event: the calving of icebergs from Antarctica's ice sheet," MacAyeal said.

In what could be one of the more lasting observations from this whole event, the bay in front of the Sulzberger shelf was largely lacking sea ice at the time of the tsunami. Sea ice is thought to help dampen swells that might cause this kind of calving. At the time of the Sumatra tsunami in 2004, the potentially vulnerable Antarctic fronts were buffered by a lot of sea ice, Brunt said, and scientists observed no calving events that they could tie to that tsunami.

"There are theories that sea ice can protect from calving. There was no sea ice in this case," Brunt said. "It’s a big chunk of ice that calved because of an earthquake 13,000 kilometers away. I think it's pretty cool."

http://www.eurekalert.org/pub_releases/2011-08/jaaj-dbs080511.php

Deep brain stimulation effects may last for 10 years in patients with Parkinson's disease

One decade after receiving implants that stimulate areas of their brains, patients with Parkinson's disease (PD) appear to sustain improvement in motor function

One decade after receiving implants that stimulate areas of their brains, patients with Parkinson's disease (PD) appear to sustain improvement in motor function, although part of the initial benefit wore off mainly because of progressive loss of benefit in other functions, according to a report published Online First by Archives of Neurology, one of the JAMA/Archives journals.

According to background information in the article, several previous clinical studies have shown deep brain stimulation of the subthalamic nucleus (STN-DBS) for PD to be effective and safe. Studies have shown that the technique, which stimulates a part of the brain involved in motor function, may have advantages compared with other medical treatments in terms of controlling motor complications and improving quality of life. "The motor improvement induced by STN stimulation has been reported to be sustained for up to five to eight years after surgery, although part of the initial benefit progressively deteriorates, mainly because of worsening axial signs," write the authors. "To date, studies with postoperative follow-up for longer than eight years are lacking."

Anna Castrioto, M.D., from the Università degli Studi di Perugia, Perugia, Italy, and colleagues conducted a study of 18 patients with advanced PD who had received DBS implants for PD between 1996 and 2000. Motor assessments were conducted before implantation and at one, five and 10 years. All motor assessments were videotaped. Patients were assessed without medication, without stimulation, without either, and with both. At each assessment, the researchers recorded every patient's medications and dosages.

At 10 years, the combination of medication and STN-DBS was associated with significantly better motor, resting and action tremor, bradykinesia (slowed movement) and rigidity scores. Compared with baseline, reductions were also seen in the scores in the medication and no medication conditions, the dyskinesia (difficulty controlling movement) and motor fluctuation scores and the levadopa-equivalent daily dose. However, axial signs (such as posture, gait and balance) showed the most progressive decline in stimulation and medication response.

"Our findings further support the long-term response to STN stimulation in patients with advanced PD, showing a prolonged motor improvement up to 10 years," conclude the authors.
(Arch Neurol. Published August 8, 2011. doi:10.1001/archneurol.2011.182. Available pre-embargo to the media at www.jamamedia.org.)

http://www.eurekalert.org/pub_releases/2011-08/eu-cas080511.php

Chimpanzees are spontaneously generous after all

New study challenges previous findings that humans are an altruistic anomaly, and positions chimpanzees as cooperative, especially when their partners are patient

Researchers at the Yerkes National Primate Research Center have shown chimpanzees have a significant bias for prosocial behavior. This, the study authors report, is in contrast to previous studies that positioned chimpanzees as reluctant altruists and led to the widely held belief that human altruism evolved in the last six million years only after humans split from apes. The current study findings are available in the online edition of Proceedings of the National Academy of Sciences.

According to Yerkes researchers Victoria Horner, PhD, Frans de Waal, PhD, and their colleagues, chimpanzees may not have shown prosocial behaviors in other studies because of design issues, such as the complexity of the apparatus used to deliver rewards and the distance between the animals. "I have always been skeptical of the previous negative findings and their over-interpretation, says Dr. de Waal. "This study confirms the prosocial nature of chimpanzees with a different test, better adapted to the species," he continues.

In the current study, Dr. Horner and colleagues greatly simplified the test, which focused on offering seven adult female chimpanzees a choice between two similar actions: one that rewards both the "actor," the term used in the paper for the lead study participant, and a partner, and another that rewards only the actor/chooser herself. Examples of the critically important simplified design aspects include allowing the study partners to sit close together and ensuring conspicuous food consumption, which the researchers achieved by wrapping pieces of banana in paper that made a loud noise upon removal.

In each trial, the chooser, which was always tested with her partner in sight, selected between differently colored tokens from a bin. One colored token could be exchanged with an experimenter for treats for both members of the pair (prosocial); the other colored token would result in a treat only for the chooser (selfish). All seven chimpanzees showed an overwhelming preference for the prosocial choice. The study also showed the choosers behaved altruistically especially towards partners who either patiently waited or gently reminded them that they were there by drawing attention to themselves. The chimpanzees making the choices were less likely to reward partners who made a fuss, begged persistently or spat water at them, thus showing their altruism was spontaneous and not subject to intimidation.

"We were excited to find female after female chose the option that gave both her and her partner food," says Dr. Horner. "It was also interesting to me that being overly persistent did not go down well with the choosers. It was far more productive for partners to be calm and remind the choosers they were there from time to time," she continues.

The authors say this study puts to rest a longstanding puzzle surrounding chimpanzee altruism. It is well-known these apes help each other in the wild and show various forms of empathy, such as reassurance of distressed parties. The negative findings of previous studies did not fit this image. These results, however, confirm chimpanzee altruism in a well-controlled experiment, suggesting human altruism is less of an anomaly than previously thought.

The study authors next plan to determine whether the altruistic tendency of the chimpanzees towards their partners is related to social interactions within the group, such as reciprocal exchanges of food or social support.

For eight decades, the Yerkes National Primate Research Center, Emory University, has been dedicated to conducting essential basic science and translational research to advance scientific understanding and to improve the health and well-being of humans and nonhuman primates. Today, the center, as one of only eight National Institutes of Health–funded national primate research centers, provides leadership, training and resources to foster scientific creativity, collaboration and discoveries. Yerkes-based research is grounded in scientific integrity, expert knowledge, respect for colleagues, an open exchange of ideas and compassionate quality animal care.

Within the fields of microbiology and immunology, neurologic diseases, neuropharmacology, behavioral, cognitive and developmental neuroscience, and psychiatric disorders, the center's research programs are seeking ways to: develop vaccines for infectious and noninfectious diseases; treat drug addiction; interpret brain activity through imaging; increase understanding of progressive illnesses such as Alzheimer's and Parkinson's diseases;

unlock the secrets of memory; determine how the interaction between genetics and society shape who we are; and advance knowledge about the evolutionary links between biology and behavior.

http://www.nature.com/news/2011/110805/full/news.2011.463.html

Antibodies linked to long-term Lyme symptoms

Researchers find molecules that might mark elusive syndrome.

Amy Maxmen

Some patients with Lyme disease still show symptoms long after their treatment has finished. Now proteins have been discovered that set these people apart from those who are easily cured.

People who experience the symptoms of Lyme disease, which include fatigue, soreness and memory or concentration loss, after treatment for the disorder are sometimes diagnosed as having chronic Lyme disease or post-Lyme disease syndrome. But these diagnoses are difficult to make, because the individuals no longer seem to harbour the bacteria that cause Lyme disease. And the symptoms could instead be indicative of chronic fatigue syndrome or depression.

Now Armin Alaedini at Weill Cornell Medical College in New York and his colleagues have found that patients diagnosed with post-Lyme disease syndrome have antibodies that suggest they carried the infection for an unusually long time. The finding, published in Clinical Immunology1, might help the syndrome to be better understood, diagnosed and treated.

Alaedini's team looked at antibodies made in response to a protein called VlsE, which is found on the surface of Borrelia burgdorferi, the tick-borne bacterium that causes Lyme disease. The antibodies recognize a snippet of the protein called an epitope, and recruit the immune system to attack the bacterium. The researchers found that post-Lyme sufferers have a greater variety of antibodies to this epitope than patients whose infection cleared up quickly.

This finding suggests that patients with chronic symptoms have experienced a prolonged infection, caused by microbes that have evaded the immune system by varying the epitopes they carry. As a result of these variations, the body makes new antibodies targeting the modified protein. The longer the microbe manages to keep changing, the more diverse its host's antibodies become.

Some post-Lyme sufferers had varied antibodies against VlsE epitopes despite being diagnosed and treated early, says Alaedini. "That could mean they naturally have a different antibody response to the infection than most people; it could mean they weren't treated properly; or it's possible they were reinfected and the second infection was never treated," he says.

Inflammatory role

"This is the first study I've seen that shows some immunologic difference between someone who resolves their Lyme and someone who develops post-Lyme disease syndrome," says Linda Bockenstedt, a rheumatologist and immunologist at Yale School of Medicine in New Haven, Connecticut.

The presence of varied antibodies hints that the chronic symptoms could be caused by an ongoing inflammatory response caused by antibodies mistakenly reacting to the body's own proteins, Bockenstedt suggests.

"The big question to me is whether this can lead to an autoimmune phenomenon," says Bockenstedt. "But if that were the case, I'd expect the disease to worsen without immune-modulating treatment, and it doesn't."

Alaedini suggests that higher levels of antibodies could increase the body's levels of cytokines, immune-system proteins that can trigger the symptoms experienced by patients with post-Lyme disease syndrome. "Various cytokine profiles have been associated with fatigue, anxiety and depression," he explains.

If these antibodies are unique to people with chronic Lyme disease, it could lead to a test and treatments for the disorder, Alaedini says. It could also guide treatment of the disease itself. "If patients with an acute infection develop antibodies to these epitopes, perhaps they require a more aggressive course of therapy," he adds.

But a predictive marker won't be useful without new therapies for the persistent symptoms, says Henry Feder Jr, a physician specializing in infectious diseases at the University of Connecticut Health Center in Farmington. If an immune response problem leads to the syndrome, antibiotics won't help. "I guarantee you that if you tell a patient they won't feel better after antibiotics, they won't," Feder says. "We need to know what's going on."

References *Chandra A. et al. Clin. Immunol. http://dx.doi.org/10.1016/j.clim.2011.06.005 (2011).*

http://www.bbc.co.uk/news/science-environment-14401305

Roman dead baby 'brothel' mystery deepens

By Louise Ord Assistant Producer, Digging For Britain

New research has cast doubt on the theory that 97 infants were killed at a Roman brothel in Buckinghamshire.

In 2008, the remains of the newborn babies were rediscovered packed in cigarette cases in a dusty museum storeroom by Dr Jill Eyers from Chiltern Archaeology.

They were excavated from the remains of a lavish Roman villa complex in Buckinghamshire almost 100 years earlier, but had remained hidden ever since.

The story caught the attention of the world's press last year as Dr Eyers suggested that the villa was operating as a brothel and its occupants committing infanticide to dispose of unwanted offspring.

"Even now, a year after all the original press attention, every other day I'm getting inquiries about this story. It seems that everyone is intrigued by this puzzle," said Dr Eyers.

She has now carefully plotted the infant burials and the associated artefacts from The Yewden Villa at Hambleden. This revealed that all those infants that could be dated were buried between 150AD and 200AD, meaning all their deaths look like they took place in a 50-year period. And she said she now had a whole host of other evidence from studying the landscape around the villa site to support her brothel theory.

She admitted: "To be honest, when I first put this idea forward last year, it was really to get people talking and debating, but the more I look into this, the more convinced I am by my original brothel theory."

