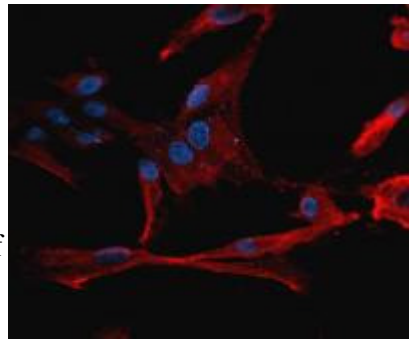
Discovery of taste receptors in the lungs could help people with asthma breathe easier

University of Maryland School of Maryland researchers show bitter compounds open lung airways better than current drugs

Taste receptors in the lungs? Researchers at the University of Maryland School of Medicine in Baltimore have discovered that bitter taste receptors are not just located in the mouth but also in human lungs. What they learned about the role of the receptors could revolutionize the treatment of asthma and other obstructive lung diseases.

"The detection of functioning taste receptors on smooth muscle of the bronchus in the lungs was so unexpected that we were at first quite skeptical ourselves," says the study's senior author, Stephen B. Liggett, M.D., professor of medicine and physiology at the University of Maryland School of Medicine and director of its Cardiopulmonary Genomics Program.



This is a slide of lung taste receptors through a microscope. Red bands are receptors, blue dots are nuclei. **University of Maryland School of Medicine**

Dr. Liggett, a pulmonologist, says his team found the taste receptors by accident, during an earlier, unrelated study of human lung muscle receptors that regulate airway contraction and relaxation. The airways are the pathways that move air in and out of the lungs, one of several critical steps in the process of delivering oxygen to cells throughout the body. In asthma, the smooth muscle airways contract or tighten, impeding the flow of air, causing wheezing and shortness of breath.

The taste receptors in the lungs are the same as those on the tongue. The tongue's receptors are clustered in taste buds, which send signals to the brain. The researchers say that in the lung, the taste receptors are not clustered in buds and do not send signals to the brain, yet they respond to substances that have a bitter taste.

For the current study, Dr. Liggett's team exposed bitter-tasting compounds to human and mouse airways, individual airway smooth muscle cells, and to mice with asthma. The findings are published online in Nature Medicine.

Most plant-based poisons are bitter, so the researchers thought the purpose of the lung's taste receptors was similar to those in the tongue – to warn against poisons. "I initially thought the bitter-taste receptors in the lungs would prompt a 'fight or flight' response to a noxious inhalant, causing chest tightness and coughing so you would leave the toxic environment, but that's not what we found," says Dr. Liggett.

There are thousands of compounds that activate the body's bitter taste receptors but are not toxic in appropriate doses. Many are synthetic agents, developed for different purposes, and others come from natural origins, such as certain vegetables, flowers, berries and trees.

The researchers tested a few standard bitter substances known to activate these receptors. "It turns out that the bitter compounds worked the opposite way from what we thought," says Dr. Liggett. "They all opened the airway more profoundly than any known drug that we have for treatment of asthma or chronic obstructive pulmonary disease (COPD)." Dr. Liggett says this observation could have implications for new therapies. "New drugs to treat asthma, emphysema or chronic bronchitis are needed," he says. "This could replace or enhance what is now in use, and represents a completely new approach."

Quinine and chloroquine have been used to treat completely different diseases (such as malaria), but are also very bitter. Both of these compounds opened contracted airways profoundly in laboratory models. Even saccharin, which has a bitter aftertaste, was effective at stimulating these receptors. The researchers also found that administration of an aerosolized form of bitter substances relaxed the airways in a mouse model of asthma, showing that they could potentially be an effective treatment for this disease.

Dr. Liggett cautions that eating bitter tasting foods or compounds would not help in the treatment of asthma. "Based on our research, we think that the best drugs would be chemical modifications of bitter compounds, which would be aerosolized and then inhaled into the lungs with an inhaler," he says.

Another paradoxical aspect of their discovery is the unexpected role that the mineral calcium plays when the lung's taste receptors are activated. The study's principal author, Deepak A. Deshpande, Ph.D., assistant professor of medicine at the University of Maryland School of Medicine, is an expert in how calcium controls muscles. "We always assumed that increased calcium in the smooth muscle cell caused it to contract, but we found that bitter compounds increase calcium and cause relaxation of airway muscle in a unique way," says Dr. Deshpande. "It appears that these taste receptors are wired to a special pool of calcium that is right at the edge of these cells," he says.

"The work of this team exemplifies what it takes to make real improvements in treating certain diseases," says E. Albert Reece, M.D., Ph.D., M.B.A., vice president for medical affairs at the University of Maryland and

dean of the University of Maryland School of Medicine. "These researchers were willing to take chances and ask questions about an unlikely concept. Why are taste receptors in the lungs? What do they do? Can we take advantage of them to devise a new therapy? In the end, their discoveries are in the best tradition of scientific research."

Asthma and COPD together affect 300 million people worldwide. According to the American Lung Association, asthma affects nearly 23 million Americans, including seven million children, and COPD is the fourth leading cause of death in the United States. The incidence of both diseases is increasing. At least half of all asthma patients have inadequate control of the disease using drugs currently available.

Two investigators from Johns Hopkins University, Steven S. An and James S. K. Sham, also contributed to some of the experiments.

This research was supported by grants from the National Heart, Lung, and Blood Institute of the National Institutes of Health.

Deshpande DA, Wang WCH, McIlmoyle EL, Robinett KS, Schillinger RM, An SS, Sham JSK, Liggett SB. "Bitter taste receptors on airway smooth muscle bronchodilate by localized calcium signaling and reverse obstruction." Nature Medicine. Published online October 24, 2010.

Kryptonite superglue improving the quality of life in heart patients recovering from surgery

Montreal - New research shows that a surgical procedure using a cutting-edge super glue pioneered a year ago by Calgary researchers can improve the recovery of heart patients recovering from open-chest surgery, Dr. Paul Fedak today told the Canadian Cardiovascular Congress 2010, co-hosted by the Heart and Stroke Foundation and the Canadian Cardiovascular Society.

The glue, called Kryptonite™, is being used to enhance the closure of the breastbone after surgery. "It has properties like natural bone and allows for new bone growth" says Dr. Fedak, a cardiac surgeon at Foothills Hospital Medical Centre. Up to this point, the breastbone has been closed only with steel wire that stays in the chest.

"One of the most common complaints among patients is sternal pain following heart surgery," he says "With this alternative procedure, significant healing occurs in hours rather than in weeks." By accelerating and improving bone stability, it allows patients to breathe deeply and painlessly without powerful painkillers, meaning fewer side effects. Enhanced bone stability results in fewer complications such as wound infections and bone separation.

Importantly, there were no associated side effects or complications after one year of follow-up.

With the new procedure, pain is cut down because the Kryptonite™ bonds so quickly and effectively to the breastbone. The breastbone becomes solid within hours, shortening the current recovery time of eight weeks by 50 per cent. "People get back to their regular activities much faster."

This much-anticipated release of the official study results prove that the Kryptonite™ adhesive is capable of enhancing the stability of the breastbone closure resulting in early benefits on post-operative recovery. Researchers found that benefits of the Kryptonite™ adhesive include:

* Increased mechanical strength of the breastbone closure by five to 10 times that of wires alone.
* Patients showing significantly less pain, a reduced need for painkillers, and improved breathing for weeks after the surgery.
* Accelerated post-operative recovery time by weeks.
* Patients have improved physical function and an improved health-related quality of life.

First reported on a year ago, when the procedure had been pilot tested on 20 patients, it has now been used on over 500 patients in hospitals across Canada and the United States. Based on the promising findings from the Calgary studies, a larger clinical trial has been established that will include 15 Canadian hospitals and three from the United States.

"It is estimated that 29,000 surgeries requiring sternotomy are performed annually in Canada. World-wide, 1.4 million are performed each year," says Heart and Stroke Foundation spokesperson Dr. Beth Abramson. "This procedure will potentially revolutionize surgical recovery around the world. It increases function, considerably improves quality of life, and ultimately saves the medical system money."

Improving a patient's function is important because walking in addition to medication is needed to keep patients healthy after surgery.

Physicians can be taught very easily to use Kryptonite™. The procedure does not require special equipment and takes only five minutes to perform.

Dr. Fedak has trained surgeons across Canada who now routinely perform the procedure on their patients.

Unexpected findings of lead exposure may lead to treating blindness

Research team at UH sees novel changes in retinal anatomy, results published in high-impact journal

HOUSTON, Oct. 25, 2010 – Some unexpected effects of lead exposure that may one day help prevent and reverse blindness have been uncovered by a University of Houston (UH) professor and his team.

Donald A. Fox, a professor of vision sciences in UH's College of Optometry (UHCO), described his team's findings in a paper titled "Low-Level Gestational Lead Exposure Increases Retinal Progenitor Cell Proliferation and Rod Photoreceptor and Bipolar Cell Neurogenesis in Mice," published recently online in Environmental Health Perspectives and soon to be published in the print edition of the prestigious peer-reviewed journal.

The study suggests that lead, or a new drug that acts like lead, could transform human embryonic retinal stem cells into neurons that would be transplanted into patients to treat retinal degenerations.

"We saw a novel change in the cellular composition of the retina in mice exposed to low levels of lead during gestation. The retina contained more cells in the rod vision pathway than normal or than we expected," said Fox, who also is a professor of biology and biochemistry, pharmacology and health and human performance. "The rod photoreceptors and bipolar cells in this pathway are responsible for contrast and light/dark detection. These new findings directly relate to the supernormal retinal electrophysiological changes seen in children, monkeys and rats with low-level gestational lead exposure."

Fox said these effects occur at blood lead levels at or below 10 micrograms per deciliter, the current low-level of concern by the Centers for Disease Control and Prevention. Because the effects occur below the "safe level," Fox says it raises more questions about what should be considered the threshold level for an adverse effect of lead on the brain and retina.

Fox has studied lead toxicity for 35 years, specifically as it relates to its effects on the brain and retina of children. His interest in gestational lead exposure started in 1999, when he and colleague Stephen Rothenberg studied a group of children in Mexico City whose mothers had lead exposure throughout their pregnancies. The study was funded to measure the adverse effects of lead poisoning on the nervous system of children born in Mexico City – a city that has elevated levels of lead in the air due to the use of leaded gasoline, as well as continued use of lead-containing pottery and glassware for food preparation. The study was funded by the U.S. Environmental Protection Agency and the Mexican government and was published in 2002 in the journal Investigative Ophthalmology and Visual Sciences.

Supported by a $1.7 million National Institutes of Health (NIH) grant, Fox and his group set out to find possible reasons for this supernormal retinal response in children. The researchers employed rat and mice models that covered the three levels of lead found in the blood of the Mexico City mothers – some below, some right at and some higher than the CDC "safe level." The researchers exposed rodents to lead throughout pregnancy and the first 10 days of life, which is a time period equivalent to human gestation.

Fox said that the early-born retinal progenitor cells give rise to four neuron types, which were not affected by lead exposure. The later-born retinal progenitor cells, he said, give rise to two types of neurons and a glial cell. Surprisingly, only the late-born neurons increased in number. The glial cells, which nurture neurons and sometimes protect them from disease, were not changed at all. The rats and mice both had "bigger, fatter retinas," according to Fox. Interestingly, the lower and moderate doses of lead produced a larger increase in cell number than the high lead dose.

