http://www.itworld.com/it-managementstrategy/194445/fired-techie-created-virtual-chaos-pharma-company

Fired techie created virtual chaos at pharma company

A former IT staffer has pleaded guilty to using a secret vSphere console to wipe company servers

By Robert McMillan, IDG News Service

Logging in from a Smyrna, Georgia, McDonald's restaurant, a former employee of a U.S. pharmaceutical company was able to wipe out most of the company's computer infrastructure earlier this year.

Jason Cornish, 37, formerly an IT staffer at the U.S. subsidiary of Japanese drug-maker Shionogi, pleaded guilty Tuesday to computer intrusion charges in connection with the attack on Feb. 3, 2011. He wiped out 15 VMware host systems that were running e-mail, order tracking, financial and other services for the Florham Park, New Jersey, company.

"The Feb. 3 attack effectively froze Shionogi's operations for a number of days, leaving company employees unable to ship product, to cut checks, or even to communicate via e-mail," the U.S. Department of Justice said in court filings. Total cost to Shionogi: $800,000.

Cornish had resigned from the company in July 2010 after getting into a dispute with management, but he had been kept on as a consultant for two more months.

Then, in September 2010, the drug-maker laid off Cornish and other employees, but it did a bad job of revoking passwords to the network. One employee, who was Cornish's friend and former boss, allegedly refused to hand over network passwords to company officials and eventually was fired because of this.

Using a Shionogi account, Cornish was able to log into the company's network from a public McDonald's Internet connection in February and fire up a vSphere VMware management console that he'd secretly installed on the company's network a few weeks earlier.

Using vSphere, he deleted 88 company servers from the VMware host systems, one by one.

Cornish was charged in July. He faces a maximum of 10 years in prison when he's sentenced on Nov. 10. He could not be reached for comment Tuesday. Shionogi did not return messages seeking comment.

Robert McMillan covers computer security and general technology breaking news for The IDG News Service.

http://www.eurekalert.org/pub_releases/2011-08/uotm-sf1081511.php

Study finds 15 minutes of moderate daily exercise lengthens life

Health benefits of physical activity found to begin before people reach the half-hour standard

HOUSTON - Taiwanese who exercise for 15 minutes a day, or 92 minutes per week, extended their expected lifespan by three years compared to people who are inactive, according to a study published today in The Lancet.

"Exercising at very light levels reduced deaths from any cause by 14 percent," said study senior author Xifeng Wu, M.D., Ph.D., professor and chair of The University of Texas MD Anderson Cancer Center Department of Epidemiology. "The benefits of exercise appear to be significant even without reaching the recommended 150 minutes per week based on results of previous research."

Lead author Chi-Pang Wen, M.D., of the National Health Research Institutes of Taiwan, and colleagues also found that a person's risk of death from any cause decreased by 4 percent for every additional 15 minutes of exercise up to 100 minutes a day over the course of the study. Those exercising for 30 minutes daily added about four years to life expectancy. "These benefits were applicable to all age groups, both sexes and those with cardiovascular disease risk," the authors note.

If inactive people in Taiwan were to do low-volume daily exercise, one in six deaths could be postponed by their reduced risk of dying, the authors report. It would be an estimated reduction in mortality similar to that from a successful tobacco control program.

The prospective observational study involved 416,175 Taiwanese who participated in a standard medical screening program run by MJ Health Management Institution between 1996 and 2008. Participants were followed for an average of eight years.

For the exercise study, participants completed a questionnaire covering their medical history and lifestyle information. They characterized their weekly physical activity for the previous month by intensity - light (walking), moderate (brisk walking), vigorous (jogging) or high vigorous (running) – and time.

To account for occupational effects, participants also characterized their physical activity at work, ranging from sedentary to hard physical labor.

Those who reported less than one hour a week of leisure time physical activity were classified as inactive – 54 percent of all participants. Others were classified as low, medium, high or very high based on the duration and intensity of their exercise. Researchers calculated mortality risk and life expectancy for each group.

Thirteen other variables were analyzed: age, sex, education level, physical labor at work, smoking, alcohol use, fasting blood sugar, systolic blood pressure, total cholesterol, body mass index, diabetes, hypertension and history of cancer.

Those who engaged in low-volume exercise had lower death rates than inactive people regardless of age, gender, health status, tobacco use, alcohol consumption or cardiovascular disease risk.

The researchers note that the World Health Organization and the U.S. Centers for Disease Control and Prevention recommend at least 150 minutes of moderate-intensity exercise per week. A third of U.S. adults meet that guideline; about 20 percent of adults in China, Japan or Taiwan meet it. "A recommendation of 15 minutes of daily exercise should be promoted to East Asian populations," the authors note.

The study's findings of reduced mortality through even moderately intense exercise are likely to hold true for other populations, Wu said, even though the total amount of time spent or workout intensity required for a health benefit might differ. "These findings can stimulate people to exercise as much as they can and to not be frustrated that they can't reach the 30 minute per day guideline."

This is the first collaboration between Wu, Wen and the MJ Health Group, a major health screening company with more than 600,000 participants in its health database. They have formed the Asian Health Screening Cohort to conduct major research projects. Wu provides scientific expertise with Wen, who also is based at China Medical University Hospital, while MJ Health Group contributes patient epidemiological and clinical data as well as a biobank of tissue samples.

Two other ongoing collaborative projects include development of a liver cancer risk prediction model and a study of telomere length, genetic variation and cancer risk. The second project is funded by an MD Anderson Sister Institute Network Fund Grant. MD Anderson and China Medical University Hospital have a sister institution agreement.

The exercise project was funded by the Taiwan Department of Health Clinical Trial and Research Center of Excellence and the Taiwan National Health Research Institutes.

Co-authors with Wen and Wu are: co-lead author Jackson Pui Man Wai, Ph.D., of the Institute of Sport Science, National Taiwan Sport University; Min Kuang Tsai, Yi Chen Yang and Hui Ting Chan of the Institute of Population Science, Taiwan National Health Research Institutes; Tsai and Yang also are with the China Medical University Hospital; Ting Yuan David Cheng, University of Washington Department of Epidemiology; Meng-Chih Lee, M.D., Institute of Medicine and Department of Family and Community Medicine, Chung Shan Medical University and Hospital; Chwen Keng Tsao of MJ Health Management Insitution; and Shan Pou Tsai, Ph.D., The University of Texas School of Public Health at Houston.

http://www.eurekalert.org/pub_releases/2011-08/luhs-mdp081511.php

Moderate drinking protects against Alzheimer's and cognitive impairment

Researchers review 143 studies

MAYWOOD, Ill. - Moderate social drinking significantly reduces the risk of dementia and cognitive impairment, according to an analysis of 143 studies by Loyola University Chicago Stritch School of Medicine researchers.

Researchers reviewed studies dating to 1977 that included more than 365,000 participants. Moderate drinkers were 23 percent less likely to develop cognitive impairment or Alzheimer's disease and other forms of dementia.

Wine was more beneficial than beer or spirits. But this finding was based on a relatively small number of studies, because most papers did not distinguish among different types of alcohol. Results are reported in the journal Neuropsychiatric Disease and Treatment. The authors are Edward J. Neafsey, PhD. and Michael A. Collins, PhD., professors in the Department of Molecular Pharmacology and Therapeutics.

Heavy drinking (more than 3 to 5 drinks per day) was associated with a higher risk of cognitive impairment and dementia, but this finding was not statistically significant. "We don't recommend that nondrinkers start drinking," Neafsey said. "But moderate drinking - if it is truly moderate - can be beneficial." Moderate drinking is defined as a maximum of two drinks per day for men and 1 drink per day for women.

Among the studies reviewed, 74 papers calculated the ratios of risk between drinkers and non-drinkers, while 69 papers simply stated whether cognition in drinkers was better, the same or worse than cognition in nondrinkers. Neafsey and Collins did a meta-analysis of the studies that calculated risk ratios and found that moderate drinkers were 23 percent less likely to develop dementia or cognitive decline.

Other findings:

The protective effect of moderate drinking held up after adjusting for age, education, sex and smoking.

There was no difference in the effects of alcohol on men and women. The beneficial effect of moderate drinking was seen in 14 of 19 countries, including the United States. In 3 of the remaining 5 countries, researchers also found a benefit, but it was not strong enough to be statistically significant.

The findings were similar across different types of studies (longitudinal cohort studies, case-control studies and cross-sectional studies).