Brett Thorn, keeper of archaeology at the Buckinghamshire County Museum, has disputed her hypothesis.

"My main concern with the brothel theory is that it's just too far away from any major population centres. I'm just not convinced," he said. He has put together an exhibition of other objects from the villa excavation that could point to the villa having associations with a series of mother goddess cults from around the world.

"There are a few significant religious objects from the site that indicate possible connections with a mother goddess cult," he explained. "They may indicate that the site was a shrine and women went there to give birth, and get protection from the mother goddess during this dangerous time. The large number of babies who are buried there could be natural stillbirths, or children who died in labour."

Last year during filming for BBC Two's Digging for Britain series, presenter Dr Alice Roberts noticed cut marks made by a sharp implement on one of the bones, a discovery that was not revealed to the public until now. Cut marks can indicate anything from ritual practices involving human sacrifice, the de-fleshing of bones before burial, or the dismembering of a baby during childbirth to save the life of the mother.

Keri Brown at the University of Manchester carried out DNA tests on the 10 sets of the ancient bones to determine the sex of some of the infants.

It is common throughout history in cases of infanticide for girls to be killed rather than boys, but the opposite holds true for brothel sites. A brothel site at Ashkelon in Israel revealed that nearly all of the babies were boys. Although the tests represented a very small sample of the total number of baby skeletons found, there seemed to be an equal number of victims of both sexes at the Buckinghamshire site, and so the mystery for now remains unsolved.

Dr Eyers said she believed that only further excavation at the site would clear up the mystery once and for all.

http://www.eurekalert.org/pub_releases/2011-08/uoc--usc080811.php

UCLA stem cell scientists uncover for the first time why the human heart can't regenerate itself

Study has implications for reprogramming human cardiac myocytes to replace damaged heart muscle

Stem cell researchers at UCLA have uncovered for the first time why adult human cardiac myocytes have lost their ability to proliferate, perhaps explaining why the human heart has little regenerative capacity.

The study, done in cell lines and mice, may lead to methods of reprogramming a patient's own cardiac myocytes within the heart itself to create new muscle to repair damage, said Dr. Robb MacLellan, a researcher with the Eli and Edythe Broad Center of Regenerative Medicine and Stem Cell Research at UCLA and senior author of the study.

Unlike newts and salamanders, human adults cannot spontaneously regrow damaged organs such as the heart. However, recent research suggests that mammals do have the ability to regenerate the heart for a very brief period, about the first week of life. But that ability is quickly lost. But if we had it once, MacLellan said, maybe it is possible to regain that ability.

Published in the Aug. 8 issue of the peer-reviewed Journal of Cell Biology, MacLellan's study suggests it might be possible to turn back the cellular clock to a time when cardiac myocytes had the ability to proliferate and re-grow heart muscle.

"These salamanders and other lower organisms have the ability to de-differentiate cardiac myocytes, or take them back to an earlier, more primitive state, which allows them to re-enter the cell cycle, creating new heart muscle," said MacLellan, who also is an associate professor of cardiology and physiology. "In mammals, we've lost that potential. If we knew how to restore that, or knew the reason why adult myocytes can't do it, we could try to figure out a way to use nature's methods to regenerate the heart."

During human development, cardiac myocytes are made by progenitor stem cells and proliferate to form the heart. Once the heart is formed, the myocytes transform from immature cells into mature cells that cannot proliferate. That's not so for newts and salamanders, whose cardiac myocytes can go back and forth between immature, or primitive, states to proliferate and repair damage and then revert back into mature cells once the damage is repaired.

MacLellan believes the reason adult human cardiac myocytes can't do this is quite simple – when the myocytes are in a more primitive state, they are not as good at contracting, which is vital for proper heart function. Because humans are much larger than newts and salamanders, we needed more heart contraction to maintain optimum blood pressure and circulation.

"The way we evolved, in order to maintain blood pressure and flow we had to give up the ability to regenerate the heart muscle," MacLellan said. "The up side is we got more efficient cardiac myocytes and better hearts. But it was a trade-off."

MacLellan said that by temporarily knocking down the proteins that block the cell cycle mechanism, it may be possible to get adult cardiac myocytes to re-enter the cell cycle and revert to a state where they can again proliferate. These therapies would need to be reversible so that the effects of the protein manipulation eventually wear off once the damage is repaired. Then myocytes would become mature again and aid in contracting the regenerated heart muscle. MacLellan currently is looking into using nanoparticles to deliver small interfering RNA to the heart to knock out the proteins that are keeping the myocytes mature.

When a heart attack occurs, oxygen is cut off to part of the heart, causing the cardiac myocytes to die and resulting in scar tissue. It's easy to locate the damaged area of the heart, and if a way could be developed to reprogram a patient's own myocytes, the protein manipulation system could be injected into the damaged area, reverting the myocytes to their primitive state and replacing the dead muscle with new, living muscle, MacLellan said.

"People have been talking about the regenerative potential of these lower organisms for a long time and why this does not occur in humans" MacLellan said. "This is the first paper that provided a rationale and mechanism for why this happens."

There has been much talk of using human embryonic stem cells or reprogrammed induced pluripotent stem cells to regenerate the heart. However, it's unknown how much regeneration is possible and how much benefit would come from it.

"From my point of view, this is a potential mechanism to regenerate heart muscle without having to harvest or expand stem cells," MacLellan said. "Each person would be their own source for cells for regeneration."

The five-year study was funded by the National Institutes of Health.

http://www.dailymail.co.uk/news/article-2024221/Marco-Polo-reached-China-picked-tales-Orient-Italians-claim.html

Marco Polo 'never went to China and picked up tales of the Orient from other travellers'

By Mail Foreign Service

Marco Polo, one of history's greatest explorers, may in fact have been a conman, it was claimed yesterday.

Far from being a trader who spent years in China and the Far East, he probably never went further east than the Black Sea, according to a team of archaeologists.

They suspect the Venetian adventurer picked up stories about the mysterious lands of the Orient from fellow traders around the Black Sea who related tales of China, Japan and the Mongol Empire in the 13th century.

He then put the stories together in a book which purports to be his account of his travels between 1271 and 1291. It details his relations with Kublai Khan, the Mongol ruler.

But now an Italian team of archaeologists studying in Japan have cast doubts about one of their country"s national heroes - although there have been competing claims to him from Croatia, which argues he was born there.

Travelling trickster? Marco Polo may have fabricated his experiences by using other people"s stories

Following the research, Professor Daniele Petrella of the University of Naples told Italian history magazine Focus Storia there were many inconsistencies and inaccuracies in Marco Polo"s description of Kublai Khan"s

invasions of Japan in 1274 and 1281. “He confuses the two, mixing up details about the first expedition with those of the second,” Professor Petrella said.

“In his account of the first invasion, he describes the fleet leaving Korea and being hit by a typhoon before it reached the Japanese coast,” said Professor Daniele Petrella of the University of Naples, the leader of the archaeology team. “But that happened in 1281 – is it really possible that a supposed eye witness could confuse events which were seven years apart?”

Polo7s description of the Mongol fleet did not square with the remains of ships the archaeologists excavated in Japan, as he had written of ships with five masts, while those which had been found had only three.

“It was during our dig that doubts began to emerge about much of what he wrote,” added Professor Petrella.

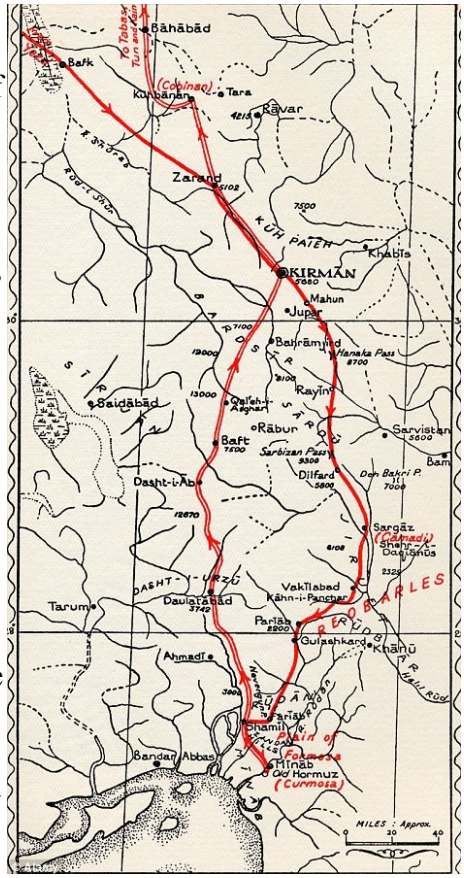
“When he describes Kublai Khan”s fleet he talks about the pitch that was used to make ships” hulls watertight. He used the word “chunam”, which in Chinese and Mongol means nothing.

“In fact, it is the Persian word for pitch. It’s also odd that instead of using, as he does in most instances, local names to describe places, he used Persian terms for Mongol and Chinese place names.”

The explorer claimed to have worked as an emissary to the court of Kublai Khan, but his name does not crop up in any of the surviving Mongol or Chinese records.

The famous travel book was said to have been dictated by Polo to a fellow prisoner named Pisa while he was in jail after returning from his adventures, and to be fair to Polo, it is thought Pisa embellished many of the stories.

But the latest claims back those made in a book by British academic Frances Wood in 1995 entitled “Did Marco Polo go to China?”. She argued he never got beyond the Black Sea and that his famed account was a collection of travellers’ tales.



Extraordinary trip: Hailed as one of the greatest explorers of history, archeologists are suspicious of how genuine Marco Polo”s tales are

http://www.eurekalert.org/pub_releases/2011-08/uops-pss080911.php

Penn study shows an ancient crop effective in protecting against a 21st century hazard

A diet of flaxseed shows protective effects against radiation in animal models

PHILADELPHIA - Flax has been part of human history for well over 30,000 years, used for weaving cloth, feeding people and animals, and even making paint. Now, researchers from the Perelman School of Medicine at the University of Pennsylvania have discovered that it might have a new use for the 21st century: protecting healthy tissues and organs from the harmful effects of radiation. In a study just published in BMC Cancer, researchers found that a diet of flaxseed given to mice not only protects lung tissues before exposure to radiation, but can also significantly reduce damage after exposure occurs.

"There are only a handful of potential mitigators of radiation effect, and none of them is nearly ready for the clinic," says the principal investigator Melpo Christofidou-Solomidou, PhD, research associate professor of Medicine, Pulmonary, Allergy and Critical Care Division. "Our current study demonstrates that dietary flaxseed, already known for its strong antioxidant and anti-inflammatory properties, works as both a mitigator and protector against radiation pneumonopathy."

In several separate experiments, the researchers fed one group of mice a diet supplemented with 10 percent flaxseed, either three weeks before a dose of X-ray radiation to the thorax or two, four, or six weeks after radiation exposure. A control group subjected to the same radiation dose was given the same diet but receiving an isocaloric control diet without the flaxseed supplement. After four months, only 40 percent of the irradiated control group survived, compared to 70 to 88 percent of the irradiated flaxseed-fed animals. Various studies of blood, fluids, and tissues were conducted.

Dr. Christofidou-Solomidou and her colleagues found that the flaxseed diet conferred substantial benefits regardless of whether it was initiated before or after irradiation. Mice on flaxseed displayed improved survival rates and mitigation of radiation pneumonitis, with increased blood oxygenation levels, higher body weight, lower pro-inflammatory cytokine levels, and greatly reduced pulmonary inflammation and fibrosis.