"This is really a novel and highly unexpected result, because lead exposure after birth or during adulthood kills retinal and brain cells, but our study showed that low-level lead exposure during gestation caused cells to proliferate, increased neurons and did not affect glia," Fox said. "So, gestational exposure produces an exact opposite to what was previously shown by our lab and others. It also shows that the timing of chemical exposure during development is just as important as the amount of exposure."

This brought the researchers to a crossroads. On the one hand, the retina is not built to have all these extra cells and, according to unpublished data from Fox's mouse studies, the retinas will start to degenerate as the mice age. This suggests that the retinas of the children from the original Mexico City study should be examined as they might start to degenerate when they are 40 years of age.

"This work has long-term implications in retinal degeneration and diseases where photoreceptors die. If we can figure out how low-level lead increases the number of retinal progenitor cells and selectively produces photoreceptors and bipolar cells, then perhaps a drug can be created to help those with degenerative retinal diseases that eventually cause blindness," Fox said. "Researchers may be able to use lead as tool in transforming embryonic retinal stem cells into rods and bipolar cells that could be transplanted into diseased retinas, ultimately saving sight and reversing blindness."

Fox said that more research is needed before such a potential drug could be developed to mimic the effects of lead. Ideally, this drug would induce human embryonic retinal stem cells to form rods and bipolars that could be transplanted into patients to treat early stages of retinal degeneration.

In addition to Fox and research assistant professor Dr. Weimin Xiao in the UHCO, the research team consisted of a number of Fox's current and former students, including Anand Giddabasappa, a former Ph.D. student and now UHCO alumnus; Jerry E. Johnson, a UH alumnus from the department of biology and biochemistry, former post-doctoral fellow in Fox's lab and now an assistant professor at UH-Downtown; and current Ph.D. students W. Ryan Hamilton, Shawntay Chaney and Shradha Mukherjee.

Supplementing the NIH research project grant, this study also was funded by National Eye Institute training and core grants and a National Institute for Occupational Safety and Health educational resource grant.

A copy of the article can be found at http://ehp03.niehs.nih.gov/article/info:doi/10.1289/ehp.1002524.

Marathons damage the hearts of less fit runners for up to 3 months

V02 max test can help determine who is at risk

Montreal - Is running a marathon good for you or can it damage the heart?

A team of researchers and runners from the Heart and Stroke Foundation have come up with a practical way of answering the question. They used data from magnetic resonance imaging (MRI) to find out what is really going on in the marathoner's heart as the kilometers pile up.

"Marathon runners can be a lot less fit than they think," Dr. Eric Larose today told the Canadian Cardiovascular Congress 2010, co-hosted by the Heart and Stroke Foundation and the Canadian Cardiovascular Society.

Lack of real aerobic fitness may directly impact the ways the heart organizes itself to survive the stress of marathon running, says Dr. Larose.

His research found that the magnitude of abnormal heart segments was more widespread and significant in a group of less fit runners. During the marathon, they had signs the heart might be at greater risk of damage than that of runners who had better training or at least had better exercise capacity.

"Without proper training, marathon running can damage your heart. Fortunately the exercise-induced injury is reversible over time," said Dr. Larose. "But it could take up to three months to completely recover."

They studied the effects using MRI measurements, which propel research beyond the traditional stethoscope as a means of estimating and measuring heart function.

The left ventricle of the heart is divided into 17 segments that make up the heart as a whole. When a segment is injured – or stressed out – during the marathon, its neighbours on either side can take over to perform the function of the damaged area. This makes the heart as a whole appear stronger and fitter than is really the case when considering each individual segment.

It also makes it practically impossible for physicians to arrive at an accurate assessment of the heart health of the marathoner when only considering the whole heart. "The heart isn't simply playing tricks - this may be an important adaptive survival mechanism, like the way the brain can switch function after a stroke," says Dr. Larose. "Unfortunately, as a result, the data produced by traditional means may be inconsistent and misleading.

"This means that, short of performing MRI in everyone, we are left with only one practical test that can accurately tell runners their level of cardiac fitness under stress, " says Dr. Larose, who is professor of medicine at Laval University and a cardiologist and clinical researcher at Institut universitaire de cardiologie et de pneumologie de Québec (IUCPQ) in Québec City. That test is V02 max – the ultimate measure of aerobic endurance.

V02 max directly measures body oxygen consumption and it is the best test to provide an accurate measure of a safe maximum heart rate (number of beats per minute) for runners. In V02 testing, treadmills or stationary bicycles may be used to establish cardiac fitness.

Dr. Larose took healthy amateur runners and performed a full evaluation on them six to eight weeks before, and then immediately after, they ran a marathon. They underwent exercise tests, blood analysis, and magnetic resonance imaging.

"What we did notice in this study is a runner with less preparation before the marathon had lower V02 max, so they had lower exercise capacity. Compared to those runners with better training, they became more dehydrated and their hearts showed greater signs of injury. The less well trained runners also experienced greater loss of function associated with lower blood flow and greater irritation of heart segments."

Heart and Stroke Foundation spokesperson Dr. Beth Abramson says that with the increasing popularity of marathon running, especially among boomers who are putting a marathon on their 'to do' lists, runners need to train properly, stay hydrated, and most importantly, speak to their physicians about what is right for them.

"You can do it – physical activity is very important for your heart health. Just be smart about it: train and get medical advice," says Dr. Abramson. "Not everyone will need extensive testing before training to run a marathon but speaking to your doctor about your cardiac risk is important."

Dr. Larose says there is no substitute for a visit to a healthcare professional and, when appropriate, to get the V02 test to measure risks to your heart.

MicroRNAs dictate the Epstein-Barr virus' elaborate waiting game, cancer formation

While most commonly associated with mononucleosis, Epstein-Barr virus (EBV) has been linked to many diseases that affect people long after the initial infection takes place, including some forms of cancer. In the current issue of the Journal of Biological Chemistry, scientists at The Wistar Institute describe how viral microRNA – small segments of RNA that suppress the effects of gene activity – allows EBV to hide within cells and evade the immune system. The scientists believe their findings may one day enable physicians to flush EBV out of hiding, allowing a healthy immune system to rid the body of the virus.

According to the scientists, EBV uses microRNA encoded among its own genes to create an elaborate timing mechanism that allows it to quietly persist until an opportune moment to reproduce en masse. In particular, a viral microRNA, called BART6, keeps EBV in a latent, or quiet, state by preventing the host cell from creating its own microRNA as part of normal gene regulation.

"Epstein-Barr infection is marked by a period of active infection and replication – the lytic stage – where it causes acute disease, but it can also remain latent, and later emerge as an effective cancer-causing agent," said Kazuko Nishikura, Ph.D., a professor in Wistar's Gene Expression and Regulation program and senior author of the study. "It is a strategy that allows EBV to survive our initial immune response and await conditions, such as weakened immunity, to reemerge."

According to the Centers for Disease Control and Prevention, up to 95 percent of Americans are infected with EBV. While only a small portion of these infections ever lead to cancer, EBV has been associated with diseases that include cancers such as Burkitt lymphoma, Hodgkin's lymphoma, and a form of sinus and throat cancer called nasopharyngeal carcinoma.

"Our findings suggest that EBV and humans have been engaged in a complex microRNA arms race, where EBV evolved microRNA that specifically exploit the human host cell's own microRNA machinery," Nishikura said.

These findings add to the growing body of evidence that suggest microRNA activity has a real and potent effect on health, Nishikura says. MicroRNAs are among a host of objects encoded within our DNA that help regulate how genes are read – or "expressed" – by our cells in the form of proteins.

MicroRNAs suppress gene activity by knocking out messenger RNAs, molecules that serve to convey genetic instructions to our cell's protein-making machinery. In effect, microRNA suppression of messenger RNA is akin to the diner manager who fires a waiter in the middle of a shift – your order may have been placed, but the kitchen will never make that grilled sandwich you want.

Nishikura and her colleagues found that BART6 directly prevents the production of the human protein, called DICER, responsible for creating microRNAs by "dicing" up stretches of RNA encoded in our DNA. In silencing DICER, BART6 also silences an EBV gene, called EBNA2, which creates a protein that can "transform" human cells into a cancerous state in the process of forcing the cell to create new copies of the Epstein-Barr virus.

The result is a complex feedback mechanism that can be tipped into inciting cancer if the human immune system is weakened by age or HIV/AIDS infection, for example. However, EBV becomes vulnerable to the immune system when infected cells begin producing viral proteins in significant numbers, so BART6 helps EBV maintain a balance between total silence and a degree of activity that would attract the attention of the immune system. BART6 serves as a sort of timer, since EBV relies on its host cell's DICER protein to create viral mircoRNA, including BART6. As BART6 levels drop, DICER and EBNA2 become active, which ultimately leads to BART6 becoming active again. Cancer may occur, for example, when the upswing in EBNA2 coincides with a drop in immune system activity.

"Epstein-Barr virus uses microRNA to achieve a balance between suppressing genes and promoting genes," Nishikura said. "This system has been so precisely tuned through evolution that BART6 only interacts with human DICER messenger RNA, which may explain why EBV doesn't infect other animals."

However, there are two sides to this RNA arms race, since humans evolved a strategy to counteract BART6 through "RNA editing," where members of the ADAR (adenosine deaminase acting on RNA) family of genes actively alter microRNA precursors. Nishikura and her colleagues also found that ADAR1 prevents the cell from making fully matured forms of BART6 and stops the formation of a critical RNA-protein machine consisting of BART6, DICER, and other proteins collectively known as RISC (RNA induced silencing complex). BART6 only works after being integrated into RISC, so RNA editing appears to be an evolutionary adaptation to BART6 activities, Nishikura says.

When the researchers removed BART6 from the feedback cycle, the cells in culture began to produce a number of viral genes, including EBNA2. In humans, Nishikura says, this would expose cells to the immune system, enabling the body to clear itself of EBV in healthy patients. "Although it may be some time before we can manipulate microRNA as a part of patient care, these findings offer evidence that we may one day use some of the same tools our cells use to regulate gene activity," Nishikura said.

This study was funded by grants from the National Institutes of Health, the Ellison Medical Foundation, and the Commonwealth Universal Research Enhancement Program, Pennsylvania Department of Health.

The first author of this study is Hisashi Iizasa, Ph.D., a former postdoctoral researcher in the Nishikura laboratory and now an assistant professor at Hokkaido University in Sapporo, Japan. Co-authors include Dai Iwakiri, Kenzo Takada, also of Hokkaido University; Manolis Maragkakis and Artemis Hatzigeorgiou of the Alexander Fleming Biomedical Sciences Research Center in Athens, Greece; and Molly Megraw, Ph.D., of the University of Pennsylvania. Wistar co-authors include Bjorn-Erik Wulff, Nageswara R. Alla, of the Nishikura laboratory; Professor Louise Showe, Ph.D.; and Professor Paul Lieberman, Ph.D.

Blood group 'affects fertility'

A woman's ability to conceive in early middle age may be influenced by her blood type, according to research.

The US study of 560 women undergoing fertility treatment found that those with type "O" blood had chemical signs linked to low egg numbers. There is no clear explanation for the results, presented to the American Society of Reproductive Medicine conference in Denver. Approximately 44% of the UK population has type "O" blood.