It is unknown why moderate drinking can have a beneficial effect. One theory suggests that the well-known cardiovascular benefits of moderate alcohol consumption, such as raising good HDL cholesterol, also can improve blood flow in the brain and thus brain metabolism.
A second possible explanation involves "sick quitters." According to this theory, nondrinkers have a higher risk of cognitive impairment and dementia because the group includes former heavy drinkers who damaged their brain cells before quitting. But the analysis by Neafsey and Collins did not support this explanation. They found that in studies that excluded former heavy drinkers, the protective effect of moderate drinking still held up.
Neafsey and Collins suggest a third possible explanation: Small amounts of alcohol might, in effect, make brain cells more fit. Alcohol in moderate amounts stresses cells and thus toughens them up to cope with major stresses down the road that could cause dementia.
For people who drink responsibly and in moderation, there's probably no reason to quit. But because of the potential for alcohol to be abused, Neafsey and Collins do not recommend that abstainers begin drinking.
The researchers note that there are other things besides moderate drinking that can reduce the risk of dementia, including exercise, education and a Mediterranean diet high in fruits, vegetables, cereals, beans, nuts and seeds. Even gardening has been shown to reduce the risk of dementia.
Moreover, there are times when people should never drink, including adolescence, pregnancy and before driving, the researchers said. *The Neafsey and Collins study was supported by the National Institutes of Health.*

http://www.economist.com/node/21525840

False confessions: Silence is golden

People have a strange and worrying tendency to admit to things they have not, in fact, done

SINCE 1992 the Innocence Project, an American legal charity, has used DNA evidence to help exonerate 271 people who were wrongly convicted of crimes, sometimes after they had served dozens of years in prison. But a mystery has emerged from the case reports. Despite being innocent, around a quarter of these people had confessed or pleaded guilty to the offences of which they were accused.

It seems hard to imagine that anyone of sound mind would take the blame for something he did not do. But several researchers have found it surprisingly easy to make people fess up to invented misdemeanours. Admittedly these confessions are taking place in a laboratory rather than an interrogation room, so the stakes might not appear that high to the confessor. On the other hand, the pressures that can be brought to bear in a police station are much stronger than those in a lab. The upshot is that it seems worryingly simple to extract a false confession from someone - which he might find hard subsequently to retract.

I must confess

One of the most recent papers on the subject, published in Law and Human Behavior by Saul Kassin and Jennifer Perillo of the John Jay College of Criminal Justice in New York, used a group of 71 university students who were told they were taking part in a test of their reaction times. Participants were asked to press keys on a keyboard as they were read aloud by another person, who was secretly in cahoots with the experimenter. The volunteers were informed that the ALT key was faulty, and that if it was pressed the computer would crash and all the experimental data would be lost. The experimenter watched the proceedings from across the table.

In fact, the computer was set up to crash regardless, about a minute into the test. When this happened the experimenter asked each participant if he had pressed the illicit key, acted as if he was upset when it was "discovered" that the data had disappeared, and requested that the participant sign a confession. Only one person actually did hit the ALT key by mistake, but a quarter of the innocent participants were so disarmed by the shock of the accusation that they confessed to something they had not done.

Robert Horselenberg and his colleagues at Maastricht University, in the Netherlands, have come up with similar results. In an as-yet-unpublished study, members of Dr Horselenberg's group told 83 people that they were taking part in a taste test for a supermarket chain. The top taster would win a prize such as an iPad or a set of DVDs. The volunteers were asked to try ten cans of fizzy drink and guess which was which. The labels were obscured by socks pulled up to the rim of each can, so to cheat a volunteer had only to lower the sock.

During the test, which was filmed by a hidden camera, ten participants actually did cheat. Bafflingly, though, another eight falsely confessed when accused by the experimenter, despite participants having been told cheats would be fined €50 ($72).

The number of innocent confessors jumps when various interrogation techniques are added to the mix. Several experiments, for example, have focused on the use of false evidence, as when police pretend they have proof of a person's guilt in order to encourage him to confess. This is usually permitted in the United States, though banned in Britain.

A second computer-crash test conducted by Dr Kassin and Dr Perillo used this technique. Another person in the room beside the experimenter said he saw the participant hitting the ALT key. In this case the confession rate jumped to 80% of innocent participants. Dr Horselenberg and his colleagues found something similar.

Dr Kassin also tested the impact of bluffing. Two participants, one of whom was again in cahoots with the investigator, sat in the same room and were asked to complete what appeared to be an academic test. Halfway through, the investigator accused them of helping each other and cited the university's honour code against cheating. The investigator went on to bluff that there was a video camera in the room, though the recording, with its definitive proof one way or the other, would not be accessible until later. In the real world, this might be like a detective telling a suspect that DNA or fingerprint evidence had been found but not yet analysed (in Britain as well as America, if such a statement were actually true, police would be permitted to say it, though in the case of the experiment it was a lie). Presumably, the innocent participants knew such a tape would exonerate them. Even so, half still confessed.

All of which is both strange and rather alarming. Dr Kassin suggests that participants may have the naive - though common - belief that the world is a just place, and that their innocence will emerge in the end, particularly in the case of the alleged video evidence. One participant, for example, told him, "it made it easier [to sign the confession] because I had nothing to hide. The cameras would prove it."

In cases like that, confession is seen as a way to end an unpleasant interrogation. But it is a risky one. In the real world, such faith can be misplaced. Though a lot of jurisdictions require corroborating evidence, in practice self-condemnation is pretty damning - and, it seems, surprisingly easy to induce.

http://www.physorg.com/news/2011-08-fish-bones-decontaminate-soil-lead-poisoned.html

Fish bones used to decontaminate soil in a lead-poisoned neighborhood

There's something fishy going on in West Oakland.

Rows of bulging white, 1-ton sacks of ground fish bone from Alaska are lined up inside a shed in the South Prescott neighborhood. In coming months, workers will till the bones into lead-contaminated soil, where it will bind with the toxic metal as it decays, creating a tiny, harmless crystal.

"It may smell fishy for a few days," said Steve Calanog, the U.S. Environmental Protection Agency scientist overseeing the nation's first fish-bone lead decontamination of a residential area. "But it's the smell of change, of a healthy community coming back, of getting rid of lead."

A poisoned neighborhood will become safer for children, and a project that will revitalize yards and gardens and employ local workers could lead to wider use of a relatively cheap, effective decontamination method.

South Prescott, a six-block neighborhood one mile west of downtown Oakland, has the heavy lead contamination found in many urban and industrialized areas. Some yards have more than five times the federal health standard of 400 parts per million of lead in soil, and the neighborhood on average has twice as much, EPA tests in 2009 revealed. Exhaust from now-banned leaded gasoline, peeling lead-based paint in old homes and businesses such as auto repair shops and metal recycling facilities account for much of the contamination.

Lead is a neurotoxin especially dangerous for young children. It damages developing brains, leading to cognitive and behavioral problems as children grow, among other effects.

Toddlers playing in contaminated dirt pick up lead by ingesting it. And they absorb more of it than do adults.

The fish bone treatment is highly effective, Calanog said. One study found it reduced lead leaching through soil 100-fold. It works fast, too. Workers in early July tilled bone into the first test site in Oakland, and it's now safe for residents, Calanog said. In late July, the former weedy vacant lot was landscaped into a small neighborhood park, with a stone patio, walkway, lawn and shrubs, paid for by the EPA.

The larger, two-year, $4 million project, means full-time jobs for 20 to 30 West Oakland residents, many previously unemployed and most graduates of the Cypress Mandela Training Center. The primary contractor is SFS Chemical Safety, a local woman-owned business, and other local enterprises are involved in security, garden design, employment screening, solar installation, site maintenance and other services.

Some 150 homes in South Prescott qualify for the lead decontamination. After the treatment, homeowners can choose to recreate the original landscaping or select a new, equal-cost one.

The fish bone, like other bone, is largely made of calcium and phosphate. As it decomposes, freed phosphate migrates through moistened soil. It chemically and permanently binds to the toxic metal, creating a microscopic crystal, pyromorphite, that is harmless even if consumed. The method has been under research for more than 15 years and has been used successfully at to clean up lead at military firing ranges and ordnance test sites.

The fish bone comes from Alaskan pollock, familiar to fans of fast-food fish sandwiches and faux crab.

South Prescott residents told the EPA they preferred the innovative treatment over the traditional approach: hauling tainted topsoil to a hazardous waste landfill and replacing it. "The community asked to look at a

different approach," Calanog said. A South Prescott neighborhood website declared, "We are a forward-looking, environmentally conscious community and want no part of giving our problem to someone else."

The fish bone cleanup costs $18 a square foot, compared with $32 a square foot for "dig-and-haul," according to an EPA spokesman. Calanog regards the South Prescott lead remediation as much a test of social science as of chemistry or geology, because it takes a neighborhood's embrace for it to succeed.

Bruce Beasley, a renowned sculptor and nearly 50-year resident of South Prescott, helped build support for the project as chairman of the neighborhood association. He sees better health, a rejuvenated neighborhood landscape and improved property values. The EPA's willingness to modify the project in response to neighbors' concerns was critical to its acceptance, Beasley said.

Neighbors insisted that an arborist watch over rototilling to ensure that prized trees and large shrubs weren't damaged. To escape the noise and smell, residents could stay for free in a hotel during the one to two weeks it takes to treat each yard. The neighborhood group also won the promise that Calanog, whom Beasley described as "a straight shooter," would remain as EPA's head of the project during its duration.

"The sense of trust is very much directed by the person, not the institution," Beasley said. "That came from our own experience with the government." The EPA also set up a solar-powered work site with a community library and meeting room and a demonstration garden with landscape options. A biodiesel truck and an electric truck haul equipment and supplies.