The latter finding is especially exciting, because while radiation-induced inflammatory damage can be potentially treated with steroidal therapy (in radiotherapy patients for example), lung fibrosis is essentially

untreatable. "There's nothing you can give to patients to prevent fibrosis," Dr. Christofidou-Solomidou points out. "Once a lung becomes "stiff" from collagen deposition, it's irreversible. We have discovered that flaxseed not only prevents fibrosis, but it also protects after the onset of radiation damage."

Dr. Christofidou-Solomidou and her colleagues are focusing further research on the bioactive lignan component of flaxseed, known as SDG (secoisolariciresinol diglucoside), which is believed to confer its potent antioxidant properties. The lignan component also "regulates the transcription of antioxidant enzymes that protect and detoxify carcinogens, free radicals and other damaging agents", she says.

Flaxseed boasts many other qualities that make it particularly attractive as a radioprotector and mitigator. "Flaxseed is safe, it's very cheap, it's readily available, there's nothing you have to synthesize," Dr. Christofidou-Solomidou notes. "It can be given orally so it has a very convenient administration route. It can be packaged and manufactured in large quantities. Best of all, you can store it for very long periods of time." That makes it especially interesting to government officials looking to stockpile radioprotective substances in case of accidental or terrorist-caused radiological disasters.

Co-author Keith Cengel, MD, PhD, assistant professor of Radiation Oncology at Penn, explains that in such cases, "a big issue is the 'worried well' -- all the folks who probably weren't exposed but are concerned and want to do something." Many potential radioprotectors, however, could have risky side effects. Dr. Christofidou-Solomidou adds, "When you give something to 4 or 5 million 'worried well,' you have people with preexisting medical conditions. You can't give just anything to people with heart disease, for example. But this is absolutely safe. In fact, it is known to increase cardiovascular health, a finding shown by another group of Penn investigators a few years ago. It's loaded with omega-3 fatty acids."

Along with other researchers at the Perelman School of Medicine, the authors are conducting further pilot studies on the potential of flaxseed for mitigation of lung damage in patients awaiting lung transplants and those undergoing radiation therapy for the treatment of intra-thoracic malignancies. Dr. Christofidou-Solomidou is even conducting a pilot study for NASA on the benefits of flaxseed for astronauts on extended deep space missions. Lengthy space exploration missions require that the astronauts perform extravehicular activities (EVAs) for repairs, during which they can face exposure to high levels of solar and galactic radiation with the added risk factor of breathing 100 percent oxygen. "Hyperoxia superimposed with radiation could potentially cause some lung damage and some reason to worry for the astronauts," she says. "We are one of a handful of teams in the US that can study radiation in addition to hyperoxia. So now we're adding another level of complexity to the one-hit, radiation damage studies; the double-hit model is something novel, nobody has done it before."

The researchers are already convinced enough to incorporate flaxseed into their own routine. "I actually eat it every morning," says Dr. Cengel, noting, "The potential health benefits are significant and there is no known toxicity—it just makes good sense to me."

The study is funded by the National Institute of Allergy and Infectious Diseases (NIAID) and the Biomedical Advanced Research and Development Authority (BARDA) under a grant initiative focused on the development of novel medical countermeasures to prevent or mitigate pulmonary injury or restore function after exposure to ionizing radiation.

http://www.eurekalert.org/pub_releases/2011-08/cu-cbo080911.php

Can blaming others make people sick?

Concordia researchers probe link between bitterness and disease

Montreal - Constant bitterness can make a person ill, according to Concordia University researchers who have examined the relationship between failure, bitterness and quality of life.

"Persistent bitterness may result in global feelings of anger and hostility that, when strong enough, could affect a person's physical health," says Carsten Wrosch, a professor in the Concordia University Department of Psychology and a member of the Centre for Research in Human Development.

Wrosch is particularly interested in why some people avoid bitterness at different stages of life and why others don't. He's incorporated his theoretical considerations regarding bitterness in "Self-Regulation of Bitterness Across the Lifespan," a chapter from the recently published book, Embitterment: Societal, psychological, and clinical perspectives (Springer 2011).

Over the last 15 years, Wrosch has investigated how negative emotions, such as regret or sadness, affect people. Most recently, he has focused his attention on the impact of bitterness. With his co-author, Concordia alumna Jesse Renaud, they single out failure as one of the most frequent causes of bitterness. Anger and recrimination are its typical attendants.

Unlike regret, which is about self-blame and a case of "woulda, coulda, shoulda," acrimony points the finger elsewhere — laying the blame for failure on external causes. "When harboured for a long time," says Wrosch,

"bitterness may forecast patterns of biological dysregulation (a physiological impairment that can affect metabolism, immune response or organ function) and physical disease."

Bitterness as a medical disorder?

It is one thing to suggest that bitterness may cause sickness, quite another to propose that it be recognized as a mental illness. Such is the proposal that was first made by Michael Linden, head of the psychiatric clinic at Free University of Berlin in 2003. Linden argues that bitterness is actually a medical disorder and should be categorized as post-traumatic embitterment disorder (PTED). He estimates that between one and two per cent of the population is embittered and by giving the condition a proper name, people with PTED will receive the therapeutic attention they deserve.

The jury is still out on this proposal. Wrosch and Renaud say bitterness can be avoided, if people who experience failure can find other ways to fulfil their goals. If they can't, the researchers stress, it's essential to disengage from the fruitless effort (e.g., to get promoted, to save a marriage) and reengage in something that's equally meaningful (e.g., a new job or passion).

Called self-regulation processes, disengaging and reengaging can be necessary for a person to avoid bitter emotions. "Any effective therapeutic intervention," says Renaud, "hinges on the affected individual finding ways to self-regulate."

In some cases, overcoming bitterness demands more than self-regulation. When bitterness arises from blaming other people, then recovery may involve others. "In order to deal with bitter emotions there may need to be something else required to enable a person to overcome the negative emotion — that something is forgiveness," says Wrosch.

Partners in research: This work was supported by the Canadian Institutes of Health Research and the Social Sciences and Humanities Research Council of Canada.

http://www.newscientist.com/article/dn20775-mental-abacus-does-away-with-words.html

Mental abacus does away with words

13:56 09 August 2011 by Ferris Jabr

Studies on a group of children trained to use a "mental abacus" suggest the technique frees mathematics from its usual dependence on language

When 11-year-old Priyanshi Somani multiplies strings of 10-digit numbers or finds the square root of a six-digit number, she doesn't use a calculator or even pencil and paper. Instead, like other specially trained youngsters, the young Mental Calculation World Cup champion manipulates an imaginary abacus.

Now studies on a group of children trained to use a "mental abacus" suggest the technique frees mathematics from its usual dependence on language.

In some parts of the world, particularly India, China and Japan, schoolchildren sign up for intense training programmes that teach them how to perform complex calculations in their heads using a mental abacus.

Intrigued, Michael Frank of Stanford University in California and David Barner at the University of California, San Diego, travelled to a school in Vadadora in Gujarat, India, where children learn mental abacus in a 3-year-long after-school programme.

Ali Baba

Previous research has suggested that mental abacus relies on visual working memory, but it wasn't clear how children kept track of all the columns: a typical abacus might have more than 15 columns, yet most people have trouble simultaneously visualising more than three or four distinct items in their minds.

In one experiment, Frank and Barner studied children who had spent a year learning to work a physical abacus and had recently begun practicing mental abacus. The pair asked the students to perform challenging additions. Most of them had difficulty performing calculations with numbers that had more than three or four digits. Frank suggests that the children only represent three or four columns of an abacus in their minds at any given time.

In a second experiment, the pair asked 15 expert mental abacus students to do complex calculations while listening to the story Ali Baba and the Forty Thieves. At the same time, these children had to repeat each word of the tale as they heard it – a language task – or drum their fingers on the table – a motor task – or do both.

Visual representation

The language and motor tasks somewhat hindered the expert children's mental calculations, with the language task interfering slightly less than the motor task. In contrast, a group of undergraduates from the University of California with no experience in mental abacus found it almost impossible to perform complex calculations while listening to the story.

All of this suggests that for practiced experts, mental abacus does not depend strongly on language systems, says Frank. Most of us need words to represent a number like 134,789 – we rely on concepts represented by

verbal numbers like "seven-hundred and eighty-nine" – but mental abacus may be largely a visual task for those who master it.

"What we found confirms and extends previous work suggesting that mental abacus is not based on language, but is really a mental image of some sort, a visual representation," Frank adds.

The design of the abacus not only makes it a powerful physical tool, it also facilitates mental visualisation. Grouping beads into a few sets makes them easier to hold in visual memory – just as dividing long telephone numbers into three or four-digit chunks helps us remember them. "Because the physical abacus groups beads into columns, it's easier to hold a mental image of the abacus in your head," says Frank.

Journal reference: Journal of Experimental Psychology, DOI: 10.1037/a0024427

http://www.eurekalert.org/pub_releases/2011-08/jhub-btf081011.php

Blood tests for active TB not accurate or cost-effective

Ba*sed on data, WHO advises against use of blood antibody test for active TB*

Commercial blood serum antibody tests—widely used in India and other developing countries to diagnose active tuberculosis—are not accurate or cost-effective, according to an analysis by researchers at the Johns Hopkins Bloomberg School of Public Health, the University of Washington School of Public Health and McGill University. Use of serological tests in India resulted in more DALYs (years of healthy life lost to premature death and illness), more secondary infections, and more false-positive diagnoses of TB, compared to the use of microscopic sputum smear analysis or culture. The findings, published in the August 9, 2011 edition of PLoS Medicine, recently led the World Health Organization to recommend against the use of commercial serology tests in the diagnosis of active TB.

"Microscopic analysis of sputum for TB is cheap and widely available, but misses half of all TB cases," said David Dowdy, MD PhD, lead author of the study and assistant professor in the Department of Epidemiology at the Bloomberg School of Public Health. "TB culture, the current gold standard, requires training and equipment not available in most resource-limited settings. Serological tests are simpler and faster than culture, and are also commercially available in India, so they are an attractive option in theory. However, we found that they are not accurate enough to be useful—after accounting for missed and false-positive TB diagnoses, serological tests cost more and delivered less than either microscopy or culture. Quite simply, serological tests should not be used to diagnose active TB."

For the study, Dowdy and his colleagues constructed a mathematical model to analyze 1.5 million patients with suspected active TB in India—about 15% of India's annual TB burden. Their analysis concluded that use of serology would result in an estimated 14,000 more TB diagnoses than microscopy, but would also incorrectly diagnose 121,000 more patients without active TB (false-positives). Serology use would also generate 102,000 more DALYs and 32,000 more secondary TB cases compared to microscopy. The estimated total cost of serologic testing (including treatment of newly diagnosed cases) was approximately four times that of microscopy, at $47.5 million versus $11.9 million.

"Unfortunately, we still do not have an accurate point-of-care test for TB, as we have for infections like HIV or malaria. The WHO policy strongly encourages future research to develop novel or improved serological tests," said the study's senior author, Madhukar Pai, MD, PhD, associate professor at McGill University and the Respiratory, Epidemiology and Clinical Research Unit at the Montreal Chest Institute and the Research Institute of the McGill University Health Centre.