The researchers, from Albert Einstein College of Medicine in New York, and Yale University, looked at the levels of a chemical called follicle-stimulating hormone (FSH) in the women, who had an average age of 35.

Blood clue

A woman has a fixed number of eggs, her "ovarian reserve", which are released gradually over her fertile life.

High levels of FSH are thought to be an indicator that this reserve is diminishing more quickly - which can reduce chances of conception once a woman reaches her 30s and 40s.

Analysis of blood samples revealed that the women with type "O" blood were more likely to have higher FSH readings. Those with type "A" blood - the other major blood group in the UK - had lower FSH levels.

Dr Edward Nejat, who led the study, said: "A woman's age remains the most important factor in determining her success of conceiving. "The baseline FSH gives us an idea of the quality and quantity of a woman's eggs."

Whether or not this will make any difference to women in the general population is not clear - all the women in the trial were already seeking fertility treatment.

Tony Rutherford, chairman of the British Fertility Society, said the research was "interesting". He said that further larger-scale research would be needed to both confirm the result, and see if an effects could be spotted in women with no diagnosed fertility problems trying to conceive. "This is the first time that I'm aware of that the researchers have shown a link between blood group and potential for fertility.

"We really need to look at it with other, more up to date tests of ovarian reserve - and to look at a prospective group of women to see if blood group affects your chance of getting pregnant."

20 die in air disaster after smuggled crocodile escapes on a plane

By John Platt Monday, October 25, 2010 15

Wildlife smugglers will do just about anything for a quick buck, including sneaking a live predator onto an airplane with no regard for the risk to the animal or fellow passengers. This illegal activity reached a devastating and absurdist extreme recently when a man reportedly smuggled a live crocodile onto a plane departing from the Democratic Republic of the Congo (DRC). The crocodile got loose, the crew and passengers panicked, and the plane crashed, killing 20 people. Oddly enough, the crocodile survived the crash, only to be hacked to death by machete-wielding locals on the ground.

Only one passenger lived to bear witness to the events. The unnamed survivor was interviewed by the France-based African news magazine Jeune Afrique, where he said the crocodile was smuggled onto the plane in a sports bag. When the croc escaped, the passengers reportedly rushed toward the cockpit, throwing the plane off balance, the man claimed.

The magazine also reported the crocodile had been intended for resale, although how the crash survivor knew this remains unclear. The incident occurred August 25, but initial reports said the plane had simply run out of fuel.

ITN News has video of the crash site on YouTube.

None of the reports identify the species of crocodile, but the DRC is home to the rare subspecies known as the Congo, or Osborn's, dwarf crocodile (Osteolaemus tetraspis osborni).

Contraceptive gel shows promise as alternative to Pill

A birth control gel that is applied to the skin could offer woman an alternative to the Pill, say experts presenting latest trial data.

Used once daily, it delivers hormones to prevent a pregnancy in the same way as oral contraceptives do.

Early studies show the gel is effective and well tolerated, with none of the typical side effects associated with the Pill, like weight gain and acne. The Nestorone gel is being developed with drug firm Antares Pharma.

Researchers told the American Society for Reproductive Medicine how they hope to bring the product to market if clinical trial results continue to be positive. The gel can be applied to the abdomen, thighs, arms or shoulders and is quickly absorbed, with no residue. Experts say it is also suitable for women who are breastfeeding, unlike the combined Pill which can interfere with milk supply.

Dr Ruth Merkatz from the not-for-profit Population Council research centre in New York led the latest study, which involved 18 women in their 20s to 30s.

The research found the optimum dose of the gel was 3mg a day. Over the course of seven months, none of the women using the treatment fell pregnant. Hormone studies showed the gel suppressed the production of eggs by the ovary.

Dr Merkatz said: "From this small study we found it was effective. "It's in early stage development but if we move on, we will obviously test it in many, many more women."

The researchers say it could offer an alternative to the Pill, which is used by over 3m women in the UK alone.

Natika Halil, director of information at the Family Planning Association, said: "Any contraceptive system that increases the choice of methods available to women and helps to prevent unwanted pregnancies is welcome.

"Our research shows that there are approximately two million women using a contraceptive method that they are unhappy with, so they will benefit from improved choices and options.

"This product won't suit everyone and will only be for women comfortable (with) putting it on their skin and having their contraceptive cover that way."

Simon Blake, chief executive of sexual health charity Brook, said: "Obviously this is still in the very early stages of development but anything that can help young women has got to be a good thing.

"Clearly what young women need is more choice."

http://www.bbc.co.uk/news/health-11619044

New polio vaccine more effective in reducing disease

By Ania Lichtarowicz Health reporter, BBC News

Child receiving polio vaccine The new oral polio vaccine could help to finally eradicate the virus

A new vaccine against the polio virus has helped reduce the number of cases by more than 90%. Research published online in the journal The Lancet, shows that the new vaccine is significantly better at protecting children against polio than the current popular vaccine. It has already been used in Afghanistan, India and Nigeria. The scientists behind the work believe this new vaccine could help to finally eradicate the disease.

Disease elimination

Mass vaccination campaigns have led to the number of polio endemic countries falling from 125 in 1988 to just four in 2005. This meant an actual drop in cases from 350,000 to just 1,606 in 2009.

Polio is caused by one of 3 versions of the polivirus: type1, type 2 or type 3. Until recently, vaccines targetting either all three forms of the virus or just one of them were used to immunise children.

The last case of type 2 polio was recorded in India in 1999, so it's the other two types that need to be targeted to finally eliminate the disease.

The authors of the study carried out a trial in India comparing the commonly used old vaccines to the new one, which is taken orally. In total, 830 newborn babies received either the new vaccine or one of the old vaccines in two doses - one at birth and one 30 days later.

Blood samples were taken before vaccination and after the first and second doses to measure seroconversion - the rise in antibodies produced by the immune system against polio. It appears that the new vaccine is about 30% more effective in protecting against polio than the most commonly used vaccine to date.

Finish Line

The new vaccine has already been used in immunisation campaigns in Afghanistan, India and Nigeria. In India the number of cases this time last year was 464. Over the same period this year there have only been only 39 cases. Nigeria has seen an even greater difference, with cases falling by 95%.

The new vaccine and improved immunisation programmes appear to be responsible for this significant decrease, according to the World Health Organization (WHO).

Dr Roland Sutter, from the WHO and the lead author of the study, told BBC News: "This (new) vaccine could get us over the top and get us to the finish line for eradication.

"The dramatic drop in the number of polio cases in India and Nigeria is attributable to the new vaccine and better coverage during immunization campaigns."

The private sector manufacturers played a key role in its development, says Dr Bruce Aylward, the Director of WHO's Global Polio Eradication Initiative. "They've held the price to the same price of what we are paying for the older polio vaccine," he says. The new vaccine can be administered in the same way as the previous one. "That's why there is so much promise with this product," says Dr Aylward.

Commenting on the research, Nigel Crawford and Jim Buttery from the Murdoch Children's Research Institute (SAEFVIC) in Melbourne, Australia, said that the new vaccine had shown great promise.

However they cautioned that the global financial crisis had resulted in a massive funding gap for immunisation programmes worldwide, including polio.

http://www.bbc.co.uk/news/health-11621842

6 new isotopes of the superheavy elements discovered

Moving closer to understanding the island of stability

Berkeley, CA—A team of scientists at the U.S. Department of Energy's Lawrence Berkeley National Laboratory has detected six isotopes, never seen before, of the superheavy elements 104 through 114. Starting with the creation of a new isotope of the yet-to-be-named element 114, the researchers observed successive emissions of alpha particles that yielded new isotopes of copernicium (element 112), darmstadtium (element 110), hassium (element 108), seaborgium (element 106), and rutherfordium (element 104). Rutherfordium ended the chain when it decayed by spontaneous fission.

Information gained from the new isotopes will contribute to a better understanding of the theory of nuclear shell structure, which underlies predictions of an "Island of Stability," a group of long-lasting isotopes thought to exist amidst a sea of much shorter-lived, intrinsically unstable isotopes of the superheavy elements.

The group that found the new isotopes is led by Heino Nitsche, head of the Heavy Element Nuclear and Radiochemistry Group in Berkeley Lab's Nuclear Science Division (NSD) and professor of chemistry at the University of California at Berkeley. Ken Gregorich, a senior staff scientist in NSD, is responsible for the group's day-to-day research operation at the 88-inch Cyclotron and the Berkeley Gas-filled Separator, the instrument used to isolate and identify the new isotopes. Paul Ellison of NSD, a graduate student in the UC Berkeley Department of Chemistry, formally proposed and managed the experiment and was first author of the paper reporting the results in the 29 October 2010 issue of *Physical Review Letters*, now available online to subscribers at http://prl.aps.org/abstract/PRL/v105/i18/e182701.

"We were encouraged to try creating new superheavy isotopes by accelerating calcium 48 projectiles with Berkeley Lab's 88-Inch Cyclotron and bombarding plutonium 242 targets inside the Berkeley Gas-filled Separator here," Nitsche says. "This was much the same set-up we used a year ago to confirm the existence of element 114."

The 20-member team included scientists from Berkeley Lab, UC Berkeley, Lawrence Livermore National Laboratory, Germany's GSI Helmholtz Center for Heavy Ion Research, Oregon State University, and Norway's Institute for Energy Technology. Many of its members were also on the team that first confirmed element 114 in September of 2009. Ten years earlier scientists at the Joint Institute for Nuclear Research in Dubna, Russia, had isolated element 114 but it had not been confirmed until the Berkeley work. (Elements heavier than 114 have been seen but none have been independently confirmed.)

The nuclear shell game

Nuclear stability is thought to be based in part on shell structure—a model in which protons and neutrons are arranged in increasing energy levels in the atomic nucleus. A nucleus whose outermost shell of either protons or neutrons is filled is said to be "magic" and therefore stable. The possibility of finding "magic" or "doubly magic" isotopes of superheavy elements (with both proton and neutron outer shells completely filled) led to predictions of a region of enhanced stability in the 1960s.

The challenge is to create such isotopes by bombarding target nuclei rich in protons and neutrons with a beam of projectiles having the right number of protons, and also rich in neutrons, to yield a compound nucleus with the desired properties. The targets used by the Berkeley researchers were small amounts of plutonium 242 (^{242}Pu) mounted on the periphery of a wheel less than 10 centimeters in diameter, which was rotated to disperse the heat of the beam.

Gregorich notes that calcium 48 (^{48}Ca), which has a doubly magic shell structure (20 protons and 28 neutrons), "is extremely rich in neutrons and can combine with plutonium"—which has 94 protons—"at relatively low energies to make compound nuclei. It's an excellent projectile for producing compound nuclei of element 114."

Ellison says, "There's only a very low probability that the two isotopes will interact to form a compound nucleus. To make it happen, we need very intense beams of calcium on the target, and then we need a detector

that can sift through the many unwanted reaction products to find and identify the nuclei we want by their unique decay patterns." The 88-Inch Cyclotron's intense ion beams and the Berkeley Gas-filled Separator, designed specifically to sweep away unwanted background and identify desired nuclear products, are especially suited to this task.