Calanog said he'll be working with a few research laboratories to monitor how well the Oakland project cuts residents' lead exposure. If the results show scientific and social success, Calanog hopes it becomes a model for other communities. "What we're trying to do is show this is accessible to all," he said.

http://medicalxpress.com/news/2011-08-chocolate-wards-hunger-sunburn.html

Chocolate wards off hunger, and maybe sunburn too

Scientists in Canada said Monday that they plan to study whether eating dark chocolate not only satisfies sweet tooth cravings, but protects against sunburn as well.

The study by researchers at Laval University in Quebec will monitor the effects of chocolate consumption on fair-skinned volunteers between the ages of 25 and 65, each of whom will be prevailed upon to eat three squares of chocolate per day for 12 weeks. Earlier research in Germany and Britain has found that chemicals in chocolate called polyphenols increase blood flow close to the skin, which helps protect against ultraviolet rays, but those studies were too small in scale to be conclusive.

The scientists at Laval University's Institute of Nutraceuticals and Functional Foods hope to confirm the link in a study of 60 people, about half of whom already have been recruited, they said.

Test subjects who have been fed chocolate are to be exposed to ultraviolet rays in a laboratory and their skin then checked for sun damage. A control group will be given a placebo.

Scientists said the study will only include female participants so that researchers can control for the variability between the body's natural hormones in men and women.

http://www.eurekalert.org/pub_releases/2011-08/nioa-apm081611.php

Any prime-boost mix of injected or spray flu vaccine shields toddlers

Broadest immune response from nasal spray vaccine, NIH-funded study finds

Children younger than 3 years old receive the same protective antibody response from the recommended two doses of licensed seasonal influenza vaccines regardless of whether the two doses are injected by needle, inhaled through a nasal spray or provided through one dose of each in any order, according to researchers funded by the National Institutes of Health. Doctors usually give young children two matching vaccines, and one goal of the study was to determine whether giving two different types of vaccines works just as well.

In addition, the researchers found that young children who received at least one dose of the nasal spray vaccine - a live, attenuated influenza virus vaccine (LAIV) - made a wide array of immune T cells. Stimulating broad T-cell responses may be important for protection against many diverse flu strains.

The study was conducted at vaccine and treatment evaluation units (VTEUs) funded by the NIH's National Institute of Allergy and Infectious Diseases (NIAID). Daniel F. Hoft, M.D., Ph.D., and his colleagues at the Saint Louis University VTEU, along with other VTEU investigators at Vanderbilt University in Nashville, and the Cincinnati Children's Hospital, co-authored the report, online in The Journal of Infectious Diseases.

Influenza vaccinations for young children are provided in a two-dose, prime-boost combination. The first vaccine dose is designed to prime the immune system to produce a favorable antibody response, and the second vaccine dose is the "boost" designed to spur an immune response.

"Severe complications from seasonal influenza can be devastating to young children," said Anthony S. Fauci, M.D., NIAID director. "This study provides initial evidence that the prime and booster doses for these

young children can be different types of flu vaccines and still provide adequate protection against matching seasonal flu strains."

The trial took place during the 2005-2006 and the 2006-2007 flu seasons and involved 53 children aged 6 to 35 months. Study participants were divided into four groups of roughly equal size. None of the children had received an influenza vaccine before. During the study, all children received an initial dose of licensed seasonal flu vaccine and a booster dose one month later. In two groups, the vaccines matched, with children receiving two injections of a trivalent, inactivated vaccine (TIV) injection or two LAIV nasal spray vaccines. Children in the other two groups received a combination of vaccines, with a dose of LAIV given either before or after TIV. "Kids who have never been immunized against flu are generally advised to receive two doses of inactivated or live, attenuated vaccine to ensure adequate antibody responses," noted Dr. Hoft, director of the division of infectious diseases, allergy and immunology at Saint Louis University. "Vaccination schedules combining one dose of TIV with one dose of LAIV have not been recommended because clinical studies of these combinations have not been done previously. However, due to vaccine availability or other factors, children may sometimes be given a mix of vaccines," he added.

The researchers found that all four dosing patterns were safe and induced similar levels of protective antibodies. However, when the investigators looked at responses from the T-cell arm of the immune system, a striking difference emerged. They could not detect influenza-specific T cells in children who received only TIV, according to Dr. Hoft. But, he added, "The kids who received at least one dose of LAIV nasal spray vaccine produced significant amounts of three important T-cell subtypes that are likely to confer additional protection beyond that afforded by antibodies alone."

Previous studies have demonstrated that children are better protected against influenza infection and disease by LAIV than by TIV, Dr. Hoft noted. However, few studies have examined the T-cell responses elicited by LAIV when given to very young children who have little prior natural exposure to seasonal influenza viruses. Distinguishing T-cell responses due to vaccination from those arising after natural exposure to influenza virus becomes more difficult as a child ages, he explained. Because the children in the trial were all younger than 3 years old, the researchers could be confident that spikes in levels of the three T-cell subtypes they detected were likely due to the vaccinations.

Children who received only one dose of LAIV had T-cell responses similar to those who received two, and the order of vaccine types did not make a significant difference in the size of the T-cell response. However, because LAIV has sometimes been associated with increased incidence of wheezing in the youngest recipients, the results of this trial suggest that the best regimen for kids younger than 24 months may be TIV followed by LAIV, Dr. Hoft said. Larger clinical trials are required to confirm the safety and efficacy of such an approach, he added.

In a separate series of experiments using influenza virus-infected cells grown in the laboratory, the scientists showed that LAIV, but not TIV, induces T cells that recognize genetic sequences shared by a diverse set of influenza viruses. In contrast to currently used flu vaccines - which must be given annually because circulating influenza viruses change from season to season - a vaccine capable of eliciting broad T-cell responses aimed at a portion of virus shared by multiple strains could provide decades-long protection against many or all flu strains, Dr. Hoft said.

Reference: *DF Hoft et al. Live and inactivated influenza vaccines induce similar humoral responses, but only live vaccines induce diverse T cell responses in young children. The Journal of Infectious Diseases DOI: 10.1093/infdis/JIR436 (2011).*

Further information about this trial (NCT00231907) can be found at clinicaltrials.gov.

http://www.eurekalert.org/pub_releases/2011-08/rumc-rrd081011.php

Rush researchers discover antibody that may help detect ovarian cancer in earliest stages

Antibody discovered in the bloodstream of infertile women, who are at high risk for the disease

CHICAGO - Using a new approach to developing biomarkers for the very early detection of ovarian cancer, researchers at Rush University Medical Center have identified a molecule in the bloodstream of infertile women that could one day be used to screen for those at high risk for the disease - or even those with early-stage ovarian cancer.

The molecule, an antibody that the human body manufactures, is an autoimmune response to mesothelin. This well-studied protein is found in abundance on the surface of ovarian cancer cells but present only in limited amounts in normal human tissue. The study is published in the online version issue of Cancer Epidemiology, Biomarkers & Prevention, published by the American Society for Cancer Research.

"The finding is extremely important because at present medical tests are unable to detect ovarian cancer in its early stages, which is why death rates from this disease are so high," said Judith Luborsky, PhD, professor of pharmacology, obstetrics and gynecology and preventive medicine at Rush and lead author of the study.

"Our approach to discovering cancer biomarkers was unique in this study. Instead of investigating molecules specific to ovarian cancer alone, we asked what molecules women with a risk of ovarian cancer and those with ovarian cancer had in common," Luborsky said.

The study enabled the researchers to explain the link between infertility and ovarian cancer that has been established in numerous epidemiological surveys.

"More important, with the discovery of the mesothelin antibody, we now have what appears to be a biomarker that can potentially be used in screening tests to help us conquer ovarian cancer," Luborsky said.

According to the American Cancer Society's most recent estimates, there are expected to be about 21,900 new cases of ovarian cancer in the U.S. in 2011 and about 15,460 deaths from the disease. Ovarian cancer is the ninth most common cancer in women (not counting skin cancer) and ranks fifth as the cause of cancer death in women. The poor prognosis for women with ovarian cancer is due to the lack of both clinical symptoms when the cancer first develops and the absence of laboratory tests specific to the disease.

In the study at Rush, researchers tested for mesothelin antibodies in the bloodstream of 109 women who were infertile, 28 women diagnosed with ovarian cancer, 24 women with benign ovarian tumors or cysts, and 152 healthy women. Infertility was due to endometriosis, ovulatory dysfunction or premature ovarian failure or was unexplained.

Significant levels of mesothelin antibodies were found in women with premature ovarian failure, ovulatory dysfunction and unexplained infertility, as well as in women with ovarian cancer, although not in women with endometriosis and not in healthy women or women with benign disease. Endometriosis is generally associated with a different kind of ovarian carcinoma than other types of infertility, which may explain why mesothelin antibodies were not found in these cases.

Why the presence of mesothelin antibodies in the bloodstream should be linked with ovarian cancer is not clear. "It has been hypothesized that an autoimmune response precedes or somehow contributes to the development and progression of malignant tumors," Luborsky said. "We think that antibodies may arise in response to very early abnormal changes in ovarian tissue that may or may not progress to malignancy, depending on additional triggering events. Or, alternatively, antibodies may bind to normal cells in the ovary, causing dysfunction and leading to infertility - and, in a subpopulation of women, to the development of ovarian cancer."