Funding for the research was provided by the Stop TB Partnership's New Diagnostics Working Group, via the subgroup on Evidence Synthesis, and support from the Canadian Institutes of Health Research (CIHR).

http://www.bbc.co.uk/news/science-environment-14466814

Giant fossil shows huge birds lived among dinosaurs

An enormous jawbone found in Kazakhstan is further evidence that giant birds roamed - or flew above - the Earth at the same time as the dinosaurs.

Writing in Biology Letters, researchers say the new species, Samrukia nessovi, had a skull some 30cm long. If flightless, the bird would have been 2-3m tall; if it flew, it may have had a wingspan of 4m. The find is only the second bird of such a size in the Cretaceous geologic period, and the first in Asia. The only other evidence of a bird of such a size during the period was a fossilised spinal bone found in France and reported in a 1995 paper in Nature.

The fossilised jawbone is nearly twice the length of that of an ostrich, the largest bird found on Earth today

Sharing space

An overwhelming majority of the birds known from the period would have been about crow-sized, but Dr Darren Naish of the University of Portsmouth said that a second find of an evidently different species suggests that large birds were common at the time.

"This fossil is only known from its lower jaw, so unfortunately we can't say anything at all with certainty about the shape and form of the whole animal. If it was flightless and sort of ostrich-shaped, it would have been maybe 2-3m tall and somewhere over 50kg," he explained to BBC News. "If it was a flying animal, then maybe it was shaped like a big albatross or a condor."

Dr Naish also wondered about the dinosaurs with which the enormous birds shared their space.

"I think the really interesting thing is that they're living alongside the big dinosaurs we know were around at the time: big tyrannosaurs, long-necked sauropods, duck-billed dinosaurs," he said. "That opens up loads of questions about ecological interactions that we can only speculate about.

"People have said there weren't big birds when there were big pterosaurs, but now we know there were."

http://www.scientificamerican.com/article.cfm?id=red-meat-diabtetes

Daily Red Meat Raises Risk for Diabetes, Large Study Says

People who eat as little as one serving of red meat a day, whether it is processed or unprocessed, have an increased chance of getting type 2 diabetes

By Katherine Harmon | Wednesday, August 10, 2011 | 19

Sugary soda and other sweet treats are likely not the only foods to blame for the surge in diabetes across the U.S. New research out of Harvard University supports the theory that regular red meat consumption increases the risk of getting type 2 diabetes.

An average of just one 85-gram (three-ounce) serving of unprocessed red meat—such as a medium hamburger or a small pork chop—per day increased by 12 percent the chances a person would get type 2 diabetes over the course of a decade or two. And if the meat was processed—such as a hot dog or two slices of bacon—the risk increased to 32 percent, even though serving sizes were smaller.

The new study, published online August 10 in the American Journal of Clinical Nutrition, is not the first to find the link between red meat and diabetes risk. But it is the largest and one of the first to look separately at unprocessed and processed meats. "On a gram-per-gram basis, unprocessed red meat is still better," says Frank Hu, a professor of nutrition and epidemiology at the Harvard School of Public Health and co-author of the new paper. "But unprocessed red meat is still associated with a significantly increased risk."

More than 8.5 percent of U.S. adults have been diagnosed with diabetes, and in some counties in the so-called "diabetes belt" in the South, the numbers exceed 11.2 percent. The rates are expected to keep climbing in the coming years. Hu suggests that based on the analysis there is indeed a "disease burden that can be attributed to consumption of either processed or unprocessed red meat."

It's what's for dinner, for many

A U.S. adult consumes an average of more than 45 kilograms (100 pounds) of red meat a year. "There's no question that consumption of red meat is too high," Hu says, suggesting nuts, whole grains and low-fat dairy products as healthier alternatives. And diabetes is not the only reason to switch to lighter forms of protein. People who ate 113 grams (four ounces) of red meat a day are more likely to die from any cause over 10 years, according to a 2009 Archives of Internal Medicine study of half of a million people.

Of course, eating red meat for a week "is not going to give someone diabetes—we're talking about habitual consumption," Hu says. And for those uncertain about trading in a filet mignon for a fistful of almonds, the researchers behind the new paper also list poultry and fish as alternatives, although Hu cautions that other research also supports the move to a more plant-based diet.

"It really confirms what other studies have suggested," says Elizabeth Seaquist, director of the Center for Diabetes Research at the University of Minnesota, who was not involved in the new study. The new report analyzed health and dietary information from three large-scale cohort studies (the "Health Professionals Follow-Up Study," and the "Nurses' Health Study I and II"), which encompassed some 200,000 adults. It combined that data with previous studies for a meta-analysis that covered a total of 440,000 men and women who were followed for 10 years or more.

Seaquist puts great confidence in the findings based on the study's sample size, but notes that as an epidemiological study like this one should, it "raises more hypotheses than gives us answers."

The additional incriminating evidence for this category of food seems to swing the pendulum away from refined carbohydrates as the only culprits in advancing diabetes. And the new paper "will heighten awareness of the potential for different dietary components to contribute to diabetes," Charles Burant, director of the

Metabolomics and Obesity Center at the University of Michigan Medical School, who also was not involved in the new paper.

For people who have been following the research, however, "the findings are not particularly surprising," Hu says. In fact, despite the big play that sugars and other highly processed carbohydrates have gotten, red meat is actually "one of the most well-established dietary risk factors," he notes.

Size matters

One of the tricky aspects of lifestyle studies like this one is that unhealthful behaviors often go together, making it tough to tease them apart to see if one is having a larger effect than others. And in the studies, those who reported eating the most red meat also tended to have other risk factors for diabetes, such has having a higher body mass index (BMI), smoking and not getting much physical activity. "People who eat a lot of meats tend to gain more weight," Hu says.

So the new findings might be more "a reflection of poor dietary intake by people who eat meat," Burant says. Seaquist explains more plainly that perhaps "people who eat red meat end up eating French fries with it."

Burant is not convinced that red meats—or any other food category in particular—are to blame for diabetes. "My own personal bias is that it's not just what you eat but how much you eat," he says. As obesity is the largest known risk factor for developing type 2 diabetes, "calories are probably the primary driver for the risk for diabetes," Burant says. As a clinician, he says, he tells his patients who might be at risk for diabetes: "I don't care what you eat—just eat less of it."

But, after Hu and his colleagues adjusted their analysis to account for weight gain, they still saw a modest association between red meat and type 2 diabetes. That might mean that there are other factors at work independent of extra pounds, the researchers posit.

One theory is that the type of iron prevalent in red meat, known as heme iron, unlike forms of iron found in plants and nutritional supplements, is readily absorbed by the body and can in fact lead to iron overload. Studies have shown that too much iron can lead to oxidative stress and higher levels of inflammation, both of which can be precursors to diabetes. Seaquist cautions that plenty of people still need to make sure they get enough iron, but adds that, "there is a strong link to iron overload in the body and risk for diabetes."

Another reason for an increase in diabetes risk could be the types of saturated fats in red meat as well as the additives (such as nitrates and sodium) in processed food. But as Hu acknowledges, "it's very challenging to pinpoint the exact components of food that are responsible."

Many researchers are waiting for more evidence. "I don't think we can tell" yet whether red meat can be separated from overall weight gain as a factor for diabetes risk, Seaquist says. But, she notes, the fact that the study was so large and there was still a correlation between red meat consumption and diabetes risk after adjusting for BMI "makes me a little more concerned that maybe there really is something specific about red meat" that pushes the body toward diabetes.

In addition to looking to better understand the mechanisms at work in the body, Hu says, he will be keeping a close eye on other countries where diets are changing. "It's very important to locate the effects of meat consumption on chronic disease risk in countries that are undergoing nutritional transitions," Hu says. But as in the U.S., the panoply of food and lifestyle choices that people make are difficult to tease apart.

In the meantime, Seaquist says, the best nutritional tactic is akin to good investing: diversify. "We need to watch our total calories," she says. But we also "need to have a broad range of foods in our diet"—especially fruits and vegetables, she says.

http://www.scientificamerican.com/article.cfm?id=tweeting-your-health-woes-fights-diseases

Tweeting Your Health Woes Could Help Fight Disease

By analyzing tweets, public health researchers can track the progression of infectious diseases

By Jason Kane and PBS NewsHour | Wednesday, August 10, 2011 | 1

That "viral" metaphor for social media just got a little more bona fide. According to a recent slate of independent studies, Twitter can accurately track the spread of a virus or disease -- and do it much faster than traditional surveillance methods.

From Iowa to Brazil, researchers are discovering there is a distinct association between complaints, worries and random rants on the social media site and the spread of medical issues as wide-ranging as the flu, dengue fever and pollen-induced allergies.

"By looking at the Twitter stream, we were able to track the public concern in real time about vaccination issues, travel issues and responses to public health," said Dr. Philip Polgreen, an associate professor at the University of Iowa Carver College of Medicine who used Twitter to track the progression of the H1N1 influenza outbreak in 2009.

"But that not only helps us track the progression of something like the flu, it can provide a way of determining the effectiveness of communication about public health and what messages should be reinforced."

Researchers from the University of Iowa discovered the association for influenza by following Twitter keywords commonly linked with H1N1, such as "swine flu" and "influenza." The team began collecting the messages in April 2009, shortly after the first wave of H1N1 struck the United States. Not only did they find that tweets from people experiencing flu-like symptoms tracked closely with the information collected by the Centers for Disease Control, they also discovered they were highly accurate in terms of both time and location. The CDC results were much slower -- arriving two to three weeks after the patients began feeling sick.

"From a clinical standpoint, data is one of the cornerstones of public health and it's important to have that data in a timely fashion," Polgreen said. "If we can get information faster, we can perhaps intervene sooner."

Previous studies have shown that search engines like Google and Yahoo can be effective at correlating outbreaks with key search phrases like "symptoms of the flu," but the Twitter method excites researchers more because it provides more context.

Twitter users often don't shy away from complaining about their exact ailments and when they developed. Their profiles often list their location, and increasingly, other users can pinpoint their whereabouts even more precisely thanks to GPS devices. That kind of rich data can help health professionals find the epicenter of the outbreak, understand how it's being passed from person to person and estimate how quickly it will spread to other parts of the country.

The researchers themselves underestimated the potential depth of the treasure trove the social network could provide when they began the study. "It's easy to dismiss Twitter," said Alberto Segre, an author of the report and a computer scientist at the University of Iowa. "It's like, 'Who's going to tweet about the flu?' I personally don't see what's in it for the person who's tweeting. I was as surprised as the next guy that there actually was good information. It can be very noisy, but there's still a signal."

In a separate study, Mark Dredze and Michael J. Paul, computer scientists at the Center for Language and Speech Processing at Johns Hopkins University, came to a strikingly similar conclusion after analyzing 1.6 million tweets related to 15 different health conditions.

Using a new algorithm, they trained their computer to sift through more than 2 billion public tweets that were posted between May 2009 and October 2010. They separated ones complaining of legitimate ailments - such as "I just want to be able to drink water - #stupidstomach #flu" - from the ones proclaiming, "I've got Justin Bieber fever!" Their study found that the national flu rate calculated through Twitter has a 96 percent correlation with the rate as reported by the CDC. The tweets also revealed allergy patterns, cancer rates, self-medication behavior and over-the-counter drug misuse.

But the data have their limits. "It's not accurate enough to replace traditional methods," Paul said. "We won't be able to use this to determine the exact percentage of people who have the flu, but we can use it to see the flu rate is going up suddenly and we should investigate this. There's a lot of potential to learn so much about people that they don't necessarily share with their doctors."