Element 114 itself was long thought to lie in the Island of Stability. Traditional models predicted that if an isotope of 114 having 184 neutrons (^{298}114) could be made, it would be doubly magic, with both its proton and neutron shells filled, and would be expected to have an extended lifetime. The isotopes of 114 made so far have many fewer neutrons, and their half-lives are measured in seconds or fractions of a second. Moreover, modern models predict the proton magic number to be 120 or 126 protons. Therefore, where ^{298}114 would actually fall inside the region of increased stability is now in question.

"Making ^{298}114 probably won't be possible until we build heavy ion accelerators capable of accelerating beams of rare projectile isotopes more intense than any we are likely to achieve in the near future," says Nitsche. "But in the meantime we can learn much about the nuclear shell model by comparing its theoretical predictions to real observations of the isotopes we *can* make."

The team that confirmed element 114 observed nuclei of two isotopes, ^{286}114 and ^{287}114, which decayed in a tenth of a second and half a second respectively. In a subsequent collaboration with researchers at the GSI Helmholtz Center for Heavy Ion Research, two more isotopes, ^{288}114 and ^{289}114, were made; these decayed in approximately two-thirds of a second and two seconds respectively.

While these times aren't long, they're long enough for spontaneous fission to terminate the series of alpha decays. Alpha particles have two protons and two neutrons – essentially they are helium nuclei—and many heavy nuclei commonly decay by emitting alpha particles to form atoms just two protons lighter on the chart of the nuclides. By contrast, spontaneous fission yields much lighter fragments.

A new strategy

So this year the Berkeley group decided to make new isotopes using a unique strategy: instead of trying to add more neutrons to 114, they would look for isotopes with fewer neutrons. Their shorter half-lives should make it possible for new isotopes to be formed by alpha emission before spontaneous fission interrupted the process.

"This was a very deliberate strategy," says Ellison, "because we hoped to track the isotopes that resulted from subsequent alpha decays farther down into the main body of the chart of nuclides, where the relationships among isotope number, shell structure, and stability are better understood. Through this connection, and by observing the energy of the alpha decays, we could hope to learn something about the accuracy of predictions of the shell structure of the heaviest elements."

The sum of protons and neutrons of ^{48}Ca and ^{242}Pu is 114 protons and 176 neutrons. To make the desired "neutron poor" ^{285}114 nucleus, one having only 171 neutrons, first required a beam of ^{48}Ca projectiles whose energy was carefully adjusted to excite the resulting compound nucleus enough for five neutrons to "evaporate."

"The process of identifying what you've made comes down to tracking the time between decays and decay energies," says Ellison. As a check against possible mistakes, the data from the experiment were independently analyzed using separate programs devised by Ellison, Gregorich, and team member Jacklyn Gates of NSD.

In this way, after more than three weeks of running the beam, the researchers observed one chain of decays from the desired neutron-light 114 nucleus. The first two new isotopes, ^{285}114 itself, and copernicium 281 produced by its alpha decay, lived less than a fifth of a second before emitting alpha particles. The third new isotope, darmstadtium 277, lived a mere eight-thousandths of second. Hassium 273 lasted a third of a second. Seaborgium 269 made it to three minutes and five seconds but managed to emit an alpha particle. Finally, after another two and a half minutes, rutherfordium 265 decayed by spontaneous fission.

Ellison says, "In the grand scheme, the theoretical predictions were pretty good" when the actual measurements were compared to the decay properties predicted by modern nuclear models. "But there were small-scale interesting differences." In particular, the heaviest new isotopes, those of 114 and copernicium, showed smaller energies associated with the alpha decay than theory predicts. These discrepancies can be used to refine the theoretical models used to predict the stability of the superheavy elements.

As Gregorich puts it, "our new isotopes are on the western shore of the Island of Stability"—the shore that's less stable, not more. Yet the discovery of six new isotopes, reaching in an unbroken chain of decays from element 114 down to rutherfordium, is a major step toward better understanding the theory underlying exploration of the region of enhanced stability that is thought to lie in the vicinity of element 114—and possibly beyond.

New superheavy element isotopes: ^{242}Pu (^{48}Ca,5n)^{285}114, by Paul Ellison, Kenneth Gregorich, Jill Berryman, Darren Bleuel, Roderick Clark, Irena Dragojević, Jan Dvorak, Paul Fallon, Carolina Fineman-Sotomayor, Jacklyn Gates, Oliver Gothe, I-Yang Lee, Walter Loveland, Joseph McLaughlin, Stefanos Paschalis, Marina-Kalliopi Petri, Ji Qian, Liv Stavsetra, Mathis

Wiedeking, and Heino Nitsche, appears in the 29 October 2010 issue of Physical Review Letters and is now available online to subscribers at http://prl.aps.org/abstract/PRL/v105/i18/e182701. This research was supported by the DOE Office of Science and the National Nuclear Security Administration.
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http://www.eurekalert.org/pub_releases/2010-10/dbnl-sni102610.php

Patients who survive sepsis are more than 3 times as likely to have cognitive problems

First large-scale study shows that most older patients hospitalized with severe sepsis face years of cognitive, physical decline, according to U-M research in JAMA

Ann Arbor, Mich. — Older adults who survive severe sepsis are at higher risk for long-term cognitive impairment and physical limitations than those hospitalized for other reasons, according to researchers from the University of Michigan Health System.

Research to be published Oct. 27 in the Journal of the American Medical Association showed that 60 percent of hospitalizations for severe sepsis were associated with worsened cognitive and physical function among surviving older adults. The odds of acquiring moderate to severe cognitive impairment were 3.3 times higher following an episode of sepsis than for other hospitalizations.

Severe sepsis also was associated with greater risk for the development of new functional limitations following hospitalization, says lead author, Theodore (Jack) Iwashyna, M.D., Ph.D., assistant professor of internal medicine at U-M. Among patients who had no limitations before sepsis, more than 40% developed trouble with walking. Nearly 1 in 5 developed new problems with shopping or preparing a meal. Patients often developed new problems with such basic things as bathing and toileting themselves.

"We used to think of sepsis as just a medical emergency, an infection that you get sick with and then recover," said Iwashyna, "But we discovered a significant number of people face years of problems afterwards.

"Those problems are bigger and more common than we expected. Most older Americans suffer real brain and body problems. We need new treatments, not just for the sepsis infection, but to prevent these new disabilities afterwards."

Sepsis is an overwhelming infection that can result in failure of multiple organ systems. The initial infections are often common problems, such as pneumonia or a urinary tract infection. About 40 percent of those with severe sepsis die from the infection. Anyone can get sepsis, but older people and those with weakened immune systems are most vulnerable. Sepsis is probably the most common cause of critical illness in the United States.

The best data available are from the 1990s, when it was estimated that 750,000 people each year were diagnosed with sepsis. Researchers believe that number has doubled each decade.

"These new data show a majority of older patients suffer with real life-changing burdens after beating sepsis. This is an underrecognized public health problem with major implications for patients, families and the health care system," Iwashyna says. "We need to make sure families have the resources they need to care for survivors of sepsis when they go home. It's not enough just to get them through the acute episode. We need to start preparing them for the years of problems they may have afterwards."

"This research underscores the need for physicians who care for older adults to focus early on preventing infections that can lead to sepsis," says study co-author Kenneth M. Langa, M.D., Ph.D., a core investigator for the Ann Arbor Veterans Administration Health Services Research and Development Service's Center of Excellence and professor of internal medicine at U-M.

Older patients need to get their flu and pneumonia vaccines in order to decrease their risk for infections, and physicians need to be aware of the long-term risk for cognitive and physical disabilities that many patients may face, Langa said. "In contrast to Alzheimer's disease and other forms of dementia, the cognitive impairment associated with sepsis is likely at least partially preventable through better acute care of the sepsis episode and better rehabilitation efforts afterwards," Langa says.

"We need to start working early – from the beginning of the hospitalization – to make sure patients do not develop new disability. There are innovative new ways to care for people that might help prevent this disability," Iwashyna says.

The research was supported primarily by the National Institute on Aging and the National Heart, Lung and Blood Institute. The researchers used data from the NIA-supported Health and Retirement Study , a long-term study that collects information on the health, economic, and social factors influencing the health and well-being of a nationally representative sample Americans over age 50.

"This research makes clearer how acute medical problems in older adults may have an important lasting impact and contribute to a downward trajectory in both cognitive and physical function," says Richard Suzman, Ph.D., director of the NIA's Division of Behavioral and Social Research, which supports the HRS.

"The unique nature of the rich HRS dataset that links both survey data and Medicare administrative data made this innovative study possible and will also facilitate future studies of the long-term impact of critical illness on older adults and the family members that care for them."

The HRS, now in its 18th year, follows more than 22,000 people over the age of 50, collecting data every two years, from pre-retirement to advanced age.

The NIA leads the federal effort supporting and conducting research on aging and the medical, social and behavioral issues of older people. For more information on research and aging, go to www.nia.nih.gov.

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http://www.eurekalert.org/pub_releases/2010-10/uomh-pws102110.php

The Claim: Lying on Your Left Side Eases Heartburn

By ANAHAD O'CONNOR

THE FACTS For people with chronic heartburn, restful sleep is no easy feat. Fall asleep in the wrong position, and acid slips into the esophagus, a recipe for agita and insomnia.

Doctors recommend sleeping on an incline, which allows gravity to keep the stomach's contents where they belong. But sleeping on your side can also make a difference — so long as you choose the correct side. Several studies have found that sleeping on the right side aggravates heartburn; sleeping on the left tends to calm it.

The reason is not entirely clear. One hypothesis holds that right-side sleeping relaxes the lower esophageal sphincter, between the stomach and the esophagus. Another holds that left-side sleeping keeps the junction between stomach and esophagus above the level of gastric acid.

In a study in The Journal of Clinical Gastroenterology, scientists recruited a group of healthy subjects and fed them high-fat meals on different days to induce heartburn. Immediately after the meals, the subjects spent four hours lying on one side or the other as devices measured their esophageal acidity. Ultimately, the researchers found that "the total amount of reflux time was significantly greater" when the subjects lay on their right side.

"In addition," they wrote, "average overall acid clearance was significantly prolonged with right side down."

In another study, this one in The American Journal of Gastroenterology, scientists fed a group of chronic heartburn patients a high-fat dinner and a bedtime snack, then measured reflux as they slept. The right-side sleepers had greater acid levels and longer "esophageal acid clearance." Other studies have had similar results.

THE BOTTOM LINE Lying on your right side seems to aggravate heartburn.

http://www.nytimes.com/2010/10/26/health/26really.html

Brain Takes Less Than Second to Fall in Love

By Liz Day | Tue Oct 26, 2010 08:47 AM ET

A recent study used functional magnetic resonance imaging to see how love affects the brain. Its calculations of love has attracted plenty of attention.

For example, the time taken to "fall in love" clocks in at about one-fifth of a second, not the six months of romantic dinners and sharing secrets some might expect.

Also, 12 areas of the brain work together during the love process, releasing euphoria-inducing chemicals like dopamine, oxytocin, adrenaline and vasopressin. Love's high is similar to cocaine's rush.

Love influences sophisticated intellectual processes of the brain too. When a person feels in love, their mental representation, metaphors and even body image are also affected.