Other researchers involved in the study were Yi Yu, MS, and Seby Edassery, MS, both from Rush, and a group led by Ingegerd Hellstrom, MD, PhD, and Karl Eric Hellstrom, MD, PhD, and including Yuan Yee Yip, BS, Jade Jaffar, BS, and Pu Liu, PhD, from Harborview Medical Center at the University of Washington.

The study was supported by funding from the National Institutes of Health and Fujirebio Diagnostics, Inc.

http://www.eurekalert.org/pub_releases/2011-08/vdio-ctv081611.php

Confirmation that vitamin D acts as a protective agent against the advance of colon cancer

A study conducted by VHIO researchers confirms that a lack of vitamin D increases the aggressiveness of colon cancer

The indication that vitamin D and its derivatives have a protective effect against various types of cancer is not new. In the field of colon cancer, numerous experimental and epidemiological studies show that vitamin D3 (or cholecalciferol) and some of its derivatives inhibit the growth of cancerous cells. Researchers at the Vall d'Hebron Institute of Oncology (VHIO), in collaboration with the Alberto Sols Institute of Biomedical Research (CSIC-UAB), have confirmed the pivotal role of vitamin D, specifically its receptor (VDR), in slowing down the action of a key protein in the carcinogenic transformation process of colon cancer cells. These results are being published in the journal PLoS One.

This protein, known as beta-catenin, which is normally found in intestinal epithelial cells where it facilitates their cohesion, builds up in large quantities in other areas of the cells when the tumour transformation begins. As a result of these changes, the protein is retained in the cell nucleus, where it facilitate the carcinogenic process, and this is the point at which vitamin D intervenes, or rather, the vitamin D receptor (VDR). "Our study has confirmed the pivotal role of the VDR in controlling the anomalous signal that sparks off the growth and uncontrolled proliferation of colon cells which, in the final instance, ends up causing a tumour to emerge", says Héctor Palmer, the coordinator of this study and head of the VHIO's Stem Cells and Cancer laboratory. He continues, "The stimulation of this receptor suppresses the action of the beta-catenin protein, intercepting the series of events that change the intestinal cell into a malignant tumour cell".

The study was conducted on mice and human colon cancer cells. The mice were used as a model to replicate the initial phases of colon cancer. "These findings show that mice of this kind, which also lack the VDR and hence do not respond to vitamin D, present larger and more aggressive tumours than mice with the VDR", explains Dr. Palmer, and concludes: "The number of tumours is not influenced by the absence of VDR, which would indicate that this factor does not protect against the appearance of the tumour but does intervene in its growth phase, reducing its aggressiveness".

The researchers then analysed the effect of the VDR on human colon cancer cell cultures and observed that the concentration of the altered protein, beta-catenin, increased in cells without the VDR. These findings were repeated in the three types of colon cancer cells studied, and confirmed the results observed in the mice.

In two-thirds of advanced colon cancer tumours there was a lack of VDR in the cancer cells, and this circumstance leads us to believe that this loss may contribute to speeding up the growth of the tumour. The findings of this study confirm this supposition.

Vitamin D: essential in the initial phases of colon cancer

In light of these findings, chronic vitamin D deficiency represents a risk factor in the development of more aggressive colon tumours. Patients in the initial stages of colon cancer, the time when the VDR still has a substantial presence in the cells, could benefit from being treated with vitamin D3. However, this would not be useful in the advanced stages of the disease when the presence of the VDR is very much reduced.

The study data support the development of anti-tumour medicines based on the structure of vitamin D, although their use in patients will require further research in the next few years.

The body not only obtains vitamin D from food, especially milk and fish oils, but also manufactures it from exposure to sunlight. Prolonged exposure is not necessary; just 10 minutes in the sun every day when it is not at its peak is sufficient to stimulate its production. During the summer, when we are more likely to sunbathe, it is important to use the appropriate protective measures against sunburn to avoid future sun damage. Use high-factor solar protection products and do not expose the skin to the sun in the middle of the day to protect against skin cancers.

http://www.physorg.com/news/2011-08-spacex-november-flight-space-station.html

SpaceX plans November test flight to space station

California-based rocket maker SpaceX said that it will make a test flight in late November to the International Space Station, now that NASA has retired its space shuttle program.

"SpaceX has been hard at work preparing for our next flight - a mission designed to demonstrate that a privately-developed space transportation system can deliver cargo to and from the International Space Station (ISS)," the company, also called Space Exploration Technologies, said in a statement.

The mission is the second to be carried out by SpaceX, one of a handful of firms competing to make a spaceship to replace the now-defunct US shuttle, which had been used to carry supplies and equipment to the orbiting outpost. "NASA has given us a November 30, 2011 launch date, which should be followed nine days later by Dragon berthing at the ISS," the company said.

It said the arrival of the vessel at the space station would herald "the beginning of a new era in space travel."

"Together, government and the private sector can simultaneously increase the reliability, safety and frequency of space travel, while greatly reducing the costs," SpaceX said.

The company won $75 million in new seed money earlier this year, after it became the first to successfully send its own space capsule, the gumdrop-shaped Dragon, into orbit and back in December 2010.

The shuttle Atlantis completed its final journey to the ISS and back last month, ending the 30-year-old US space shuttle program.

http://well.blogs.nytimes.com/2011/08/15/really-the-claim-to-prevent-migraines-drink-more-water/

Really? The Claim: To Prevent Migraines, Drink More Water

By ANAHAD O'CONNOR

THE FACTS ***For migraine sufferers, summer can be a perilous time of year.***

Oppressive heat and spikes in temperature have long been thought to precipitate attacks in people prone to chronic headaches. One large study in the journal Neurology even showed that the risk of migraines jumps nearly 8 percent for every nine-degree rise in temperature.

But a simple step that may lower the risk, especially in warm weather, is to stay properly hydrated. Dehydration causes blood volume to drop, researchers say, resulting in less blood and oxygen flow to the brain and dilated blood vessels. Some experts suspect that a loss of electrolytes causes nerves in the brain to produce pain signals.

Anyone who has ever woken up dehydrated after a night of heavy drinking knows this feeling as a hangover. But migraine sufferers may be more sensitive to the effects of dehydration.

In one study, also published in Neurology, scientists recruited migraine sufferers and divided them into two groups. Those in the first group were given a placebo medication to take regularly. The others were told to drink 1.5 liters of water, or about six cups, in addition to their usual daily intake. At the end of two weeks, the researchers found that those in the water group had increased their fluid intake by just four cups a day. But on average they experienced 21 fewer hours of pain during the study period than those in the placebo group, and a decrease in the intensity of their headaches.

To stay adequately hydrated, health officials recommend that men drink about 13 cups of liquid a day - from water, juice and other sources - and that women drink about 9 cups.

THE BOTTOM LINE Research suggests that dehydration can increase the risk of migraines.

http://news.discovery.com/animals/eel-living-fossil-110816.html

'Living Fossil' Retains Dinosaur-Era Look

A recently discovered eel living in a remote submarine cave has evolved out of step with the rest of the world.

By Jennifer Viegas | Tue Aug 16, 2011 07:00 PM ET

This newly discovered eel's existence dates back to dates back to 200 million years ago. **Jiro Sakaue**

An eel recently discovered in an underwater cave appears to have evolved out of step with the rest of us, retaining primitive characteristics associated with animals from the Dinosaur Era. The eel, described in the latest Proceedings of the Royal Society B, is what Charles Darwin referred to as a "living fossil." These are extremely long-lasting species that have undergone few bodily changes over the millennia.

In this case, the enigmatic eel dubbed Protoanguilla ("first eel") palau represents a new family, genus and species that dates back to around 200 million years ago.

"The eel looks so bizarre - large head with relatively short body and various unique, internal characters - that no ichthyologist, including us, correctly identified it as a member of true eel at first sight," co-author Masaki Miya told Discovery News. Miya is curator of fishes and an adjunct associate professor at Chiba University's Natural History Museum and Institute.

His colleague, diver Jiro Sakaue, found the eel nearly 115 feet under the surface of the Pacific Ocean in a cave at the western fringing reef of Ngemelis Island, Republic of Palau. Although just 1.7 inches long, the eel's reddish brown body is striking, especially given its iridescent fins tipped in bright white.

Both morphological and molecular analysis place Protoanguilla in a sister lineage independent of other eels. There are more than 800 species of true eels classified into 19 families. The oldest known eel in the fossil record dates to 100 million years ago, and this newly discovered species remarkably has features even more ancient than that. "Those characters assumed to be more primitive than, and equally primitive with, the oldest fossil record actually represent those descended from the Dinosaur Era," Miya said.

Lead author David Johnson explained that these primitive features include fewer vertebrae, certain fused skull bones, presence of an upper jaw bone found in Cretaceous eels, and toothed gill rakers, which could be involved in feeding and gill maintenance.

Johnson, curator of the Division of Fishes at the Smithsonian National Museum of Natural History, added that the eel's "tail fin rays extend back slightly farther than the adjacent fin rays. This is another feature in which Protoanguilla appears to be primitive with respect to living eels."

Co-author Hitoshi Ida shared that the cave home of this eel "is extremely young (110,000 to 10,000 years ago) compared with the evolutionary history of the living fossil eel. I think that what we see is a remnant of their habitat," Ida said. It's possible that this unusual eel may be found in other remote marine habitats, but so far, the small Palau cave is its only known home.