And as Twitter spreads throughout the globe, that potential will likely grow. In fact, software created recently by a computer scientist at Brazil's Federal University of Minas Gerais has been used to identify a high correlation between official dengue fever statistics and the time and place Brazilians send a tweet saying they've contracted the virus.

Polgreen of the University of Iowa finds the Brazil report among the most interesting - perhaps because it could be a game-changer for worldwide disease control -- especially in developing countries. "Social media is emerging as an important source of information in Middle East politics, in entertainment, in science," Polgreen said. "Why not for health?"

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http://www.eurekalert.org/pub_releases/2011-08/uoc--dsr081111.php

Decade-long study reveals recurring patterns of viruses in the open ocean

Santa Barbara, Calif. ***Viruses fill the ocean and have a significant effect on ocean biology, specifically marine microbiology, according to a professor of biology at UC Santa Barbara and his collaborators.***

Craig A. Carlson, professor with UCSB's Department of Ecology, Evolution, and Marine Biology, is the senior author of a study of marine viruses published this week by the International Society for Microbial Ecology Journal, of the Nature Publishing Group. The new findings, resulting from a decade of research, reveal striking recurring patterns of marine virioplankton dynamics in the open sea, which have implications regarding our understanding of cycling of nutrients in the world's oceans.

Marine viruses encompass enormous genetic diversity, affect biogeochemical cycling of elements, and partially control aspects of microbial production and diversity, according to the scientists. Despite their importance in the ocean, there has been a surprising lack of data describing virioplankton distributions over time and depth in open oceanic systems.

"Microbial interactions, between oceanic viruses and bacteria, take place on the nanometer scale but are extremely important in governing the flow of energy and the cycling of nutrients like carbon, nitrogen, and phosphorus on the ecosystem scale of the world's oceans," said Carlson. The scientists studied microbes in the water column of the Saragasso Sea, off of Bermuda, for a decade.

"Although we can't see them with our naked eye, marine microbes are the dominant life forms in our oceans," said Rachel J. Parsons, first author and a microbial oceanographer with the Bermuda Institute of Ocean Science. "They comprise 95 percent of the living biomass in the oceans — more than all the krill, fish and whales put together. They grow at rates many times faster than larger animals. As a result of their sheer numbers, and the rates at which they grow, they are responsible for transforming and shaping the distribution of life's essential elements — and they help control climate on our planet. Without marine microbes, life as we know it could not persist."

According to the scientists, there are approximately 10 million viruses in every drop of surface seawater, yet despite the high number of viruses very few are infectious agents to larger animals like fish, whales, or humans. That is because almost all of the marine viruses are "phages" — viruses that specifically attack marine bacteria. Marine phages cannot carry out cellular metabolism and must therefore rely on the metabolic machinery of their bacterioplankton hosts to replicate. This warfare often kills the hosts, causing them to spill their internal nutrient content into the surrounding water.

In the new paper, the authors describe remarkably regular annual patterns of virioplankton abundance, tied to ocean physics and chemistry. These patterns in turn control the dynamics of the bacterioplankton hosts. The data suggest that a significant fraction of viruses in the upper photic, or light, zone of the subtropical oceanic gyres may be cyanophages — viruses that infect photosynthetic bacterioplankton.

If true, the dominance of cyanophages in open ocean systems has significant biogeochemical implications. Viral-mediated breakdown of cyanobacteria could benefit phytoplankton through the release of macro- and micronutrients. Viral breakdown of host cells converts particulate material to suspended or dissolved materials such as amino acids and nucleic acids, effectively resulting in the retention of nitrogen, phosphorous, and iron within the surface water. These dissolved materials fuel microbial activity in an otherwise nutrient-poor open ocean system.

In this decade-long study, the scientists studied in unprecedented detail the temporal and vertical patterns of virioplankton abundance within the open ocean. Samples were collected throughout the upper 300 meters of the water column every month, beginning in the year 2000, at an open ocean hydrostation called the Bermuda Atlantic Time-series Study (BATS) site. The additional data collected as part of the BATS program provides oceanographic details regarding ocean physics, chemistry, and biology that are extremely valuable for interpreting the observed trends in marine phages.

"This high-resolution, decadal survey provides insight into the possible controls of virioplankton dynamics and the role they play in regulating biology and nutrient cycling in the open ocean," said Carlson. "The data provided by this study will now be utilized by ecosystem and biogeochemical modelers in an attempt to better understand how microbial processes affect the larger biogeochemical cycling in the ocean."

Other co-authors of the study are Mya Breitbart, of the University of South Florida, and Michael W. Lomas, of the Bermuda Institute of Ocean Science.

http://www.eurekalert.org/pub_releases/2011-08/uom-hsf080911.php

Hidden soil fungus, now revealed, is in a class all its own

ANN ARBOR, Mich. - ***A type of fungus that's been lurking underground for millions of years, previously known to science only through its DNA, has been cultured, photographed, named and assigned a place on the tree of life.***

Researchers say it represents an entirely new class of fungi: the Archaeorhizomycetes. Like the discovery of a weird type of aquatic fungus that made headlines a few months ago, this finding offers a glimpse at the rich diversity of microorganisms that share our world but remain hidden from view.

The fungal phenomenon, brought to light by researchers at the University of Michigan, the Swedish University of Agricultural Sciences, the Imperial College London and Royal Botanic Gardens and the University of Aberdeen, is described in the Aug. 12 issue of the journal Science.

Although unseen until recently, the fungus was known to be extremely common in soil. Its presence was detected in studies of environmental DNA - genetic material from a living organism that is detected in bulk environmental samples, such as samples of the soil or water in which the organism lives.

"You couldn't really sample the soil without finding evidence of it," said Timothy James, a U-M assistant professor of ecology and evolutionary biology and an assistant curator at the university's herbarium. "So people really wanted to know what it looks like."

That became possible thanks to the work of the Swedish researchers, led by mycologist Anna Rosling. The researchers were studying mycorrhizae - fungi that colonize plant roots - when they discovered that some root tips harbored not only the mycorrhizae they were interested in, but also an unfamiliar fungus.

"When culturing mycorrhizal fungi from coniferous roots we were exited to find that one of the cultures represented this unfamiliar fungus," said Anna Rosling.

Later the culture was identified as a member of Soil Clone Group 1 (SCG1), a ubiquitous but enigmatic lineage known only from environmental DNA. It's not especially impressive to look at, James concedes: "It doesn't make some crazy structure that nobody's ever seen." But simply seeing and photographing a form of life that's been invisible until now is cause for excitement.

Having in hand a member of the elusive fungal group, the Swedish scientists and their collaborators have been able to study the group in more detail than ever before possible, using electron microscopy, DNA sequencing and in vitro growth studies to characterize it. The fungus they cultured is a slow-growing form that produces none of the typical aerial or aquatically dispersed spores most fungi typically reproduce with, suggesting it seldom if ever sees the light of day.

"By finding that it is slow growing and only produces spores in the soil, we can provide an explanation for why it has taken so long to be cultured," James said. The researchers also performed experiments aimed at understanding how the fungus, dubbed Archaeorhizomyces finlayi, interacts with the environment and with other organisms.

"We don't have any evidence that it's pathogenic; we don't have any evidence that it's mutualistic and doing anything beneficial for the plant," James said. "It's a little bit of a boring fungus." It may, however, help break down and recycle dead plants, a common - and extremely important - job for fungi. Hints of this role come from the observation that A. finlayi grows in the lab if provided with food in the form of glucose or cellulose (the main structural component of plant cell walls).

"Because it is so common in the soil, it must be very successful at what it does, and that role must be ecologically relevant," Rosling said.

Now that the researchers have ruled out some typical fungal roles - such as pathogen, benign endophyte, and member of a mycorrhizal association - they hope to find out through additional experiments exactly what role the fungus does play in nature and how it interacts with plants and other fungi.

"At this point we're still in the early stages of understanding what it's doing out there," James said.

Whether A. finlayi turns out to be beneficial or detrimental to the plants or microbes it interacts with, it's sure to contribute to understanding the diverse array of fungi in the world.

Though environmental DNA of SCG1 had been collected and reported in more than 50 previous studies, the type of DNA collected in the past didn't lend itself to analyses that would definitively pinpoint the group's position on the tree of life.

"Now that we have the culture, we can sequence almost any gene we want, so that's what we've done," James said.

The resulting information, combined with DNA data from the previous studies, revealed that A. finlayi belongs in an eclectic subphylum known as Taphrinomycotina, other members of which include the yeast Schizosaccharomyces, often used in studies of cell biology and evolution, and Pneumocystis, which can cause pneumonia in people with weakened immune systems, such as those who have cancer or HIV/AIDS or are undergoing treatment with immune-suppressing drugs.

In addition to James and Rosling, who is currently a visiting research associate at Indiana University, the paper's authors include Filipa Cox of the Imperial College London and Royal Botanic Gardens; Karelyn Cruz-Martinez, Katarina Ihrmark, Björn Lindahl and Audrius Menkis of the Swedish University of Agricultural Sciences; and Gwen-Aëlle Grelet of the University of Aberdeen.

The research was funded by the Carl Trygger Foundation, The Swedish Research Council Formas and the National Environment Research Council (UK).

http://www.eurekalert.org/pub_releases/2011-08/cchm-ipm080911.php

Intestinal protein may have role in ADHD, other neurological disorders

A biochemical pathway long associated with diarrhea and intestinal function may provide a new therapeutic target for treating ADHD other neuropsychiatric disorders

CINCINNATI – A biochemical pathway long associated with diarrhea and intestinal function may provide a new therapeutic target for treating ADHD (Attention Deficit Hyperactivity Disorder) other neuropsychiatric disorders, according to a team of scientists from China and the United States reporting Aug. 11 in Science.

Scientists have for the last quarter century studied the intestinal membrane receptor protein, guanylyl cyclase-C (GC-C) for its role in diarrheal disease and other intestinal functions, according to Mitchell Cohen, M.D., U.S. author on the study and director of Gastroenterology, Hepatology and Nutrition at Cincinnati Children's Hospital Medical Center. In fact, it had been thought that GC-C was found primarily in the intestine.

In the current study, scientists in China who collaborated with Dr. Cohen discovered that the receptor is also expressed in critical areas of the brain. The senior author on the study is Dr. Minmin Luo, a researcher at the National Institute of Biological Sciences and Tsinghua University in Beijing.

Using a mouse model developed in Dr. Ralph Giannella's laboratory at the University of Cincinnati, in which the GC-C receptor is deleted, or knocked out, the researchers found the mice exhibit hyperactivity and attention deficits. It is the first time that GC-C has been linked to neuropsychiatric disorder, according to the researchers.

"We show that the neurons selectively express GC-C and that its activation amplifies the excitatory responses mediated by other receptors on dopamine neurons in the midbrain," said Dr. Luo. "Working through a protein kinase called PKG, GC-C activity increases brain dopamine levels and thus regulate mouse attention and activity level." When the researchers treated the GC-C knockout mice with amphetamine-based ADHD medication and a PKG activator, it reversed their hyperactive, inattentive behavior.

"The results indicate important behavioral and physiological functions for the GC-C/PKG signaling pathway in the brain," said Dr. Luo. "The data also suggest new therapeutic targets for neuropsychiatric disorders related to malfunctions of midbrain dopamine receptors."

One of the most prevalent human behavioral disorders, ADHD has been linked to imbalances in the dopamine system. The researchers noted in the study that its findings – mice exhibiting reduced dopamine levels and related behavioral problems – are consistent with the biochemical characteristics of human ADHD.