Researchers from Syracuse University, West Virginia University and the Geneva University Psychiatric Center retrospectively reviewed pertinent neuroimaging literature. They published their findings in a recent issue of the Journal of Sexual Medicine.

Overall, they found, love is really good for you.

Couples who had just fallen in love had significantly higher levels of nerve growth factor, or NGF. NGF is crucial to the survival of sympathetic and sensory neurons. Some believe NGF can reduce neural degeneration. Not a bad side effect.

Just as love is diverse, the part of the brain affected is also different.

Unconditional love, the type often seen between a mother and child, lights up the common and different brain areas, including the middle of the brain.

Not surprisingly, passionate love fires the reward part of the brain, but it also affects the higher-order cognitive function seen in body image.

A follow-up study about the speed of love in the human brain is expected to follow soon.

http://news.discovery.com/human/brain-takes-less-than-second-to-fall-in-love.html

Singapore scientist leads team to discover origin of brain immune cells

Scientist also successfully visualizes, in a living cell culture, how the immune cells colonize the brain; findings could lead to new strategies to treat various brain disorders

A team of international scientists led by Dr Florent Ginhoux of the Singapore Immunology Network (SIgN) of Singapore's Agency of Science, Technology and Research (A*STAR), have made a breakthrough that could lead to a better understanding of many neurodegenerative and inflammatory brain disorders. Their work, published in top scientific journal Science, uncovered the origins of microglia, which are white blood cells specific to the brain, and showed that, in mice, microglia had a completely different origin than other white blood cells. This understanding may lead to the development of new strategies to manipulate microglia for the treatment of various brain disorders.

Microglia have been implicated in many neurodegenerative and inflammatory brain disorders, underscoring the need to study and understand these cells. Dr Ginhoux's team is the first to show that microglia, unlike other white blood cells, are derived from a particular structure in the mouse embryo (the embryonic yolk-sac), implying that microglia may have specific functional properties not shared by other white blood cells.

In addition, Dr Ginhoux is the first to directly visualise how microglia develop in the brain. This will advance basic understanding of the mouse immune system, which is needed to understand how controlling the development of the precursors of microglia may one day be used to treat brain diseases in humans.

Prof Paola Castagnoli, Scientific Director of SIgN, commented, "Neurodegenerative diseases and inflammatory brain disorders are a major cause of suffering in the world. At SIgN, our focus and mission is to study human immunology and in particular, inflammatory reactions in human diseases. Inflammation occurs when the immune system overreacts to "danger" signals that can either be infectious or non-infectious, for instance, caused by cell or tissue damage. We know that the immune system does not work in isolation within the body, and that the interactions between immune and brain cells is occurring all the time. Therefore a better knowledge of the microglial cells' function and origin will open new avenues in the field of neuro-immunology."

Said Dr Ginhoux, "Several key experiments which were crucial to my work could only have been completed in SIgN. In particular, my work involved the use of a type of microscopy to directly visualise, in a living cell culture, how microglia colonise the brain. This is the first time this sort of work has been done, and it couldn't have been possible without the help of Dr. Lai Guan Ng, my colleague here at SIgN."

Dr Ginhoux plans to continue his investigation into how the unique origin of microglia, as compared to other white blood cell populations in the body, could give rise to the properties of microglia that makes them especially suitable for their role in the brain.

Research publication: *The research findings described in the press release can be found in the 21 October, 2010 advance online issue of Science under the title "Fate Mapping Analysis Reveals That Adult Microglia Derive from Primitive Macrophages".*

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http://www.eurekalert.org/pub_releases/2010-10/afst-ssl102710.php

Revising the timeline for deadly pancreatic cancer

Pancreatic tumors are one of the most lethal cancers, with fewer than five percent of patients surviving five years after diagnosis. But a new study that peers deeply into the genetics of pancreatic cancer presents a bit of good news: an opportunity for early diagnosis. In contrast to earlier predictions, many pancreatic tumors are, in fact, slow growing, taking nearly 20 years to become lethal after the first genetic perturbations appear.

"There have been two competing theories explaining why pancreatic cancers are so lethal," says Bert Vogelstein, the Howard Hughes Medical Institute investigator who helped lead the new study. "The first is that pancreatic tumors are aggressive right from the get-go and spread to other organs very quickly. The second theory is that pancreatic tumors are, in fact, not more aggressive than other tumors, but that symptoms appear so late in the process that patients have little chance of surviving. We were surprised and pleased to discover that this second theory is correct, at least for a major fraction of tumors. It means that there is a window of opportunity for early detection of pancreatic cancer."

The new work is published in the October 28, 2010, issue of the journal Nature. Christine Iacobuzio-Donahue, a pathologist at Johns Hopkins University School of Medicine, is the senior author of the paper.

Working with Iacobuzio-Donahue, Vogelstein obtained samples of primary pancreatic tumors from seven autopsied patients, as well as metastatic lesions from their lungs, liver, and other organs. Their team sequenced the DNA of every gene in each metastatic tumor as well as in the primary tumor. These genetic read-outs

provided data to compare the genetic mutations found in each patient's metastatic lesions with the mutations found in the primary tumor.

The investigators found that each metastatic lesion contained, on average across all patients, 61 cancer-related genetic mutations. Further, the majority of these mutations – 64 percent on average – were also present in the primary tumor. The researchers then worked with Martin Nowak, an evolutionary biologist at Harvard, to estimate how long it took these mutations to accumulate. Using a "molecular clock" technique commonly used in evolutionary biology, it is possible to generate a hypothesis about when a mutation occurred. By comparing the genomes of, say, monkeys and man, evolutionary biologists can estimate how long ago the two species diverged.

Similarly, each genetic mutation seen in a cancer cell represents a tick of the molecular clock. Because such mutations accumulate at a steady rate – as observed in cancer cells growing in petri dishes - Vogelstein and his colleagues could estimate how long it took for all of the mutations seen in each metastatic lesion to appear.

The technique showed that it took a surprisingly long time – 11.7 years on average – for a mature pancreatic tumor to form after the appearance of the first cancer-related mutation in a pancreatic cell. Another 6.8 years passed, on average, before the primary tumor sent out a metastatic lesion to another organ. From that point, another 2.7 years went by, on average, before the patient died. In total, more than 20 years elapsed between the appearance of the first mutated pancreatic cell and death.

"This time scale is similar to what we've previously seen in colorectal cancers," says Vogelstein. "These tumors evolve over long periods --decades."

Unlike other cancers, though, pancreatic tumors usually produce no symptoms until they've spread. Jaundice is often the first symptom, but that arrives only after a pancreatic tumor has metastasized to the liver. But Vogelstein says the new data suggest that a blood or stool test might be able to pick up early cancer-causing mutations. His team is already examining the efficacy of such tests for detecting early signs of colorectal cancer.

"For disease control in the future, this finding is paramount," Vogelstein says. "It gives us hope that we will eventually be able to reduce morbidity and mortality from pancreatic cancer through earlier detection."

The research also provided a glimpse into how pancreatic tumors evolve. In the pancreatic tumors of two of the patients, Iacobuzio-Donahue sectioned the tumors into smaller pieces, and then examined the genetics of each section. Surprisingly, she found that each tumor comprised genetically distinct sub-tumors. That is, the tumor continued to accumulate genetic mutations after the tumor first appeared.

"We saw a whole lot of evolution within the primary tumor, producing what looked like a series of generations of tumor clones – fathers, grandfathers, great-grandfathers, you could say," Vogelstein says. "The primary tumor is, in fact, not a single tumor but an accretion of several genetically distinct tumors. Moreover, we could find a subclone within the primary tumor that gave rise to each metastasis," Vogelstein says. "That's fascinating from a basic science perspective and gives us some deep insights into how these tumors evolve."

http://www.eurekalert.org/pub_releases/2010-10/hhmi-rtt102610.php

Glucosamine causes the death of pancreatic cells

Quebec City, October 27, 2010—High doses or prolonged use of glucosamine causes the death of pancreatic cells and could increase the risk of developing diabetes, according to a team of researchers at Université Laval's Faculty of Pharmacy. Details of this discovery were recently published on the website of the Journal of Endocrinology.

In vitro tests conducted by Professor Frédéric Picard and his team revealed that glucosamine exposure causes a significant increase in mortality in insulin-producing pancreatic cells, a phenomenon tied to the development of diabetes. Cell death rate increases with glucosamine dose and exposure time. "In our experiments, we used doses five to ten times higher than that recommended by most manufacturers, or 1,500 mg/day," stressed Professor Picard. "Previous studies showed that a significant proportion of glucosamine users up the dose hoping to increase the effects," he explained.

Picard and his team have shown that glucosamine triggers a mechanism intended to lower very high blood sugar levels. However, this reaction negatively affects SIRT1, a protein critical to cell survival. A high concentration of glucosamine diminishes the level of SIRT1, leading to cell death in the tissues where this protein is abundant, such as the pancreas.

Individuals who use large amounts of glucosamine, those who consume it for long periods, and those with little SIRT1 in their cells are therefore believed to be at greater risk of developing diabetes. In a number of mammal species, SIRT1 level diminishes with age. This phenomenon has not been shown in humans but if it were the case, the elderly—who constitute the target market for glucosamine—would be even more vulnerable.

"The key point of our work is that glucosamine can have effects that are far from harmless and should be used with great caution," concluded Professor Picard.

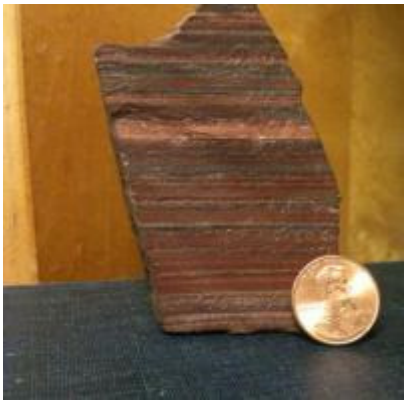
The results obtained by Picard and his team coincide with recent studies that cast serious doubt on the effectiveness of glucosamine in treating joint problems.

This study was co-authored by Mathieu Lafontaine-Lacasse and Geneviève Doré.

http://www.eurekalert.org/pub_releases/2010-10/ul-gct102710.php

New evidence supports 'Snowball Earth' as trigger for early animal evolution

A spike in ancient marine phosphorus concentrations from 750 to 635 million years ago is linked to emergence of complex life, UC Riverside researchers say



RIVERSIDE, Calif. – A team of scientists, led by biogeochemists at the University of California, Riverside, has found new evidence linking "Snowball Earth" glacial events to the rise of early animals.

The controversial Snowball Earth hypothesis posits that the Earth was covered from pole to pole by a thick sheet of ice lasting, on several occasions, for millions of years. These glaciations, the most severe in Earth history, occurred from 750 to 580 million years ago. The researchers argue that the oceans in the aftermath of these events were rich in phosphorus, a nutrient that controls the abundance of life in the oceans.

This image shows a close-up view of a field sample of 2.7 billion-year-old iron formation from Zimbabwe used in the study. The red color is caused by the iron oxide mineral hematite. **Lyons lab, UC Riverside**

The UC Riverside team and colleagues tracked phosphorus concentrations through Earth's history by analyzing the composition of iron-rich chemical precipitates that accumulated on the seafloor and scavenged phosphorus from seawater. Their analyses revealed that there was a pronounced spike in marine phosphorus levels in the mid-Neoproterozoic (from ~750 to ~635 million years ago).