John McCosker, chair of Aquatic Biology at the California Academy of Sciences, told Discovery News that he and other eel experts are "chagrined that such a remarkable eel turned up in an underwater cave in Palau."

"Eels, to most amateur naturalists, aren't even thought of as fish," McCosker said. "They are, in fact, an excellent case of evolution involving the loss of body parts rather than their exaggeration, and in discovering the basal lineage of true eels, the authors have helped to trace the process of eel evolution further back in its ancestry." He added, "The analysis they have performed using morphology and genetics is brilliant and invites as many questions about eel evolution as it solves."

One particularly important question that remains about this eel, like all newfound species, is what now can be done to protect it. The researchers expressed concern, given that its uniqueness and location are now known.

"Professional as well as amateur divers are always curious about animals and those working for the aquarium trade are notorious for trying to keep every animal in their tanks," Miya said. "We should promote a campaign for preserving animals to the Palau government shortly after the (study's) publication."

http://www.eurekalert.org/pub_releases/2011-08/nmh-phs081711.php

Popular herbal supplements may adversely affect chemotherapy treatment

Doctors urge cancer patients to discuss supplements with their doctors before beginning treatment

CHICAGO- Acai berry, cumin, herbal tea, turmeric and long-term use of garlic – all herbal supplements commonly believed to be beneficial to your health – may negatively impact chemotherapy treatment according to a new report presented at the American Society of Clinical Oncology (ASCO) meeting in Chicago this summer. Researchers from Northwestern Memorial hospital say there is growing evidence that these popular supplements may intensify or weaken the effect of chemotherapy drugs and in some cases, may cause a toxic, even lethal reaction.

"With the growth of the Internet, patients have better access to information about alternative products and often turn to dietary and herbal supplements to treat their illness because they think they're natural and safe," said June M. McKoy, MD, geriatrician at Northwestern Memorial Hospital and lead investigator on the ASCO presentation. "What people don't realize is that supplements are more than just vitamins and can counteract medical therapies if not taken appropriately".

McKoy, who is also director of geriatric oncology at the Robert H. Lurie Comprehensive Cancer Center of Northwestern University, says more research is needed to understand which supplements interact with chemotherapy drugs and the extent of those interactions and encourages patients to openly communicate with their physicians about the use of supplements.

"Patients need to tell their doctors what medications they are taking – including vitamins and supplements – to avoid any possible interaction," said McKoy who is also an assistant professor of medicine and preventive medicine at Northwestern University Feinberg School of Medicine.

Herbal supplements, defined as plant or plant parts used for therapeutic purposes, can interact with chemotherapy drugs through different mechanisms. Some herbs can interfere with the metabolism of the drugs, making them less effective while other herbs such as long-term use of garlic may increase the risk of bleeding during surgery. While culinary herbs used in small quantities for flavoring are generally safe, consuming large amounts for prolonged periods of time may have a negative effect on the body when going through chemotherapy.

Recent research shows that 50 percent of patients undergoing chemotherapy did not tell their doctor they were taking alternative therapies. "Some believe it's not important, while others are uncomfortable admitting they are pursuing alternative therapies," said McKoy. "The truth is, integrative approaches can be beneficial for cancer patients, but it's important to take these approaches at the right time and under the supervision of your doctor."

McKoy urges patients to stop taking herbal supplements while receiving chemotherapy until more is known about possible interactions, but encourages those who are interested in complementary approaches to have a conversation with their doctor about other approaches that may be beneficial.

"Integrative therapies such as massage, acupuncture and meditation can address important patient needs by alleviating stress, addressing pain and helping patients cope," said Melinda Ring, MD, medical director for the Northwestern Memorial Physicians Group's Center for Integrative Medicine and Wellness.

No matter the course of treatment, McKoy stresses the importance of physicians and patients being more cognizant of this potential interaction and encourages communication about all herbal supplement intakes. "Patients should bring in labels and bottles to their appointments. This can help the doctor calibrate drug dosage with other supplements in mind in order to prevent toxicities," stated McKoy.

McKoy plans to launch a pilot study this fall to examine how frequently conversations about supplements come up between cancer patients and their doctors. "By identifying communication barriers, we can take steps to improve doctor patient communication in order to prevent potentially dangerous drug interactions," said McKoy.

http://www.eurekalert.org/pub_releases/2011-08/acs-fat081711.php

Fading ability to taste iron raises health concerns for people over age 50

Older people may be at risk for an unhealthy over-exposure to iron

People lose the ability to detect the taste of iron in drinking water with advancing age, raising concern that older people may be at risk for an unhealthy over-exposure to iron, scientists are reporting in results they term "unique." The study appears in the ACS' journal Environmental Science & Technology.

Andrea Dietrich, Susan Mirlohi, and Susan Duncan and colleagues point out that perception of a metallic flavor in water can help people limit exposure to metals such as iron, which occurs naturally in water or from corrosion of iron water-supply pipes. Although commonly referred to as "metallic taste," iron and other metals actually produce both a taste and an odor; this combination is a flavor. People need less iron after age 50. And studies suggest that older people who consume too much - especially in dietary supplements and iron-rich foods - may be at increased risk for Alzheimer's disease and other age-related conditions. Scientists long have known that taste sensory perception fades with age. Dietrich's group set out to fill in gaps in knowledge about how aging affects perception of a metallic flavor in water.

Their results with 69 volunteers aged 19 to 84 years identified a distinctive age-related decline in ability to taste iron. People over age 50 tended to miss the metallic taste of iron in water, even at levels above the thresholds set by the U. S. Environmental Protection Agency and the World Health Association. "Our findings are unique in that drinking water is the source of the environmental sensory contaminant and evidence is provided for wide variation in the human population," the report states. "Whereas our research focused on iron, there are implications for other metals of health concern, most notably copper from copper pipes as our previous research has demonstrated that copper is less flavorful than iron and it is known that copper is also more toxic than iron."

The scientists acknowledge funding from the Institute for Critical Technology and Applied Science at Virginia Tech.

http://www.physorg.com/news/2011-08-microsoft-streaming-storage-patent-os.html

Microsoft 'streaming storage' patent maps OS future

PhysOrg.com - *Microsoft might be planning a future where Windows open to something far bigger, the next time you push your power button on.*

A patent filed by Microsoft points to its plan for an operating system environment beyond Windows 8 that depends on cloud computing, not locally installed software. The patent suggests your computer will be booted through remote storage in a cloud computing construct, where software services control your digital work.

The patent calls for an operating system booted through a chain of storage devices with various priorities forming a centralized environment. The patent says:

“Various aspects of the subject matter described herein are directed towards a technology by which a virtual storage device for a physical or virtual computing machine is maintained as a chain of data structures (e.g., files) including far data maintained at a far (e.g., remote) backing store and near data maintained at a near (e.g., local) backing store (which initially may be empty).”

Reports in ConceivablyTech and Geek.com detail the patent, filed in February 2010, but which are surfacing now. The patent 20110197052 is called “Fast Machine Booting Through Streaming Storage.” Inventors’ names are listed as Dustin Green, Jacob Oshins, and Michael Neil.

One of the advantages is called out as fast booting time. “Described is a technology by which a virtual hard disk is maintained between a far (e.g., remote) backing store and a near (e.g., local) backing store, which among other advantages facilitates fast booting of a machine coupled to the virtual hard disk,” says the patent.

The patent explains that “the virtual disk is available for use immediately, rather than needing to download an entire operating system image before booting from that downloaded image. For example, during a boot operation, only a relatively small amount of data is needed from the boot disk, which is available from the far data and/or the near data.”

News of the patent is seen as proof that rumors and tips over Microsoft’s research project Midori back in 2008 and 2009 were on to something big. Midori, an operating system project at Redmond, was believed to be focusing on the company’s OS future directions and a path to integration with Azure, which is Microsoft’s cloud platform.

The patent’s details and intent will most likely be the subject of conversation in the corridors if not meeting places next month in Anaheim, at the sold-out Microsoft BUILD conference for developers.

More information: Patent: http://appft1.uspt ... /20110197052

http://www.eurekalert.org/pub_releases/2011-08/dlnl-mae081711.php

Moon and Earth may be younger than originally thought

LIVERMORE, Calif. -- *New research using a technique that measures the isotopes of lead and neodymium in lunar crustal rocks shows that the moon and Earth may be millions of years younger than originally thought.*

The common estimate of the moon's age is as old as 4.5 billion years old (roughly the same age as the solar system) as determined by mineralogy and chemical analysis of moon rocks gathered during the Apollo missions. However, Lawrence Livermore National Laboratory scientist Lars Borg and international collaborators have analyzed three isotopic systems, including the elements lead, samarium and neodymium

found in ancient lunar rocks, and determined that the moon could be much younger than originally estimated. In fact, its age may be 4.36 billion years old.

The new research has implications for the age of Earth as well. Common belief is that the moon formed from a giant impact into the Earth and then solidified from an ocean of molten rock (magma).

"If our analysis represents the age of the moon, then the Earth must be fairly young as well," said chemist Borg. "This is in stark contrast to a planet like Mars, which is argued to have formed around 4.53 billion years ago. If the age we report is from one of the first formed lunar rocks, then the moon is about 165 million years younger than Mars and about 200 million years younger than large asteroids."