"This could make the GC-C knockout mouse a good research model for ADHD and other behavioral disorders," said Dr. Cohen. "Efforts to develop activators or inhibitors of the GC-C/PKG signaling pathway may lead to novel treatments for other disorders, such schizophrenia, Parkinson's disease and addiction."

The first author on the study is Rong Gong, who is in the joint graduate program of Peking Union Medical College and the National Institute of Biological Sciences in Beijing. Other institutions collaborating on the study include: the College of Life Sciences, Beijing Normal University and the Wuhan Institute of Physics and Mathematics at the China Academy of Sciences in Wuhan, China.

http://www.eurekalert.org/pub_releases/2011-08/hcfa-awi081111.php

Alien world is blacker than coal

Astronomers have discovered the darkest known exoplanet

Astronomers have discovered the darkest known exoplanet - a distant, Jupiter-sized gas giant known as TrES-2b. Their measurements show that TrES-2b reflects less than one percent of the sunlight falling on it, making it blacker than coal or any planet or moon in our solar system. "TrES-2b is considerably less reflective than black acrylic paint, so it's truly an alien world," said astronomer David Kipping of the Harvard-Smithsonian Center for Astrophysics (CfA), lead author on the paper reporting the research.

In our solar system, Jupiter is swathed in bright clouds of ammonia that reflect more than a third of the sunlight reaching it. In contrast, TrES-2b (which was discovered in 2006 by the Trans-Atlantic Exoplanet Survey, or TrES) lacks reflective clouds due to its high temperature.

TrES-2b orbits its star at a distance of only three million miles. The star's intense light heats TrES-2b to a temperature of more than 1,800° Fahrenheit - much too hot for ammonia clouds. Instead, its exotic atmosphere contains light-absorbing chemicals like vaporized sodium and potassium, or gaseous titanium oxide. Yet none of these chemicals fully explain the extreme blackness of TrES-2b.

"It's not clear what is responsible for making this planet so extraordinarily dark," stated co-author David Spiegel of Princeton University. "However, it's not completely pitch black. It's so hot that it emits a faint red glow, much like a burning ember or the coils on an electric stove."

Kipping and Spiegel determined the reflectivity of TrES-2b using data from NASA's Kepler spacecraft. Kepler is designed to measure the brightnesses of distant stars with extreme precision.

The team monitored the brightness of the TrES-2 system as the planet orbited its star. They detected a subtle dimming and brightening due to the planet's changing phase. TrES-2b is believed to be tidally locked like our moon, so one side of the planet always faces the star. And like our moon, the planet shows changing phases as it orbits its star. This causes the total brightness of the star plus planet to vary slightly.

"By combining the impressive precision from Kepler with observations of over 50 orbits, we detected the smallest-ever change in brightness from an exoplanet: just 6 parts per million," said Kipping. "In other words, Kepler was able to directly detect visible light coming from the planet itself."

The extremely small fluctuations proved that TrES-2b is incredibly dark. A more reflective world would have shown larger brightness variations as its phase changed.

Kepler has located more than 1,200 planetary candidates in its field of view. Additional analysis will reveal whether any other unusually dark planets lurk in that data.

TrES-2b orbits the star GSC 03549-02811, which is located about 750 light-years away in the direction of the constellation Draco. (One light-year is about 6 trillion miles.)

http://www.newscientist.com/blogs/shortsharpscience/2011/08/worlds-oldest-wood-found.html

Found: World's oldest wood – so far

Michael Marshall, environment reporter

Two 400-million-year-old fossil plants are the oldest known examples of wood.

They are small herbs, suggesting that wood did not evolve to help plants grow tall.

Both fossils date from the early Devonian period, by which time simple plants had long colonised the land and begun diversifying. One was found in France and dates from 407 million years ago, while the other, from Canada, is 397 million years old.

According to lead researcher Philippe Gerrienne of the University of Liège, Belgium, they predate the previous record-holders by at least 10 million years.

Trees would not evolve until about 385 million years ago, at which point they began scrambling to grow taller in order to capture more light. Wood was crucial for this, because it made their trunks sturdier.

But Gerrienne thinks that was not why wood first evolved. His fossils are stems only 12 centimetres long, so they wouldn't need the support. Instead he thinks the wood improved the flow of water up the stems.

Journal reference: Science, vol 333, p 837

http://www.eurekalert.org/pub_releases/2011-08/ason-wsp081111.php

Warning signs predict kidney injury after surgery

Markers may transform how kidney disease is diagnosed

Washington, DC Acute kidney injury (AKI) is a common – but preventable -- complication after surgery that can lead to other complications or even death. The use and development of biomarkers will help physicians diagnose and treat acute kidney injury. Three protein measurements indicate who has a high risk of developing kidney injury after heart surgery, according to two studies appearing in an upcoming issue of the Journal of the American Society of Nephrology.

"To date, these are the largest studies in adults and children comparing and validating the performance of three of the most frequently studied markers of kidney injury," said author Chirag Parikh, MD, PhD (Yale University School of Medicine).

The studies included more than 1,200 adults and 300 children undergoing heart surgery throughout North America. Frequent urine and blood samples were collected to measure levels of three proteins -- urine interleukin-18 (IL-18) and urine and plasma (blood) neutrophil gelatinase-associated lipocalin (NGAL)—and assess their ability to predict who will develop kidney injury after surgery.

Traditionally, kidney trouble is assessed by measuring the blood protein creatinine, which is not ideal because it has a delayed result—it does not pick up early damage and injury to the kidneys.

"We demonstrated that the three proteins in our study identify kidney injury soon after surgery and 24 to 48 hours earlier than creatinine, and shows a similar result," according to Parikh.

Risk of kidney injury was especially high—more than six times higher—for adults and children with the highest levels of urine IL-18. Plasma NGAL also predicted kidney injury in adults, whereas urine NGAL was not an accurate predictor in adults once results were adjusted for other factors. Urine IL-18 and urine, but not plasma, NGAL were accurate predictors in children.

Doctors may wish to measure these urine or blood proteins immediately after surgery to predict which patients are at high risk of developing kidney injury. These patients might benefit from kidney protective therapies.

The studies' results could also transform the diagnosis of kidney disease, Parikh believes. "Developing markers of structural kidney damage, before kidney function fails, is a top priority," he said.

The research's main limitation was that the adults enrolled were mainly Caucasian. Future studies should consider whether the results are the same in other races.

Other members of the research team (www.yale.edu\tribeaki) included Amit X. Garg, Steven G. Coca, Prasad Devarajan, Charles Edelstein, Richard W. Kim, Jay L. Koyner, Catherine D. Krawczeski, Simon Li, Cary S. Passik, Uptal D. Patel, Michael G. Shlipak, Kyaw Sint, Madhav Swaminathan, Heather Thiessen-Philbrook, Zhu Wang, and Michael Zappitelli.

Dr. Edelstein and Dr. Parikh are named co-inventors on the IL-18 patent. Dr. Devarajan is a co-inventor on the NGAL patents.

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The articles, entitled "Postoperative Biomarkers Predict Acute Kidney Injury and Poor Outcomes after Adult Cardiac Surgery" (doi 10.1681/ASN.2010121302) and " Postoperative Biomarkers Predict Acute Kidney Injury and Poor Outcomes after Pediatric Cardiac Surgery"" (doi 10.1681/ASN.2010111163 will appear online at http://jasn.asnjournals.org/).

http://www.newscientist.com/article/dn20788-experimental-drug-could-defeat-any-virus.html

Experimental drug could defeat any virus

11:00 12 August 2011 by Ferris Jabr

Viruses might soon meet their kryptonite: an experimental drug that can, in theory, obliterate cells infected by any type of virus without harming healthy neighbours.

For 50 years, we have been fighting viruses in two ways: drugs for existing infections and vaccines to prevent infection in the first place. However, most drugs or vaccines are specific to one virus, viral strain or family of related viruses. When a virus mutates – as they so often do – researchers must retool our medicines.

The new drug targets a molecule common to all virus-infected cells. Nearly every virus generates strings of double-stranded RNA longer than 30 base pairs during transcription and replication, in an attempt to duplicate itself and commandeer its host cell's machinery. Healthy mammalian cells do not produce double-stranded RNA longer than 23 base pairs.

The immune artillery within mammalian cells includes a protein that exploits this viral characteristic. Todd Rider of the Massachusetts Institute of Technology's Lincoln Laboratory in Lexington, Massachusetts, and his colleagues combined this protein with another from the immune system to produce their new drug.

When a sentinel enzyme called protein kinase R (PKR) finds double-stranded RNA longer than 23 base pairs inside a cell, it binds to the RNA, blocks the production of viral proteins and activates the cell's defences. Many viruses, though, have evolved ways to evade PKR.

Unleash the enzymes

So Rider and his colleagues glued PKR to apoptotic protease activating factor 1 (APAF-1), a protein that triggers cell suicide by unleashing a team of destructive enzymes. Healthy cells normally reserve APAF-1 for extreme situations – to trigger self-destruction in a cancerous cell, for instance – but as part of the new antiviral drug, APAF-1 is activated as soon as PKR identifies and binds to lengthy molecules of double-stranded RNA in an infected cell.

The drug "catches the virus with its pants down", by destroying the cell before new viruses have been assembled inside it, explains Rider. Even if fragmented virus molecules escape the obliterated cell, they will be missing the protein coat that helps them to travel between cells, and so will not infect surrounding healthy tissue. Rider calls his drug double-stranded RNA (dsRNA)-activated caspase oligomeriser (DRACO).

In tests, Rider infected human and mouse cells in Petri dishes with rhinovirus, which causes some forms of the common cold in humans. DRACO prevented the virus from spreading by rapidly killing infected cells without harming healthy ones. Further tests showed that DRACO performs just as well against 14 other viruses, including the one responsible for dengue fever. It also helped boost survival rates in mice given an otherwise lethal dose of the H1N1 flu virus.

"Just as antibiotics revolutionised the treatment of bacterial infections, this project has a lot of potential to prevent or treat a whole range of infectious illnesses," says Rider. Infections on the hit list range from "the common cold to quite serious diseases – [and] even [the most] drug-resistant HIV", he says. DRACO could also act a shield against viruses that might appeal to bioterrorists, such as Ebola and smallpox.

Draconian measures

Andrea Branch of the Mount Sinai School of Medicine in New York City thinks the work is intriguing but has some reservations about its practicality. She points out that DRACO is a large protein, which may not enter cells easily.

That said, she agrees that administering DRACO early in an infection could be effective – but adds that destroying all cells infected with the virus can be dangerous in people with advanced viral infections. "Suppose 100 per cent of your hepatocytes [liver cells] are infected and you used this – you would die of liver failure."

Timothy Tellinghuisen of the Scripps Research Institute in Jupiter, Florida, adds that some viruses have evolved ways to conceal their double-stranded RNA, and so could elude DRACO. Still, "this is really an interesting paper, a very clever approach to getting rid of cells containing double-stranded RNA", he says.

Journal reference: PLoS One, DOI: 10.1371/journal.pone.0022572

http://www.eurekalert.org/pub_releases/2011-08/sumc-sdm081211.php

Stanford discovery may eliminate potentially lethal side effect of stem cell therapy

STANFORD, Calif. - ***Like fine chefs, scientists are seemingly approaching a day when they will be able to make nearly any type of tissue from human embryonic stem cells.***

You need nerves or pancreas, bone or skin? With the right combination of growth factors, skill and patience, a laboratory tissue culture dish promises to yield therapeutic wonders. But within these batches of newly generated cells lurks a big potential problem: Any remaining embryonic stem cells - those that haven't differentiated into the desired tissue - can go on to become dangerous tumors called teratomas when transplanted into patients.