To explain these anomalously high concentrations, the researchers argue that the increase in erosion and chemical weathering on land that accompanied Snowball Earth glacial events led to the high amounts phosphorus in the ocean. The abundance of this nutrient, which is essential for life, in turn, led to a spike in oxygen production via photosynthesis and its accumulation in the atmosphere, facilitating the emergence of complex life on Earth. Study results appear in the Oct. 28 issue of Nature.

"In the geological record, we found a signature for high marine phosphorus concentrations appearing in the immediate aftermath of the Snowball Earth glacial events," said Noah Planavsky, the first author of the research paper and a graduate student in the Department of Earth Sciences. "Phosphorus ultimately limits net primary productivity on geological timescales. Therefore, high marine phosphorus levels would have facilitated a shift to a more oxygen-rich ocean-atmosphere system. This shift could have paved the way for the rise of animals and their ecological diversification. Our work provides a mechanistic link between extensive Neoproterozoic glaciations and early animal evolution."

Planavsky explained the link between marine phosphorus concentrations and the levels of oxygen in the atmosphere. "High phosphorus levels would have increased biological productivity in the ocean and the associated production of oxygen by photosynthesis," he said. "Much of this organic matter is consumed, in turn, as a result of respiration reactions that also consume oxygen. However, the burial of some proportion of the organic matter results in a net increase of oxygen levels in the atmosphere."

Until now, scientists believed that geochemical conditions in the iron-rich ocean would have led to low phosphorus concentrations. The UC Riverside researchers found no evidence of a phosphorus crisis after Snowball Earth glacial events, however, finding instead indications of an abundance of phosphorus.

"There are several known chemical fingerprints for increasing oxygen in the ocean and, by inference, in the atmosphere during the middle part of Neoproterozoic, and the rise of animals is an expected consequence," said Timothy Lyons, a professor of biogeochemistry and the senior investigator in the study. "But our results may be the first to capture the nutrient driver that was behind this major step in the history of life, and that driver was ultimately tied to the extreme climate of the period."

The researchers present data from approximately 700 individual samples of iron-oxide-rich rocks that included new results as well as those obtained from a comprehensive survey of the literature.

Planavsky and Lyons were joined in the study by Christopher Reinhard of UC Riverside; Olivier J. Rouxel of Woods Hole Oceanographic Institute, Mass.; Andrey Bekker of the University of Manitoba, Canada; and Stefan V. Lalonde and Kurt O. Konhauser of the University of Alberta, Canada.

The UC Riverside researchers were supported by grants from the National Science Foundation and the NASA Astrobiology Institute and the NASA Exobiology Program.

http://www.eurekalert.org/pub_releases/2010-10/uoc--nes102510.php

Research rejects green tea for breast cancer prevention

Green tea does not protect against breast cancer. A study of data from approximately 54,000 women, published in BioMed Central's open access journal Breast Cancer Research, found no association between drinking green tea and breast cancer risk.

Motoki Iwasaki, from the National Cancer Center, Tokyo, worked with a team of researchers to carry out the study. He said, "Although in vitro and animal-based studies have suggested that green tea may have beneficial protective effects against breast cancer, results from human studies have been inconclusive. Our large-scale, population-based prospective cohort study is one of the first to include a wide range of tea intakes; women who drank green tea less than 1 cup per week to those who drank 10 or more cups per day. It found no overall association between green tea intake and the risk of breast cancer".

Tea intake was assessed by questionnaire, once at the beginning of the study and then again five years later. Cancer incidence was assessed by notification from major local hospitals in the study area and data linkage with population-based cancer registries. Approximately 12% of women drank green tea less than 1 cup per week while 27% drank 5 or more cups per day. Speaking about the survey, Iwasaki said, "The other major strength of the present study was its prospective design, in which information was collected before the subsequent diagnosis of breast cancer, thereby avoiding the exposure recall bias inherent to case-control studies. Drinking green tea as a beverage is unlikely to reduce the risk of breast cancer regardless of green tea type and number of cups".

1. Green tea drinking and subsequent risk of breast cancer in a population-based cohort of Japanese women
Motoki Iwasaki, Manami Inoue, Shizuka Sasazuki, Norie Sawada, Taiki Yamaji, Taichi Shimazu, Walter C Willett, Shoichiro Tsugane and for the Japan Public Health Center-based Prospective Study Group (JPHC)
Breast Cancer Research (in press) http://breast-cancer-research.com/imedia/2113150999395802_article.pdf?random=472472
http://www.eurekalert.org/pub_releases/2010-10/bc-rrg102610.php

New methods detect subtleties in human genomes' repetitive landscapes

These techniques spot minute variations linked to evolution, diversity and brain development

Scientists have invented methods to scout the human genome's repetitive landscapes, where DNA sequences are highly identical and heavily duplicated. These advances, as reported today in Science, can identify subtle but important differences among people in the number and content of repeated DNA segments.

These copy number variations partly account for the normal diversity among people. Copy number variations might also be why some people, and not others, have certain disorders or disease susceptibilities, and might also determine how severely they are affected.

Until about a year ago, locating and counting the number of duplicated copies of DNA sequences was almost impossible. The more copies of a duplicated gene that are present, the harder they are to assess accurately.

"These difficulties resulted in a lack of understanding of the true extent of human copy number variation, " said Dr. Evan E. Eichler, University of Washington (UW) professor of genome sciences and senior author of the Science paper, "The most dynamic and variable genes are frequently excluded from genome-wide studies." These hard-to-study genes are also among the most interesting because of their suspected contributions to human evolution, brain development, metabolism and disease immunity.

Researchers in Eichler's lab have developed several analytical and computational techniques to overcome obstacles in looking at multicopy genes. The lead authors of the study are Peter H. Sudmant and Jacob O. Kitzman, both graduate students in the UW Department of Genome Sciences.

Working with colleagues in the 1000 Genomes Project and at Agilent Technologies, the UW group used the new techniques to deeply probe and compare the genomes of 159 individuals. In assessing the entire genomes of these individuals, the researchers were able to accurately assay previously intractable duplicated genes and gene families.

The researchers demonstrated that the methods could estimate correctly the absolute number of copies of segments as small as 1,900 DNA base pairs, and could count numbers of copies ranging from 0 to 48. A human genome is made up of about 3 billion DNA base pair. Each pair consists of two bonded molecules called nucleotides, the basic structural unit of DNA.

"We identified 4.1 million singly unique nucleotide positions informative in distinguishing specific copies," the authors reported. The researchers took this information to genotype the number of copies and the content of genes that had been duplicated to or more different positions on the genome thereby became free to function on their own. These duplicated genes reveal changes that occurred during evolution.

The data allowed the researchers to identify duplicated genes specific to humans, in comparison to apes like gorilla, orangutans, and chimps. The researchers observed that these duplications occurred in genes associated with brain development. These include genes implicated in the growth and branching of brain cell connections,

in abnormally large or small head size, in a particular dopamine (reward signal in the brain) receptor, in visual-spatial and social deficits, in reducing the severity of spinal muscular atrophy, and in intellectual disability and epilepsy.

Copy number variations occur in only about 7 percent to 9 percent of human genes, the researchers found. Most of our genes come standard: two copies. Even among copy number variable genes, the researchers learned that 80 percent of them vary between 0 and 5 copies.

"Extreme gene variation," the researchers noted, "is limited to only a few gene families." In this study, they identified 56 of the most variable gene families. These ranged in median copy number from 5 to approximately 368. "These genes were dramatically enriched for segmental duplication," the researchers noted. Segmental duplications are regions that were originally identified in the Human Genome Project as long, repeated blocks of the genome.

The researchers report discovering about 44 "hidden" members of duplicated gene families never before identified in the reference model of the human genome.

"The missing members of these gene families," the researchers suggested, "should be targeted for sequence finishing in order to more accurately capture the architecture and diversity of the human genome."

While duplications of segments of the genome appear to have led to many of the qualities that distinguish human beings from other primate species, areas of the genome in which duplications promote recurrent rearrangements have also been associated with debilitating diseases like intellectual disability, schizophrenia and autism. The researchers hypothesize, "Extreme variation resulting from duplications may contribute to genomic instability associated with disease."

Overall, the results of the study shows scientists can now leverage newly developed techniques to explore some of the most complex genetic regions of the human genome. Still, a portion of the genome remains impenetrable. About 28 large regions of the human genome have such extraordinary complexity that as yet it is impossible to interpret the underlying pattern of genetic diversity, the authors said.

Despite this limitation, the approaches tested in the study hold promise for improving the understanding of how copy number variation contributes to human health and illness.

"Our approach," the researchers concluded," makes many of the highly duplicated regions of the human genome – and the more than 1,000 previously inaccessible human genes that lie therein –accessible to genetic studies of disease association."

In addition to Eichler, Sudmant, and Kitzman, the scientists on the study are Francesca Antonacci, Can Alkan and Maika Malig, all from the UW Department of Genome Sciences; Anya Tsalenko, Nick Sampas, and Laurakay Bruhn, all of Agilent Technologies in Santa Clare, Calif., Jay Shendure, UW assistant professor of genome sciences, and participants and institutions in the 1000 Genome Project.

The work was supported by a Natural Sciences and Engineering Research Council of Canada Fellowship, a National Science Foundation Fellowship, and a grant from the National Human Genome Research Institute, National Institutes of Health. Eichler is an investigator of the Howard Hughes Medical Institute.

http://www.eurekalert.org/pub_releases/2010-10/uow-nmd102510.php

Portable breast scanner allows cancer detection in the blink of an eye

(PhysOrg.com) -- Professor Zhipeng Wu has invented a portable scanner based on radio frequency technology, which is able to show in a second the presence of tumours – malignant and benign – in the breast on a computer.

Using radio frequency or microwave technology for breast cancer detection has been proven by researchers in the US, Canada and UK. However, up to now, it can take a few minutes for an image to be produced, and this had to be done in a hospital or specialist care centre. Now Professor Wu, from the University's School of Electrical and Electronic Engineering, says concerned patients can receive real-time video images in using the radio frequency scanner which would clearly and simply show the presence of a tumour.

Not only is this a quicker and less-intrusive means of testing, it also means women can be tested at GP surgeries, which could help dramatically reduce waiting times and in some cases avoid unnecessary X-ray mammography. The scanner could also be used at home for continuous monitoring of breast health.

The patented real-time radio frequency scanner uses computer tomography and works by using the same technology as a mobile phone, but with only a tiny fraction of its power. This makes it both safe and low-cost and the electronics can be housed in a case the size of a lunch box for compactness and portability. Other existing systems are much larger.

Breast cancer is the second biggest killer in women, accounting for 8.2% of all cancer deaths. October is National Breast Cancer Awareness month.

The usual way of detecting breast cancer up to now is mammography, which works well for women over the age of 50 and can give results of up to 95% accuracy.

But it is far less effective for younger women. The detection rate could be as low as 60% for women under the age of 50, which accounts for 20% of all breast cancer cases. At that stage it is even more important get accurate diagnosis. Early diagnosis and treatment could save thousands of lives.

The main difference between the two methods is that mammography works on density, while radio frequency technique works on dielectric contrasts between normal and diseased breast tissues.