The isotopic measurements were made by taking samples of ferroan anorthosite (FAN), a type of moon crustal rock, which is considered to represent the oldest lunar crustal rock type. Borg said that these analyses showed that the moon likely solidified significantly later than most previous estimates or that the long-held belief that FANs are flotation cumulates of a primordial magma ocean is incorrect.

Chemical evolution of planetary bodies ranging from asteroids to large rocky planets is thought to begin with differentiation through solidification of magma oceans hundreds of kilometers in depth. The Earth's moon is the typical example of this type of differentiation. However, one interpretation of Borg's findings is that this may not have occurred on the moon.

"The moon is supposed to be old and have a lunar magma ocean, but our new measurements show the moon is young and did not have a magma ocean," Borg said. "The isotopic measurements showed that a specific FAN yields consistent ages from multiple isotopic dating techniques and strongly suggest that the ages record the time at which the rock crystallized," Borg said. "Other studies have not been able to do this."

Other research institutions include the University of Copenhagen, Université Blaise Pascal Laboratoire Magmas et Volcans in France and the Department of Terrestrial Magnetism in Washington D.C.

The research appears in the Aug. 17 online edition of the journal, Nature.

http://www.eurekalert.org/pub_releases/2011-08/plos-pir081711.php

Parasite-infected rodents attracted to cat odor study finds

New research shows how a brain parasite can manipulate rodent fear responses for the parasite's own benefit.

The study, authored by Patrick House and Dr. Robert Sapolsky of Stanford University and released this week in PloS One, addressed how the single-celled parasite Toxoplasma gondii makes infected rodents more likely to spend time near cat odors. The study finds Toxoplasma-infected male rats have altered activation in brain regions involved in fear and increased activation of brain regions involved in sexual attraction after exposure to cat odors. The findings may help explain the biological bases of innate fear and sexual attraction.

Toxoplasma requires the cat digestive system for sexual reproduction. Infected rodents, with reduced fear response to cat odors, are presumably more susceptible to predation by cats, thereby enabling completion of the parasite lifecycle. Toxoplasma is manipulating the fear response specifically to the urine of cats - infected rats behave normally on anxiety, fear, social and memory tasks, and retain fear behavior to non-feline predator odors.

"These findings support the idea that in the rat, Toxoplasma is shifting the emotional salience of the detection of the cat. They also suggest that fear and attraction might lie on the same spectrum, or at least that the emotional processing of fear and attraction are not entirely unrelated," House said.

The study does not advance evidence for how Toxoplasma is altering the brain, only evidence that it does. Previous research showed that Toxoplasma invades the brain of the host and settles near the amygdala, a region involved in a wide range of fear and emotional behaviors. This study extends these findings by showing that not only is Toxoplasma found in the amygdala of infected male rat hosts, but it also changes the way certain subregions of the amygdala respond to cat odor – specifically, by increasing neural activity in the presence of cat odor in regions normally activated by exposure to a female rat.

Up to a third of humans test positive for Toxoplasma, due largely to the consumption of undercooked meat or contact with cat litter. In humans, Toxoplasma exposure is most dangerous to developing fetuses and pregnant women. However, many recent studies find Toxoplasma exposure linked with schizophrenia, a disease noted for amygdala dysfunction and improper emotional response, compelling further investigation into what exactly Toxoplasma is doing in the host brain.

Citation: *House PK, Vyas A, Sapolsky R (2011) Predator Cat Odors Activate Sexual Arousal Pathways in Brains of Toxoplasma gondii Infected Rats. PLoS ONE 6(8): e23277. doi:10.1371/journal.pone.0023277*

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Competing Interests: The authors have declared that no competing interests exist.

http://www.eurekalert.org/pub_releases/2011-08/uok-rpt081711.php

Researchers push to import top anti-bullying program to US schools

The program takes a holistic approach to the bullying problem, including a rigorous classroom curriculum, videos, posters, a computer game and role-play exercises that are designed to make schools inhospitable to bullying

LAWRENCE, Kan. - An interdisciplinary team of researchers at the University of Kansas plan to bring a highly successful anti-bullying effort, the KiVa program, to American schools. Starting as early as the 2012-13 school year, a pilot program could kick off in selected classrooms in Lawrence, Kan. If shown to be successful there, soon afterward the model could expand nationally.

KiVa, implemented in Finland in 2007, has impressed researchers with its proven reduction in bullying incidents. According to one recent study, KiVa "halved the risk of bullying others and of being victimized in one school year."

"Any time you see an intervention reported in the literature, if they work, they barely work," said Todd Little, KU professor of psychology and director of the Center for Research Methods and Data Analysis. "This is one of the first interventions we're seeing with effects that are impressive and pervasive. We here at KU are going to be the sole source for testing KiVa in the U.S."

The program takes a holistic approach to the bullying problem, including a rigorous classroom curriculum, videos, posters, a computer game and role-play exercises that are designed to make schools inhospitable to bullying. When bullying episodes do occur within the school, a small team of trained employees addresses the incident individually with the victim and bully or bullies to ensure bullying is ultimately stopped.

"The KiVa program targets the peer environment, trying to create an ecology where bullying is no longer tolerated," said Anne Williford, assistant professor of social welfare at KU. "Instead of targeting only a bully and victim for intervention, it targets the whole class, including kids who are uninvolved in bullying behavior. KiVa fosters skills to help students take actions, either large or small, to shift the peer ecology toward one that does not support bullying."

The researchers said the program works because it recognizes that bullies sometimes may earn higher social status from their behavior.

"People have traditionally framed bullying as social incompetence, thinking that bullies have low self-esteem or impulse problems," said Patricia Hawley, KU associate professor of developmental psychology. "But recent research shows that bullying perpetrators can be socially competent and can win esteem from their peers."

By changing perceptions of peers who are neither bullies nor victims, the program undercuts a social environment that supports bullying.

"It changes the rewards structure," Hawley said. "At the end of the day, the goals of the bully are like yours and mine - they want friendship and status. They have human goals, not pathological ones. With KiVa, bystanders are set up to win by intervening, and their status can go up. As a bystander, I can achieve goals of friendship and status by standing up to a bully."

In Lawrence schools, the KU researchers hope to compare instances of bullying and victimization in both intervention and control groups to establish the strength of the KiVa program in a U.S. setting.

School district officials welcomed the opportunity.

"We're pleased to have the opportunity to collaborate with Dr. Williford and KU's School of Social Welfare on the KiVa anti-bullying project," said Kim Bodensteiner, Lawrence USD 497's chief academic officer. "Given our experiences using other bullying prevention programs, Lawrence Public Schools' teachers and staff can provide valuable feedback to researchers during the development of KiVa program materials. We look forward to the possibility of participating in a future pilot study."

Results from the pilot program are to be measured by KU's Center for Research Methods and Data Analysis.

http://www.eurekalert.org/pub_releases/2011-08/uog-hpk081111.php

Human pathogen killing corals in the Florida Keys

A research team has identified human sewage as the source of the coral-killing pathogen that causes white pox disease of Caribbean elkhorn coral.

Winter Park, Fl. and Athens, Ga. – A research team from Rollins College in Florida and the University of Georgia has identified human sewage as the source of the coral-killing pathogen that causes white pox disease of Caribbean elkhorn coral. Once the most common coral in the Caribbean, elkhorn coral was listed for protection under the United States Endangered Species Act in 2006, largely due to white pox disease. The team's findings have just been published in the peer-reviewed open access journal PLoS ONE.

White pox disease on a frond of the endangered elkhorn coral on Carysfort Reef in the Florida Keys. White pox disease comes from humans, but when it infects coral, as in this case from the Upper Keys, it causes white blotches by killing the overlying coral tissue and revealing the coral's white limestone skeleton underneath. **James W. Porter, University of Georgia**

Kathryn P. Sutherland, associate professor of biology at Rollins College, and her research collaborators, Associate Professor of Environmental Health Science Erin K. Lipp and Professor of Ecology James W. Porter of the University of Georgia, have known since 2002 that the bacterium that killed coral was the same species as found in humans. "When we identified Serratia marcescens as the cause of white pox, we could only speculate that human waste was the source of the pathogen because the bacterium is also found in the waste of other animals," Sutherland said.

In order to determine a source for the pathogen, the research team collected and analyzed human samples from the wastewater treatment facility in Key West and samples from several other animals, such as Key deer and seagulls. While Serratia marcescens was found in these other animals, genetic analyses showed that only the strain from human sewage matched the strain found in white pox diseased corals on the reef. The final piece of the investigative puzzle was to show that this unique strain was pathogenic to corals.

With funding from Florida's Mote Marine Laboratory "Protect Our Reefs" grant program, Sutherland, Lipp and Porter conducted challenge experiments by inoculating fragments of coral with the strain found in both humans and corals to see if it would cause disease. The experiments were carried out in a laboratory in closed seawater tanks to eliminate any risk of infection to wild populations of corals.

"The strain caused disease in elkhorn coral in five days, so we now have definitive evidence that humans are a source of the pathogen that causes this devastating disease of corals," Sutherland said.