Now researchers at the Stanford University School of Medicine have developed a way to remove these pluripotent human embryonic stem cells from their progeny before the differentiated cells are used in humans. ("Pluripotent" describes cells that are able to become all types of adult tissue.)

"The ability to do regenerative medicine requires the complete removal of tumor-forming cells from any culture that began with pluripotent cells," said Irving Weissman, MD, director of the Stanford Institute for Stem Cell Biology and Regenerative Medicine. "We've used a combination of antibodies to weed out the few undifferentiated cells that could be left in the 10 or 100 million differentiated cells that make up a therapeutic dose."

Weissman pointed out that the production of therapeutic cells from pluripotent stem cells for regenerative medicine was a major goal of Proposition 71, the ballot measure that established the California Institute for Regenerative Medicine to allocate $3 billion to advance stem cell science. CIRM funded this research.

The scientists believe the technique could also be used to remove residual tumor-initiating cells from populations of cells derived from induced pluripotent stem, or iPS, cells. These cells may also be useful for therapy but, unlike embryonic stem cells, iPS cells are created in the laboratory from adult tissue.

"Commonly used differentiation protocols for embryonic stem and iPS cells often give rise to mixed cultures of cells," said research associate Micha Drukker, PhD. "Because even a single undifferentiated cell harbors the ability to become a teratoma, we sought to develop a way to remove these cells before transplantation."

Drukker is the senior author of the research, which will be published online Aug. 14 in Nature Biotechnology. Stanford medical student Chad Tang is the first author. Weissman, who is also the Virginia and D.K. Ludwig Professor for Clinical Investigation and Cancer Research and a member of the Stanford Cancer Institute, is a co-author. The research was conducted in his lab.

Teratomas are the Frankensteins of the tumor world - a hodgepodge of tissues like teeth, hair and bone. They owe their remarkable composition to the fact that the cells from which they arise early in development are pluripotent. In fact, the ability to form teratomas in animals is a defining feature of true pluripotent cells.

But the very feature that confirms a cell's pluripotency also makes it potentially dangerous to use therapeutically. That's why Tang, Drukker and Weissman decided to try to develop an antibody that would recognize and bind to only pluripotent cells and enable their removal from a mixture of cells. Although a few such antibodies already existed, they were not specific enough on their own to completely weed out the tumor-causing cells.

The researchers studied two sets of antibodies - one commercially available and one they generated themselves - to identify which among them bound most strongly to pluripotent, but not differentiated, cells. They found one newly generated antibody that was highly specific for a previously unknown marker on undifferentiated cells that they termed stage-specific embryonic antigen-5, or SSEA-5. The cells bound by this antibody, anti-SSEA-5, expressed high levels of pluripotent-specific genes and resembled embryonic stem cells in appearance. Anti-SSEA-5 also bound strongly to the inner cell mass of an early human embryo, the group of cells from which embryonic stem cell lines are derived.

When the researchers injected human embryonic stem cells recognized by anti-SSEA-5 into mice, they found that in seven out of seven times, the cells formed rapidly growing teratomas. However, cells that were not bound by anti-SSEA-5 formed smaller teratomas in only three of 11 experiments. Combining anti-SSEA-5

with two other antibodies known to bind to pluripotent cells completely separated the pluripotent from the differentiated cells, although the researchers did see some smaller, less-diverse growths in some cases.

Upon analysis, Tang and his colleagues found that anti-SSEA-5 recognizes and binds to a cell-surface carbohydrate structure called a glycan. As the pluripotent cell differentiates, this glycan is modified to other glycan structures not recognized by the antibody.

"The study of glycans is becoming an active area of stem cell biology," said Tang. "Many glycans are highly expressed in embryonic stem cells, but not in differentiated cells. This warrants further study and may lead to new understandings about embryonic stem cell biology."

In addition to Tang, Drukker and Weissman, other Stanford researchers who participated in the study include graduate student Andrew Lee; postdoctoral scholar Jens-Peter Volkmer, MD; instructor of pathology Debashis Sahoo, PhD; undergraduate student Divya Nag; research assistant Adriane Mosley; research associate Matthew Inlay; instructor of medicine Reza Ardehali, MD; postdoctoral scholar Shawn Chavez, PhD; professor of obstetrics and gynecology and director of Stanford's Human Embryonic Stem Cell Research and Education Center Renee Reijo Pera, PhD; professor of obstetrics and gynecology Barry Behr, PhD; and associate professor of cardiovascular medicine and of radiology Joseph Wu, MD, PhD.

Stanford University has filed for patent protection for the use of monoclonal antibody-based protocols to remove teratogenic pluripotent stem cells from a cell mixture.

In addition to CIRM, the research was supported by the Howard Hughes Medical Institute and the Deutsche Forschungsgemeinschaft.

http://www.eurekalert.org/pub_releases/2011-08/uoc--rro080511.php

Recurrence risk of autism in younger siblings higher than thought

The risk among male children is greater than 25 percent, and over 30 percent for 'multiplex' families

The risk that an infant with an older sibling with autism also will develop the disorder, previously estimated at between 3 and 10 percent, is substantially higher at approximately 19 percent, a large, international, multi-site study led by researchers at the UC Davis MIND Institute has found. While the study found a combined estimated risk for all participants of nearly19 percent, it found an even more elevated risk of recurrence of over 26 percent for male infants, and over 32 percent for infants with more than one older sibling with autism.

The study is the largest prospective investigation of autism spectrum disorder and sibling recurrence to date. It is published online today and will appear in print in the September issue of the journal Pediatrics.

The study has important implications both for genetic counseling for parents and for referral to early intervention for the infant siblings of children with autism if concerns arise about their development, said Sally Ozonoff, professor of psychiatry and behavioral sciences at the MIND Institute and the study's lead author.

"This is the largest study of the siblings of children with autism ever conducted," Ozonoff said. "There is no previous study that identified a risk of recurrence that is this high," she said.

Autism is a complex disorder that affects a child's ability to think, communicate, interact socially and learn. The U.S. Centers for Disease Control and Prevention places the incidence of autism at 1 in 110 children born today.

The participants in the study were enrolled in separate studies that are part of the Baby Siblings Research Consortium, an international network supported by Autism Speaks that pools data from individually funded research sites to facilitate the study of infants at high risk of developing autism because they have an older sibling with the condition. There is strong evidence that genetic factors play a critical role in vulnerability for developing autism.

Twelve consortium sites located in the United States and Canada participated in the study, with additional sites as far away as Israel engaged in analyses and interpretation of the data. The study included 664 subjects, infants whose average age at enrollment was 8 months, with two-thirds recruited prior to 6 months of age. The researchers followed the participants' development until 36 months, when they were tested for autism.

The study subjects were tested using the Autism Diagnostic Observation Schedule (ADOS), an autism diagnostic tool, and the Mullen Scales of Early Learning, which measures nonverbal cognitive, language and motor skills. Of the 664 participants, a total of 132 infants met the criteria for an autism spectrum disorder. Fifty-four received a diagnosis of autistic disorder and 78 received a diagnosis of Pervasive Developmental Delay Not Otherwise Specified, considered a milder form of autism.

More males than females are affected with autism — 80 percent of all affected children are male. The risk to male children held true in the current study. Among the study participants, 26.2 percent of male infants versus 9 percent of female infants were diagnosed with an autism spectrum disorder.

The overall rate of autism spectrum outcomes for all study participants was 18.7 percent. However, there was a significant difference in the recurrence rate based on whether the child had one sibling or more than one

sibling with autism. In families with one older child with autism, or simplex families, the rate of incidence was 20.1 percent. Only 37 of the study participants had more than one sibling with autism. But for those families, called multiplex families, the recurrence rate was 32.2 percent.

"It's important to recognize that these are estimates that are averaged across all of the families. So, for some families, the risk will be greater than 18 percent, and for other families it would be less than 18 percent. At the present time, unfortunately, we do not know how to estimate an individual family's actual risk," Ozonoff said.

Ozonoff said that the study's large size, prospective design, the young age of study participants at enrollment and the gold-standard direct assessment methods used, as well as the geographic diversity of participants, reinforce the accuracy of its findings. The study design also minimized the effects of other factors such as "stoppage," the tendency of families with a child with autism to stop having children, which would lead to an underestimate of potential recurrence rates. The study accounted for stoppage by studying only families with later-born siblings.

She said that the study has significant family-planning and genetic-counseling implications.

"Parents often ask what their risk of having another child with ASD is and, until now, we were really not sure of the answer," she said.

The study also highlights the critical importance of routine surveillance and rapid referral for treatment of infant siblings of children with autism. Ozonoff said that it is of paramount importance that primary care professionals monitor these children's development closely and refer them for early intervention immediately when concerns arise.

In practice guidelines published by the American Academy of Pediatrics in 2007, being a younger sibling of a child with autism is considered a risk factor requiring special developmental evaluation and the current investigation supported that recommendation.

"This study shows that the younger siblings of children with autism spectrum disorders need to be tracked very carefully, and this may require more than the normal surveillance that a pediatrician might typically do," Ozonoff said. "This should include very explicitly and regularly checking in with parents on whether developmental milestones are being reached."

More information about Ozonoff's research can be found at the UC Davis MIND Institute's Infant Sibling Study Web page. For more information about the early signs of autism, visit the Autism Speaks Learn the Early Signs Web page.

Other authors of the study are Gregory S. Young and Sally J. Rogers of the UC Davis MIND Institute; Alice Carter of the University of Massachusetts, Boston; Daniel Messinger of the University of Miami; Nurit Yirmiya of the University of Jerusalem; Lonnie Zwaigenbaum of the University of Alberta, Canada; Susan Bryson of Dalhousie University, Halifax, Nova Scotia; Leslie J. Carver and Karen Dobkins of UC San Diego; John Constantino of Washington University, St. Louis; Ted Hutman and Marian Sigman of UCLA; Jana M. Iverson of the University of Pittsburgh; Rebecca Landa of Kennedy Krieger Institute; and Wendy L. Stone of the University of Washington, Seattle.

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http://medicalxpress.com/news/2011-08-virus-high-blood-pressure-chinese.html

Virus can cause high blood pressure: Chinese study

High blood pressure could be caused by a common virus, according to a study carried out by a team of Chinese doctors which could lead to better treatment for millions of people around the world.

The team from Beijing Chaoyang Hospital's cardiology centre says it has found the first evidence of a link between the human cytomegalovirus (HCMV) and the most commonly occurring form of hypertension, or high blood pressure. The virus infects most people at some time during their lives, but frequently causes no symptoms, so goes undetected.

One of the report's authors, Yang Xinchun, told AFP the findings could eventually lead to the development of a vaccine to control or prevent high blood pressure. "If we can get conclusive evidence of the relationship, we can get better medical vaccines and remedies for hypertension," said Yang, head of the cardiology centre.

However, he added that it was "too early" to say when a vaccine could be available and his research was still in its early stages. "It is the first time someone managed to find this relationship... so we need to undergo more tests with a wider scope of patients," he said.