In Professor Wu's design, as soon as the breast enters the cup an image appears on screen.

The presence of a tumour or other abnormality will show up in red as the sensor detects the difference in tissue contrasts at radio frequencies. Malignant tissues have higher permittivity and conductivity and therefore appear differently than normal ones to a screen. Up to 30 images are generated every second, meaning a breast scan could be over in a far shorter time than they are currently.

Professor Wu said: "The system we have is portable and as soon as you lie down you can get a scan – it's real-time. "The real-time imaging minimises the chance of missing a breast tumour during scanning.

"Other systems also need to use a liquid or gel as a matching substance, such as in an ultrasound, to work but with our system you don't need that – it can be done simply in oil, milk, water or even with a bra on. "Although there is still research to be done, the system has great potential to bring a new way for breast cancer diagnosis.

"This will benefit millions of women in both developed and developing countries bearing in mind that one in nine women may develop breast cancer in their lifetime."

Professor Wu submitted his innovation of the sensor system to the IET Innovation Awards. The technology has been shortlisted in both Electronics and Measurement in Action categories. The winners will be announced in November. *Provided by University of Manchester*

http://www.physorg.com/news/2010-10-portable-breast-scanner-cancer-eye.html

Caught on video: Why BA 009 rose from the ashes

* 16:21 27 October 2010 by Paul Marks

The passengers on board a 1982 British Airways flight from London to Auckland, New Zealand, know that volcanic ash and jet engines are a dangerous mix. All four of BA flight 009's engines failed simultaneously after it flew through an ash cloud. But mysteriously, they all kicked into life before disaster could strike. Now we might finally be able to say why.

Although 100,000 flights were cancelled earlier this year following the eruption of the Eyjafjallajökull volcano in Iceland, little is known about the effects of ash on jet engines. One reason for this is the reluctance of engine makers to destructively test many different types of engine, each costing in the region of $12 million.

So Thorsteinn Sigfusson at the University of Iceland in Reykjavik and his team decided to mimic in the lab what happens when a jet engine flies through ash. They built a ceramic crucible and placed turbine vanes from used jet engines inside, before heating it to 1150 ºC – the typical in-flight temperature.

Next, they sieved volcanic ash collected from farmland near Eyjafjallajökull and kept those particles that were 57 micrometres in diameter or less. These are the particles that reached aircraft cruising altitudes, above 9 kilometres, in European skies during the eruption. They then loaded the ash into a sandblaster and fired it at the hot blades.

Jumping ash

The red-hot vanes became coated in a glassy film as the ash melted, so much so that a real jet engine would clog up and fail. But when the team dropped the temperature to 720 ºC - a temperature an engine will cool to after failing – the ash began jumping off the vanes until most of it had gone.

The glass, says Sigfusson, had reached a transition temperature and changed into a crystal structure that could not stick to the alloy.

"With the BA plane in 1982, the natural transition in the basaltic glass somehow cleaned the deposits from the blades so they could restart the engines. I think we are seeing that same glass transition," says Sigfusson.

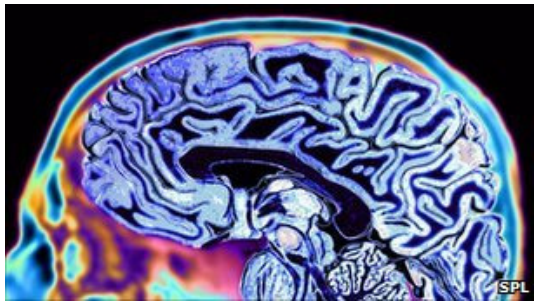
"My vision would be that we can look at ways to somehow de-ash an engine that has passed a volcano with this knowledge," he says. "It shows that cooling the engine can cause self-removal of the trouble."

The finding will be submitted to the Icelandic Civil Aviation Administration. It will be seized upon by engineers, says Fred Prata at the Norwegian Institute for Air Research in Kjeller. "I have never seen ash actually melting on a turbine blade or other hot engine part before," he says. Analysis of the video could be "helpful and fascinating", he adds.

http://www.newscientist.com/article/dn19647-caught-on-video-why-ba-009-rose-from-the-ashes.html

Dream recording device 'possible' researcher claims

By Pallab Ghosh Science correspondent, BBC News



The researchers have developed a way to record higher brain activity

A US researcher has said he plans to electronically record and interpret dreams.

Writing in the journal Nature, researchers said they have developed a system capable of recording higher-level brain activity. "We would like to read people's dreams," says the lead scientist Dr Moran Cerf.

The aim is not to interlope, but to extend our understanding of how and why people dream.

For centuries, people have been fascinated by dreams and what they might mean; in ancient Egypt for example, they were thought to be messages from the gods. More recently, dream analysis has been used by psychologists as a tool to understand the unconscious mind. But the only way to interpret dreams was to ask people about the subject of their dreams after they had woken up.

The eventual aim of Dr Cerf's project is to develop a system that would enable psychologists to corroborate people's recollections of their dream with an electronic visualisation of their brain activity.

"There's no clear answer as to why humans dream," according to Dr Cerf. "And one of the questions we would like to answer is when do we actually create this dream?"

Dr Cerf makes his bold claim based on an initial study that he says suggests that the activity of individual brain cells, or neurons, are associated with specific objects or concepts.

He found, for example, that when a volunteer was thinking of Marilyn Monroe, a particular neuron lit up.

By showing volunteers a series of images, Dr Cerf and his colleagues were able to identify neurons for a wide range of objects and concepts - which they used to build up a database for each patient. These included Bill and Hilary Clinton, the Eiffel Tower and celebrities. So by observing which brain cell lit up and when, Dr Cerf says he was effectively able to "read the subjects' minds".

Dream catcher

He admits that there is a very long way to go before this simple observation can be translated into a device to record dreams - a "dream catcher". But he thinks it is a possibility - and he said he would like to try.

The next stage is to monitor the brain activity of the volunteers when they are sleeping. The researchers will only be able to identify images or concepts that correlate with those stored on their database. But this data base could in theory be built up - by for example monitoring neuronal activity while the volunteer is watching a film.

Dr Roderick Oner, a clinical psychologist and dream expert, believes that while this kind of limited visualisation might be of academic interest, it will not really help in the interpretation of dreams or be of use in therapy. "For that you need the entire complex dream narrative," he said.

Another difficulty with the technique is that to get the kind of resolution needed to monitor individual neurons, subjects had to have electrodes surgically implanted deep inside their brain.

In the Nature study, the researchers obtained their results by studying patients who had electrodes implanted to monitor and treat them for brain seizures.

Translating thoughts

But Dr Cerf believes that sensor technology is developing at such a pace that eventually it might be possible to monitor brain activity in this way without invasive surgery. If this were to happen it would open up a range of possibilities. "It would be wonderful to read people's minds where they cannot communicate, such as people in comas," said Dr Cerf.

There have been attempts to create machine interfaces before that aim to translate thoughts into instructions to control computers or machines. But in the main these have tried to tap into areas of the brain involved in controlling movement. Dr Cerf's system monitors higher level areas of the brain and can potentially identify abstract concepts.

"We can sail with our imaginations and think about all the things we could do if we had access to a person's brain and basically visualise their thoughts. "For example, instead of just having to write an email you could just think it. Or another futuristic application would be to think a flow of information and have it written in front of your eyes."

Professor Colin Blakemore, a neuroscientist at Oxford University, believes that it is quite a jump from the limited results obtained in the study to talking about recording dreams.

http://www.bbc.co.uk/news/science-environment-11635625

Study says solar systems like ours may be common

Nearly 25 percent of all sun-like stars may have planets the size of Earth

Nearly one in four stars like the sun could have Earth-size planets, according to a University of California, Berkeley, study of nearby solar-mass stars. UC Berkeley astronomers Andrew Howard and Geoffrey Marcy chose 166 G and K stars within 80 light years of Earth and observed them with the powerful Keck telescope for five years in order to determine the number, mass and orbital distance of any of the stars' planets. The sun is the best known of the G stars, which are yellow, while K-type dwarfs are slightly smaller, orange-red stars.

The researchers found increasing numbers of smaller planets, down to the smallest size detectable today – planets called super-Earths, about three times the mass of Earth.

"Of about 100 typical sun-like stars, one or two have planets the size of Jupiter, roughly six have a planet the size of Neptune, and about 12 have super-Earths between three and 10 Earth masses," said Howard, a research astronomer in UC Berkeley's Department of Astronomy and at the Space Sciences Laboratory. "If we extrapolate down to Earth-size planets – between one-half and two times the mass of Earth – we predict that you'd find about 23 for every 100 stars."

"This is the first estimate based on actual measurements of the fraction of stars that have Earth-size planets," said Marcy, UC Berkeley professor of astronomy. Previous studies have estimated the proportion of Jupiter and Saturn-size exoplanets, but never down to Neptunes and super-Earths, enabling an extrapolation to Earth-size planets.

"What this means," Howard added, "is that, as NASA develops new techniques over the next decade to find truly Earth-size planets, it won't have to look too far."

Because the researchers detected only close-in planets, there could be even more Earth-size planets at greater distances, including within the habitable zone located at about the same distance as the earth is from our sun. The habitable, or "Goldilocks," zone is the distance from a star neither two hot nor too cold to allow the presence of liquid water.

The researchers' results conflict with current models of planet formation and migration, Marcy noted. After their birth in a protoplanetary disk, planets had been thought to spiral inward because of interactions with the gas in the disk. Such models predict a "planet desert" in the inner region of solar systems.

"Just where we see the most planets, models predict we would find no cacti at all," Marcy said. "These results will transform astronomers' views of how planets form."

Howard and Marcy report their results in the Oct. 29 issue of the journal Science.

The astronomers used the 10-meter Keck telescopes in Hawaii to measure the minute wobble of each star. Current techniques allow detection of planets massive enough and near enough to their stars to cause a wobble of about 1 meter per second. That means they saw only massive, Jupiter-like gas giants up to three times the mass of Jupiter (1,000 times Earth's mass) orbiting as far as one-quarter of an astronomical unit (AU) from the star, or smaller, closer super-Earths and Neptune-like planets (15-30 times the mass of the earth). An AU is 93 million miles, the average distance between the earth and the sun.

Only 22 of the stars had detectable planets – 33 planets in all – within this range of masses and orbital distances. After accounting statistically for the fact that some stars were observed more often than others, the researchers estimated that about 1.6 percent of the sun-like stars in their sample had Jupiter-size planets and 12 percent had super-Earths (3-10 Earth masses). If the trend of increasing numbers of smaller planets continues, they concluded, 23 percent of the stars would have Earth-size planets.

Based on these statistics, Howard and Marcy, who is a member of NASA's Kepler mission to survey 156,000 faint stars in search of transiting planets, estimate that the telescope will detect 120-260 "plausibly terrestrial worlds" orbiting some 10,000 nearby G and K dwarf stars with orbital periods less than 50 days.

"One of astronomy's goals is to find eta-Earth (ηEarth), the fraction of sun-like stars that have an earth," Howard said. "This is a first estimate, and the real number could be one in eight instead of one in four. But it's not one in 100, which is glorious news."

Twelve possible planets also were detected, but they need further confirmation, Marcy said. If these candidate planets are included in the count, the team detected a total of 45 planets around 32 stars.