"These bacteria do not come from the ocean, they come from us," said Porter. Water-related activities in the Florida Keys generate more than $3 billion a year for Florida and the local economy. "We are killing the goose that lays the golden egg, and we've got the smoking gun to prove it," Porter said.

Serratia marcescens is also a pathogen of humans, causing respiratory, wound and urinary tract infections, meningitis, and pneumonia. Human diseases caused by this bacterium are most often associated with hospital-acquired infections of newborn infants and immune-compromised adults. This research reveals a new disease pathway, from humans to wildlife, which is the opposite of the traditional wildlife-to-human disease transmission model. The movement of pathogens from wildlife to humans is well documented - for example, bird flu or HIV - but the movement of disease-causing microbes from humans to marine invertebrates has never been shown before. This is the first time that a human disease has been shown to cause population declines of a marine invertebrate.

"Bacteria from humans kill corals - that's the bad news," said Porter. "But the good news is that we can solve this problem with advanced wastewater treatment facilities," like one recently completed in Key West. "This problem is not like hurricanes, which we can't control. We can do something about this one," he said. The entire Florida Keys is in the process of upgrading local wastewater treatment plants, and these measures will eliminate this source of the bacterium.

The Rollins College and University of Georgia collaborative research group is currently funded by a $2.2 million grant from the National Science Foundation to investigate the ecology of white pox disease in the Florida Keys. The five-year study will focus on mechanisms of transmission of the coral pathogen and the factors that drive the emergence and maintenance of white pox outbreaks, including water quality, climate variability and patterns of human population density. "We are concerned that disease incidence or severity may increase with rising temperatures," Lipp said, "reinforcing the importance of protecting near-shore water quality in a changing climate."

Besides Sutherland, Porter and Lipp, the study's co-authors were Sameera Shaban of Rollins College and Jessica L. Joyner of UGA. The article is available online at http://dx.plos.org/10.1371/journal.pone.0023468.

http://www.physorg.com/news/2011-08-harmless-bacteria.html

Researchers modify harmless bacteria to kill harmful bacteria

PhysOrg.com - ***Researchers in Singapore have modified the DNA of one type of bacterium, Escherichia coli, to first sense the presence of another bacterium, Pseudomonas aeruginosa, and then to explode, releasing a special kind of toxin that will kill it.***

Chueh Loo Poh and Matthew Wook Chang of Nanyang Technological University in Singapore, describe their research in Molecular Systems Biology.

P.aeruginosa is a common microbe that is responsible for difficult to treat infections in people, particularly those with comprised immune systems. It generally colonizes the gastrointestinal tract or the respiratory system and is believed to be responsible for up to ten percent of all hospital acquired infections. The general approach to treating it is massive amounts of antibiotics which don't always work and also tend to kill off good bacteria in the process.

To get around this problem Poh and Chang modified the DNA of E. coli in such a way as to allow it to be able to detect LasR, a molecule used by P.aeruginosa bacteria to communicate with one another. When the LasR is detected, the E. coli begins producing a toxin called pyocin until it's full, at which point it explodes releasing the pyocin which kills P.aeruginosa by eating holes in its exterior, allowing its innards to pour out.

This approach is the first time that bacteria have been used to kill other bacteria and is a step up in the ongoing battle against infectious diseases. It's one that is of critical importance due to the dearth of new anti-bacterial drugs; only two new ones have come on the market in the last ten years and the old ones are becoming increasingly ineffective as new strains of bacteria have evolved that are resistant to them.

The research team says that in the lab, the modified E. coli were able to kill up to 99% of the P.aeruginosa when they were in standalone mode. Perhaps more importantly, they were also able to kill off nearly 90% of them when they were banded together in large communities called biofilms, which are notoriously difficult to treat with conventional methods.

The one major obstacle to using such engineered E. coli as a stealth agent, at least at this stage, is its inability to actually hunt for its victim, rather than sit by passively waiting for the right bacterium to pass by before exploding itself. The hope is that other bacteria with sensing abilities could be used instead of E. coli; ones that could actually track down the specific target, perhaps allowing for a kill rate of 100%.

The next step in the testing of the new treatment will be introducing the modified E. coli into mice to see if it will work as well in a live animal, and also of course, to see what side effects might occur.

More information: *Engineering microbes to sense and eradicate Pseudomonas aeruginosa, a human pathogen, Molecular Systems Biology 7 Article number: 521 doi:10.1038/msb.2011.55*

Abstract

Synthetic biology aims to systematically design and construct novel biological systems that address energy, environment, and health issues. Herein, we describe the development of a synthetic genetic system, which comprises quorum sensing, killing, and lysing devices, that enables Escherichia coli to sense and kill a pathogenic Pseudomonas aeruginosa strain through the production and release of pyocin. The sensing, killing, and lysing devices were characterized to elucidate their detection, antimicrobial and pyocin release functionalities, which subsequently aided in the construction of the final system and the verification of its designed behavior. We demonstrated that our engineered E. coli sensed and killed planktonic P. aeruginosa, evidenced by 99% reduction in the viable cells. Moreover, we showed that our engineered E. coli inhibited the formation of P. aeruginosa biofilm by close to 90%, leading to much sparser and thinner biofilm matrices. These results suggest that E. coli carrying our synthetic genetic system may provide a novel synthetic biology-driven antimicrobial strategy that could potentially be applied to fighting P. aeruginosa and other infectious pathogens.

http://www.physorg.com/news/2011-08-human-precursors-sea-team.html

Human precursors went to sea, team says

Early manlike creatures may have been smarter than we think. Recent archaeological finds from the Mediterranean show that human ancestors traveled the high seas.

A team of researchers that included an North Carolina State University geologist found evidence that our ancestors were crossing open water at least 130,000 years ago. That's more than 100,000 years earlier than scientists had previously thought.

Their evidence is based on stone tools from the island of Crete. Because Crete has been an island for eons, any prehistoric people who left tools behind would have had to cross open water to get there.

The tools the team found are so old that they predate the human species, said Thomas Strasser, an archaeologist from Providence College who led the team. Instead of being made by our species, Homo sapiens, the tools were made by our ancestors, Homo erectus.

The tools are very different from any others found on Crete, Strasser said. They're most similar to early stone-age tools from Africa that are about 700,000 years old, he said.

Initially the team didn't have any way to date the tools. That's where NCSU geologist Karl Wegmann came in.

At the time, Wegmann didn't know much about archaeology, but he did know quite a bit about Crete's geology. He had been figuring out the ages of Crete's rock formations to study earthquakes.

A few of the stone tools the team had discovered were embedded in those same rock formations. Those rocks were formed from ancient beach sands, Wegmann said.

Today, the rocks and the tools embedded in them are hundreds of feet above the shore. The same process that drives the region's strong earthquakes - colliding continents - is pushing Crete upward out of the sea at a rate of less than {0 of an inch every year - more than 35 times more slowly than fingernails grow.

The island's slow rise has preserved beaches from many eras as terraces along the coast. The lower terraces are the easiest to date. Scientists can measure the age of seashells embedded in the rock using radioactive carbon dating. This method estimates the age of those terraces at about 45,000 and 50,000 years old.

"We know that (the tools) are tens of meters above the terrace we dated at 50,000 years old, so we know right off the bat that they have to be at least that old," Wegmann said.

But 50,000 years ago is carbon dating's limit. Anything older has to be dated using another method.

Crete's rise from the sea gives a fairly simple way of doing that. Once they know the age of lower terraces, geologists can calculate the age of higher terraces just by measuring the difference in the beaches' elevation.

If geologists know how much farther the older terrace traveled upward from the newer, and they know how fast it was going, they can figure out how long it took to get there. Or, in other words, its age, in this case a record-smashing 130,000 years old.

"The thing to me that really makes this unique and exciting is ... these other sister species maybe weren't entirely stupid like we portray them," Wegmann said. "They were capable of really complex things."

http://www.bbc.co.uk/news/health-14557224

Sniffer dogs detect lung cancer

By James Gallagher Health reporter, BBC News

Sniffer dogs can be used to reliably detect lung cancer, according to researchers in Germany.

Writing in the European Respiratory Journal, they found that trained dogs could detect a tumour in 71% of patients. However, scientists do not know which chemical the dogs are detecting, which is what they say they need to know to develop a screening programme. Cancer Research UK said that was still a "long way" off.

It was first suggested that dogs could "sniff out" cancer in 1989 and further studies have shown that dogs can detect some cancers such as those of the skin, bladder, bowel and breast.

Cancer Scent

It is thought that tumours produce "volatile chemicals" which a dog can detect.

Researchers trained four dogs - two German shepherds, an Australian shepherd and a Labrador - to detect lung cancer. Three groups of patients were tested: 110 healthy people, 60 with lung cancer and 50 with chronic obstructive pulmonary disease, a narrowing of the airways of the lungs. They all breathed into a fleece filled tube, which absorbed any smells. The dogs sniffed the tubes and sat down in front of those in which they detected lung cancer smells.

They were successful 71% of the time. The researchers showed the dogs were not getting confused by chemicals associated with chronic obstructive pulmonary disease or smoking.