The study could have widespread health implications -- the World Health Organization says around a billion people worldwide suffer from high blood pressure, including more than 200 million Chinese. The vast majority experience so-called essential hypertension, which has no proven cause, but has been previously associated with genetic factors and unhealthy lifestyles. Chinese doctors believe that variety is linked to the HCMV virus.

The results of their study were published in the US medical journal Circulation, whose former chief editor James Willerson posted comments on Beijing Chaoyang Hospital's website.

The findings "might present a new strategy for preventing and treating cardiovascular disease," he said.

A recent study led by Jiang He, a professor at the the Tulane University School of Public Health and Tropical Medicine in New Orleans, found that high blood pressure plays a part in 2.3 million cardiovascular deaths in China each year. Of these 1.3 million were "premature" deaths, meaning they occurred before the age of 72 in men and 75 in women, the average life expectancy in China in 2005.

"Increased blood pressure is the leading preventable risk factor for premature mortality in the Chinese general population," the authors said, describing their findings as "striking and unexpected". *(c) 2011 AFP*

http://www.eurekalert.org/pub_releases/2011-08/ddps-rdg081511.php

Researchers demonstrate green tea is effective in treating genetic disorder and types of tumors

ST. LOUIS, MO - ***A compound found in green tea shows great promise for the development of drugs to treat two types of tumors and a deadly congenital disease.***

The discovery is the result of research led by Principal Investigator, Dr. Thomas Smith at The Donald Danforth Plant Science Center and his colleagues at The Children's Hospital of Philadelphia. Their findings are published in the recent article, "Green Tea Polyphenols Control Dysregulated Glutamate Dehydrogenase In Transgenic Mice By Hijacking The ADP Activation Site" in The Journal of Biological Chemistry.

Glutamate dehydrogenase (GDH) is found in all living organisms and is responsible for the digestion of amino acids. In animals, GDH is controlled by a complex network of metabolites. For decades it was not clear why animals required such regulation but other kingdoms did not. This was partially answered by the Stanley group's finding that a deadly congenital disease, hyperinsulinism/hyperammonemia (HHS), is caused by the loss of some of this regulation. In this disorder, patients (typically children) respond to the consumption of protein by over secreting insulin, becoming severely hypoglycemic, often leading to death.

Using atomic structures to understand the differences between animals and plants, Dr. Smith and his colleagues discovered that two compounds found naturally in green tea are able to compensate for this genetic disorder by turning off GDH in isolated and when the green tea compounds were administered orally. The Smith lab also used X-ray crystallography to determine the atomic structure of these green tea compounds bound to the enzyme. With this atomic information, they hope to be able to modify these natural compounds to design and develop better drugs.

Interestingly, two other research groups have validated and extended these findings to demonstrate that blocking GDH with green tea is very effective at killing two different kinds of tumors; glioblastomas, an aggressive type of brain tumor, and tuberous sclerosis complex disorder, a genetic disease that causes non-malignant tumors to grow on a number of organs.

"While these compounds from green tea are extremely safe and consumed by millions every day, they have a number of properties that make them difficult to use as actual drugs. Nevertheless, our ongoing collaboration with the Stanley lab shows that there are natural compounds from plants that can control this deadly disorder and, with the atomic structure in hand, can be used as a starting point for further drug design."

http://www.eurekalert.org/pub_releases/2011-08/uof-rdo081511.php

Researchers discover oldest evidence of nails in modern primates

GAINESVILLE, Fla. — ***From hot pink to traditional French and Lady Gaga's sophisticated designs, manicured nails have become the grammar of fashion.***

But they are not just pretty — when nails appeared on all fingers and toes in modern primates about 55 million years ago, they led to the development of critical functions, including finger pads that allow for sensitive touch and the ability to grasp, whether it's a nail polish brush or remover to prepare for the next trend.

In a new study co-authored by University of Florida scientists, researchers recovered and analyzed the oldest fossil evidence of fingernails in modern primates, confirming the idea nails developed with small body size and disproving previous theories nails evolved with an increase in primate body size. More than 25 new specimens of Teilhardina brandti – an extinct primate originally described from a single lower molar — include pieces of upper teeth and ankle bones that show the mammal lived in trees. Its nails allowed the lemur-like animal to grasp onto branches and move through the trees with more agility, researchers said.

"If you take all the primates that are alive today, they're all going to have characteristics that look the same, but unlike people, many of them live in trees," said co-author Jonathan Bloch, an associate curator of vertebrate paleontology at the Florida Museum of Natural History on the UF campus. "By finding parts of the skeleton of this primitive primate, we are able to test whether nails were present in the common ancestor of the group that includes lemurs, monkeys, and humans — it's direct evidence as opposed to speculation."

Appearing in the current online edition of the American Journal of Physical Anthropology, the study provides a better understanding of the evolutionary relationships of one of the oldest known modern primates,

as well as the time frame and environmental conditions that allowed for the development of nails on all fingers and toes, an exclusive feature among primates.

Specimens of T. brandti were collected over the last seven years in northwestern Wyoming's Bighorn Basin and represent the earliest North American species from the group of euprimates, also known as "true" primates. The fossils date to the early Eocene epoch, about 55.8 million years ago, at the same time as a 200,000-year global warming event known as the Paleocene-Eocene Thermal Maximum occurred, Bloch said. Mammals evolved to be smaller during that time, when even- and odd-toed hoofed mammals, distantly related to modern deer and horses, also first appeared in the fossil record.

"The appearance of the first modern primates in North America co-occurred with the appearance of other modern mammals such as horses, and it's all associated with a major global warming event," said co-author Stephen Chester, a Yale University doctoral student and research associate at UF. "It in part set the stage for what we see today in terms of modern mammalian biodiversity."

Less than 6 inches long, T. brandti was omnivorous, Bloch said. While archaic primates mostly had claws, some of the characteristics of modern primates include forward-facing eyes, an enlarged brain and nails on all digits. "They are the smallest true nails known on record, whether living or fossil," said first author Ken Rose, a professor in the Center for Functional Anatomy & Evolution at Johns Hopkins University School of Medicine. "That certainly doesn't suggest nails developed with larger bodies."

Based on the age of the fossils and analyses of Teilhardina species from other parts of the world, researchers were also able to analyze the hypothesis that mammals migrated from Asia into North America. Instead, they likely passed from Asia, through Europe and into North America on high-latitude land connections.

"This research really suggests that we are looking at something extremely close [to the species found in Europe] and that's of great interest in itself," Rose said. "We can show these species were extremely close morphologically in time and found in Europe and Wyoming."

During the Paleocene-Eocene Thermal Maximum, average temperatures were about 15 degrees Fahrenheit higher than today, and the large variety of mammals found in the fossil record from that time remains a mystery to scientists.

"The finding of this animal and the concentrated effort of this period of time might be one of those things where the closer you look, the less you know," said Gregg Gunnell, director of the Division of Fossil Primates at the Duke Lemur Center. "But any time we have the opportunity to add more morphological information to analyze the relationships of animals to answer these biogeographic questions, we can hopefully get closer and closer to an understanding of what led to this big radiation (diversification) of primates in the first place."

Study co-authors also include Rachel Dunn of Johns Hopkins University and Doug Boyer of Brooklyn College, City University of New York. The research was supported by the National Science Foundation and Yale University.

http://www.eurekalert.org/pub_releases/2011-08/ru-me081511.php

More evidence that caffeine lowers risk of skin cancer

There might be a time when instead of just drinking that morning cup of coffee you lather it on your skin as a way of preventing harmful sun damage or skin cancer.

A new Rutgers study strengthens the theory that caffeine guards against certain skin cancers at the molecular level by inhibiting a protein enzyme in the skin, known as ATR. Scientists believe that based on what they have learned studying mice, caffeine applied directly to the skin might help prevent damaging UV light from causing skin cancer.

Prior research indicated that mice that were fed caffeinated water and exposed to lamps that generated UVB radiation that damaged the DNA in their skin cells were able to kill off a greater percentage of their badly damaged cells and reduce the risk of cells becoming cancerous.

"Although it is known that coffee drinking is associated with a decreased risk of non-melanoma skin cancer, there now needs to be studies to determine whether topical caffeine inhibits sunlight-induced skin cancer," said Allan Conney, director of the Susan Lehman Cullman Laboratory for Cancer Research.

In this newly-published study, instead of inhibiting ATR with caffeinated water, Rutgers researchers, in collaboration with researchers from the University of Washington, genetically modified and diminished ATR in one group of mice. The results: the genetically modified mice developed tumors more slowly than the unmodified mice, had 69 percent fewer tumors than regular mice and developed four times fewer invasive tumors.

The study also found, however, that when both groups of mice were exposed to chronic ultraviolet rays for an extended period of time, tumor development occurred in both the genetically modified and regular mice. What this seems to indicate, says Conney, is that inhibiting the ATR enzyme works best at the pre-cancerous stage before UV-induced skin cancers are fully developed.

According to the National Cancer Institute, sunlight-induced skin cancer is the most prevalent cancer in the United States with more than 1 million new cases each year. Although multiple human epidemiologic studies link caffeinated beverage intake with significant decreases in several different types of cancer, including skin cancer, just how and why coffee protects against the disease is unknown.

"Caffeine might become a weapon in prevention because it inhibits ATR and also acts ad as a sunscreen and directly absorbs damaging UV light," said Conney.

http://www.newscientist.com/article/mg21128255.100-wire-robot-yanks-your-golf-game-into-shape.html

Wire robot yanks your golf game into shape

15 August 2011 by Paul Marks

A robotic aid made from wires could pull your game into shape

IS YOUR golf game lacking a finishing touch? Driving your balls straight down the fairway but struggling when it comes to the finer art of putting? Never fear: a robotic aid made from wires could pull your game into shape.

The novel training machine has been created by Katherine Kuchenbecker at the University of Pennsylvania in Philadelphia and colleagues, who specialise in haptics - the technology of tactile feedback.

The final stage of a hole of golf occurs on the green, when a player attempts to tap the ball into a 10-centimetre-wide hole using a flat-faced club called a putter. To do this consistently well, players must adopt a steady stance while ensuring the putter's flat face is directed at the hole during their swing's follow-through. There is little room for error, as small changes in the putter's angle send the ball's trajectory askew. What's needed, says team member Jacquelyn Kunkel, is a way of teaching a player how to putt that instils the correct muscle memory of the action.

To do this, the team created a 1.5-metre-long by 70-centimetre wide-metal training frame with a green baize base and a practice hole. The player stands at the centre, using a practice putter with four steel wires attached, two to the front and two to the back. These wires go to the four corners of the frame, where they are each attached to an electric motor, which can alter the wires' tension.

As the player swings the putter, fast-acting software controlling the tension of the wires corrects any deviation to the swing, keeping it straight. "The golfer feels forces pulling them back to the correct position when they go wrong, making it feel natural to swing correctly," says Kunkel.

Early tests show promise, with some volunteers having less variation in shot accuracy, the team told the World Haptics Conference 2011 in Istanbul, Turkey, in June. "The cues it delivers are subtle, not even noticeable if you're trying to putt straight," says Kuchenbecker. "The system helps you feel what it is like to putt correctly, and the ball tends to end up closer to the target." They plan to improve the system by reducing the friction in the wires so the altered swing feels more natural.

Many golf training devices make the user rigidly follow a certain line, says Steve Otto, director of research and testing at the R&A in St Andrews, UK, the governing body of golf outside the US. "But I haven't seen a training device that lets you deviate and which then brings you back on line. That sounds an interesting new approach."