Other coauthors of the paper are John Asher Johnson of the California Institute of Technology (Caltech), Debra A. Fischer of Yale University, Jason T. Wright of Pennsylvania State University, Howard Isaacson of UC Berkeley, Jeff A. Valenti and Jay Anderson of the Space Telescope Science Institute in Baltimore, Md., Doug N. C. Lin of the UC Observatories/Lick Observatory and UC Santa Cruz, and Shigeru Ida of the Tokyo Institute of Technology in Japan.

The research was funded by NASA and the W. M. Keck Observatory, which is operated by the University of California and Caltech.

http://www.eurekalert.org/pub_releases/2010-10/uoc--sss102010.php

Cancer's hiding spots revealed

Discovery that tumor cells can escape from chemotherapy could lead to new treatments that prevent relapse

CAMBRIDGE, Mass. -- In a study of mice with lymphoma, MIT biologists have discovered that a small number of cancer cells escape chemotherapy by hiding out in the thymus, an organ where immune cells mature. Within the thymus, the cancer cells are bathed in growth factors that protect them from the drugs' effects. Those cells are likely the source of relapsed tumors, said Michael Hemann, MIT assistant professor of biology, who led the study.

The researchers plan to soon begin tests, in mice, of drugs that interfere with one of those protective factors. Those drugs were originally developed to treat arthritis, and are now in clinical trials for that use. Such a drug, used in combination with traditional chemotherapy, could offer a one-two punch that eliminates residual tumor cells and prevents cancer relapse, according to the researchers.

"Successful cancer therapy needs to involve a component that kills tumor cells as well as a component that blocks pro-survival signals," said Hemann, who is a member of MIT's David H. Koch Institute for Integrative Cancer Research. "Current cancer therapies fail to target this survival response."

Hemann and graduate student Luke Gilbert described the findings in the Oct. 29 issue of the journal Cell.

In the new study, the researchers treated mice with lymphoma with doxorubicin, a drug commonly used to treat a wide range of cancers, including blood cancers. They found that during treatment, cells that line the blood vessels release cytokines — small proteins that influence immune responses and cell development.

The exact mechanism is not known, but the researchers believe that chemotherapy-induced DNA damage provokes those blood-vessel cells to launch a stress response that is normally intended to protect progenitor cells — immature cells that can become different types of blood cells. That stress response includes the release of cytokines such as interleukin-6, which promotes cell survival.

"In response to environmental stress, the hardwired response is to protect privileged cells in that area, i.e., progenitor cells," said Hemann. "These pathways are being coopted by tumor cells, in response to the frontline cancer therapies that we use."

The discovery marks the first time scientists have seen a protective signal evoked by chemotherapy in the area surrounding the tumor, known as the tumor microenvironment. "It's completely unexpected that drugs would promote a survival response," said Hemann. "The impact of local survival factors is generally not considered when administering chemotherapy, let alone the idea that frontline chemotherapy would induce pro-survival signals."

It remains to be seen if the results will translate to human patients, but the finding does suggest several potential drug targets, including IL-6 and a protein called Bcl2, which is activated by IL-6 and signals cells to stay alive. While the MIT researchers observed this protective effect only in the thymus, they believe there may be other protected areas where tumor cells hide, such as the bone marrow.

This finding could help explain why tumors that have spread to other parts of the body before detection are more resistant to frontline chemotherapy: They may have already engaged a protective cytokine system, helping them to survive the drugs' effects.

Hemann hopes to further clarify the mechanism in future studies, in collaboration with Professor Michael Yaffe, also a member of the Koch Institute. He also plans to investigate whether this kind of pro-survival signal is elicited in other types of cancer, including tumors that have metastasized.

http://www.eurekalert.org/pub_releases/2010-10/miot-chs102510.php

Potential new treatment for deadly nipah and hendra viruses identified by Weill Cornell researchers

Finding may also lead to new treatments for measles, mumps and influenza

NEW YORK (Oct. 28, 2010) -- Scientists at Weill Cornell Medical College have identified a potential new treatment for the Nipah and Hendra viruses, two lethal and emerging viruses for which there is currently no treatment or vaccine available. The approach could also lead to new therapies for measles, mumps and the flu. The new research appears in today's edition of the prestigious journal Public Library of Science (PLoS) Pathogens.

The Nipah and Hendra viruses are members of the genus Henipavirus, a new class of virus in the Paramyxoviridae family, which includes the measles and the human parainfluenza virus (HPIV) that causes pediatric respiratory disease. The henipaviruses are carried by fruit bats (flying foxes) and are capable of causing illness and death in domestic animals and humans.

"These viruses are of great concern. The Hendra virus is highly fatal and is a considered a potential agent of bioterrorism. It currently poses a serious threat to livestock in Australia, where sporadic and deadly transmission to humans has occurred, with the potential for broader dissemination," says Dr. Matteo Porotto,

the study's lead author and assistant professor of microbiology in pediatrics at Weill Cornell Medical College. "And the Nipah virus, which causes fatal encephalitis in up to 70 percent of human cases, causes seasonal outbreaks in Asia with person-to-person transmission now becoming a primary mode of infection. This virus could certainly cause global outbreaks."

Dr. Porotto and colleagues present a new strategy to prevent and treat these infections that may be broadly applicable for other "enveloped" viral pathogens, characterized by an outer wrapping that comes from the infected host cell. The new treatment was successfully tested in an animal model demonstrating central nervous system symptoms similar to those seen in humans.

Dr. Anne Moscona, professor of pediatrics and microbiology & immunology at Weill Cornell Medical College, vice chair of pediatrics for research at NewYork-Presbyterian Hospital/Weill Cornell Medical Center, chief of pediatric infectious diseases and co-corresponding author of the paper, says, "It's crucial that we find treatments for the Nipah and Hendra viruses. In addition to acute infection, they can cause asymptomatic infection in as many as 60 percent of exposed people. They may also lead to late-onset disease or relapse of encephalitis years after initial infection, as well as persistent or delayed neurological problems."

According to Dr. Porotto, it is difficult to treat these pathogens because their "envelope" helps the virus survive and infect other cells. "We know that enveloped viruses must fuse their membrane with the target cell membrane in order to initiate infection, and blocking this step can prevent or treat infection, as has been clinically validated for the HIV virus."

Building on their past work, the team demonstrated in this study that the addition of a cholesterol group to HRC peptides that are active against Nipah virus dramatically increases their antiviral effect. The approach works by using the cholesterol-tagged peptides to target the membrane where the fusion occurs. There, the peptides interact with the fusion peptide before it inserts into the target cell membrane, disrupting the crucial membrane fusion process and preventing infection.

"The cholesterol-tagged HRC-derived peptides cross the blood-brain barrier and help prevent and treat the infection in animals for what would otherwise be fatal Nipah virus encephalitis," Dr. Porotto reports. "This suggests that they are promising candidates for the prevention or therapy of infection by Nipah and other lethal paramyxoviruses and may lead to better treatments for people affected by similar viruses including the measles, mumps and the flu."

Additional co-authors include Christine C. Yokoyama, Aparna Talekar, Ilaria DeVito, Laura M Palermo and Min Lu from Weill Cornell Medical College; Barry Rockx and Heinz Feldmann from the National Institute of Allergy and Infectious Diseases, National Institutes of Health, Hamilton, MT; Riccardo Cortese from CEINGE, Naples, Italy; and Antonello Pessi from PeptiPharma, Rome, Italy.

http://www.eurekalert.org/pub_releases/2010-10/nyph-pnt102810.php

Helping fish get rid of the 'Ich'

Copper sulfate has emerged as an effective treatment for Ichthyophthirius multifiliis, also known as "Ich," a protozoan parasite that appears as white spots on infected fish, according to a U.S. Department of Agriculture (USDA) scientist.

Aquatic toxicologist David Straus with USDA's Agricultural Research Service (ARS) investigated copper sulfate as a method to control both Ich in catfish and a fungus—Saprolegnia—on catfish eggs. Straus works at the ARS Harry K. Dupree Stuttgart National Aquaculture Research Center in Stuttgart, Ark. ARS is the chief intramural scientific research agency of USDA, and this research supports the USDA priority of promoting international food security.

Ich is considered the most prevalent parasite worldwide in ornamental fish, baitfish and food fish, according to Straus. Ich is less common in U.S. aquaculture because of management techniques, but when it occurs, it can kill all the fish in a pond or raceway. It is calculated that Ich was directly responsible for $1.2 million in losses to the catfish industry in 2003. The freshwater fungus Saprolegnia is another major pathogen in fish culture, killing eggs and invading wounds and lesions on juvenile and adult fish.

Straus found copper sulfate is an effective treatment for Ich on fish and fungus on eggs. According to Straus, copper sulfate is the only practical treatment to control Ich in catfish ponds that average about 10 acres in area. It is easy to use, effective and inexpensive, and is safe for the user to handle.

Current approved treatments for fungus on eggs, such as formalin and hydrogen peroxide, are much more expensive. Also, both compounds are hazardous, and there are human safety concerns as well as required storage precautions.

Copper sulfate is not currently approved by the U.S. Food and Drug Administration for therapeutic use in aquaculture, but regulatory action has been deferred pending the outcome of Straus' ongoing research. The chemical is approved by the U.S. Environmental Protection Agency as an algicide and molluscicide. Fish

farmers use copper sulfate to control cyanobacteria that cause off-flavor in fish, and to control snails that transmit parasitic flatworms to fish.

Read more about this and other aquaculture-related research in the October 2010 issue of Agricultural Research magazine, available online at: http://www.ars.usda.gov/is/AR/archive/oct10/fish1010.htm.

http://www.eurekalert.org/pub_releases/2010-10/usdo-hfg102810.php

Dinosaur Skull Found in Church

By Rossella Lorenzi | Thu Oct 28, 2010 10:42 AM ET

Encased in pinkish marble-like slabs supporting a balustrade, this dinosaur -- or what's left of it -- has for centuries been the most faithful presence in the Cathedral of St. Ambrose in Vigevano, a town about 20 miles from Milan.

"The rock contains what appears to be a horizontal section of a dinosaur's skull. The image looks like a CT scan, and clearly shows the cranium, the nasal cavities, and numerous teeth," Andrea Tintori, the University of Milan paleontologist who spotted the fossil near the altar, told Discovery News.

Measuring about 30 cm (11.8 inches), the skull was cut in sections as slabs of the marble-like rock were used to build the Cathedral between 1532 and 1660.

The marble-like balustrade in the Cathedral of Vigevano where the dinosaur skull was found.Andrea Tintori

Indeed, Tintori found a second section of the same skull in another slab nearby.

The calcareous rock in which the dinosaur remains are embedded comes from the rich fossil-bearing site of Mount San Giorgio, which is on the Unesco World Heritage List.

"It is called Broccatello and was mined in Arzo, Switzerland. We know that this type of rock dates geologically to the Lower Jurassic, about 190 million years ago," Tintori said.

It is not clear what animal the skull belonged to. Tintori hopes to solve the mystery with a three-dimensional reconstruction of the fossilized remains.

Horizontal section of the dinosaur skull. The cranium, the nasal lobes and numerous teeth are visible. **Andrea Tintori**

http://news.discovery.com/dinosaurs/dinosaur-skull-found-in-church.html