Dr Thorsten Walles, the report's author from Schillerhoehe Hospital, said: "In the breath of patients with lung cancer, there are likely to be different chemicals to normal breath samples and the dogs' keen sense of smell can detect this difference at an early stage of the disease. "Our results confirm the presence of a stable marker for lung cancer. This is a big step forward."

Dogs are unlikely to become regular fixtures in doctors surgeries so researchers are working on "electronic noses" which would be able to detect the same chemical as the dog. This chemical or combination of smells has not yet been found. As the researchers lament: "Unfortunately, dogs cannot communicate the biochemistry of the scent of cancer."

Dr Laura McCallum, science information officer at Cancer Research UK, said: "Although there are now some intriguing studies suggesting that dogs may be able to smell cancer in some situations, we're still a long way from understanding exactly which 'smelly molecules' they are detecting and if these studies are accurate.

"Because it would be extremely difficult to use dogs in the clinic, further research is being carried out to learn more about these molecules that are released from tumours and whether devices such as 'electronic noses' could help sniff them out."

http://www.eurekalert.org/pub_releases/2011-08/jhmi-sfc081611.php

Study finds coronary calcium beats C-reactive protein for predicting heart attack and stroke risk

The presence of calcium in coronary arteries is a much better predictor of heart attack and stroke than C-reactive protein among people with normal levels of LDL cholesterol, according to a study of more than 2,000 people led by a Johns Hopkins heart specialist.

Results of the study, published in the August 19, 2011 issue of The Lancet, have important implications for deciding whether cholesterol-lowering statin medication should be prescribed for people who have heart

disease risk factors but normal levels of LDL, the so-called "bad" cholesterol. An estimated 6 million American adults fall into that gray-zone category.

The goal of the new study, which followed 2,083 people for six years, was to further refine who was at higher risk and, therefore, might benefit from taking statin medications. Conversely, the study also looked to define which groups may be at low risk and not in need of the drugs. The participants in the study were volunteers in the ongoing Multi-Ethnic Study on Atherosclerosis, known as MESA, which is an NIH-funded Hopkins-affiliated study.

"This was a direct comparison to see which patients with a normal LDL level of less than 130 mg/dL would have the greater risk of having a heart attack or stroke—those with evidence of calcium in coronary arteries, as determined on a cardiac CT test, or those with high levels of C-reactive protein, which is measured in blood and is an indicator of inflammation somewhere in the body," says Michael J. Blaha, M.D. M.P.H, a cardiology fellow at the Johns Hopkins University School of Medicine and the Johns Hopkins Heart and Vascular Institute, who is the lead author of the study.

Blaha and colleagues found that 95 percent of the heart attacks, strokes or heart-related deaths in the study population occurred in people with some measurable calcium in their heart arteries. Meanwhile, 13.4 percent of those with the highest levels of coronary calcium (with scores greater than 100 on a calcium scoring test) had a heart attack or stroke during the study, whereas only 2 percent of those with high C-reactive protein in their blood, but no calcium buildup, had a heart attack or stroke.

In their study, the researchers determined that high levels of C-reactive protein in the blood, a score at or above 2 milligrams per liter, offered little predictive value after accounting for such risk factors as age, gender, ethnicity, hypertension, obesity, diabetes, smoking and a family history of heart disease.
"A calcium test directly looks for the disease we propose to treat with statins. Without measurable amounts of calcium, which indicates atherosclerosis, you are likely to be at very low risk in the short-term," explains Blaha.

This new study was designed to address some unanswered questions from a 2008 study called JUPITER, short for the Justification for the Use of Statins in Primary Prevention: An Interventional Tool Evaluating Rosuvastatin. That study found a 46 percent reduction in heart attacks among people with normal LDL cholesterol and a high level of C-reactive protein who took the statin medication rosuvastatin, which is marketed as Crestor.

JUPITER only included people with high C-reactive protein and none of those participants were tested to see whether they had evidence of calcium in their coronary arteries. So, Blaha says, it could not be determined from JUPITER whether people with low levels of C-reactive protein would benefit in the same way from statin therapy, or how the presence of coronary calcium may have affected the results. All of the participants in the MESA trial had undergone coronary CT scanning, known as a calcium scoring test. Blaha and colleagues identified a group of participants in MESA who had high C-reactive protein levels and fit the criteria for JUPITER. The researchers also selected a group from MESA who had low levels of C-reactive protein. Then they were able to directly compare the prognostic importance of coronary artery calcium to C-reactive protein.

A statistical comparison of the results showed that among those with no measurable coronary calcium, it would be necessary to treat 549 patients with statin medication in order to prevent one heart attack. However, for those with high levels of coronary calcium buildup (with a calcium score greater than 100), the predicted number needed to treat to prevent one heart attack was only 24.

"Statin medications, which are a lifelong therapy, should not be considered the same as other preventive measures, such as diet and exercise, to reduce the risk of cardiovascular disease," says Roger Blumenthal, M.D., a cardiologist, professor of Medicine and director of the Ciccarone Center for the Prevention of Heart Disease at Johns Hopkins. He also was a co-investigator on the new study. "All drugs have the potential to cause side effects in some people, although with statins, the side effects are rare," he adds.

According to Blumenthal, "Many patients fall into the gray zone of being healthy with normal LDL cholesterol, but also having some risk factors, including being overweight, having elevated blood sugar levels or a family history of heart disease. Our study provides clear evidence that high levels of calcium in coronary arteries will increase the risk of a heart attack or a stroke. And the risk increases with the amount of calcium, whether or not patients have high levels of C-reactive protein."

"While not everyone needs a calcium scoring test," Blaha says, "we believe looking for calcification in coronary vessels in certain patients makes sense in order to predict who may benefit from statin therapy because the test gets right to the heart of the disease we want to treat."

"Our data support recent American Heart Association guidelines, which say it is reasonable to order a coronary calcium scan for adults who are considered to be at intermediate risk of a heart attack over the next 10

years. A high coronary calcium score would indicate that statin therapy would likely be a useful strategy to lower that person's cardiovascular risk," according to Blumenthal.

In addition to Blaha and Blumenthal, other Johns Hopkins investigators involved in this study were study senior investigator Khurram Nasir, M.D., M.P.H., who is now at Yale University School of Medicine in New Haven, Conn.; Andrew DeFilippis, M.D., M.Sc.; and João Lima, M.D. Other researchers were Matthew Budoff, M.D., at the Los Angeles Biomedical Research Institute at Harbor-UCLA Medical Center in Torrance, Calif.; Juan Rivera, M.D., M.P.H., and Arthur Agatston, M.D., both at the University of Miami; Ron Blankstein, M.D., at Brigham and Women's Hospital in Boston; and Dan O'Leary at Carney Hospital in Dorchester, Mass. ***For more information:***

http://www.hopkinsmedicine.org/heart_vascular_institute/clinical_services/centers_excellence/ciccarone_center.html

http://www.eurekalert.org/pub_releases/2011-08/uoc--two081511.php

3 waves of evolutionary innovation shaped diversity of vertebrates

Analysis of genomes finds 3 periods of innovation in gene regulation occurred during the evolution of vertebrate animals

SANTA CRUZ, CA - Over the past 530 million years, the vertebrate lineage branched out from a primitive jawless fish wriggling through Cambrian seas to encompass all the diverse forms of fish, birds, reptiles, amphibians, and mammals. Now researchers combing through the DNA sequences of vertebrate genomes have identified three distinct periods of evolutionary innovation that accompanied this remarkable diversification.

The study, led by scientists at the University of California, Santa Cruz, and published this week in Science, focused on regulatory elements that orchestrate the activity of genes. They found three broad categories of evolutionary innovations in gene regulation that increased in frequency during different periods in vertebrate evolution. The first period, for example, was dominated by regulatory innovations affecting genes involved in embryonic development. These changes occurred during the period leading up to about 300 million years ago, when mammals split off from birds and reptiles.

"So many new body plans evolved during this time, it makes sense that the strongest signal in our analysis is for changes affecting genes involved in the development of the body plan and the complex regulation of other genes," said David Haussler, a distinguished professor of biomolecular engineering in the Baskin School of Engineering at UC Santa Cruz and corresponding author of the paper. First author Craig Lowe worked on the study as a graduate student in Haussler's group at UCSC and is now a postdoctoral researcher at Stanford University.

Many previous studies have shown that important evolutionary changes in animals have resulted from the gain, loss, or modification of gene regulatory elements, rather than from the evolution of new protein-coding genes. "Most of the changes that have happened during vertebrate evolution, as animals acquired new body plans and features like feathers and hair, were not the result of new genes but of new regulatory elements that turn genes on and off in different patterns," Haussler said.

The new study identified millions of these regulatory innovations by using computational methods to look for DNA sequences that are still the same in species that have evolved separately over long periods of time. These sequences have presumably been conserved by natural selection because they serve an important function, so most mutations that change them would be harmful to the organism. Conserved sequences outside of known genes are likely to be gene regulatory elements. By comparing the genomes of species whose evolutionary lineages diverged at different times in the past, researchers can see when in evolutionary history a particular conserved sequence first appeared.

"These new regulatory elements are evolutionary innovations that have been passed on to all the descendants of the species in which they first arose," Haussler said. "We document millions of these events. We're not sure every one is rock solid, but we have so many that the statistical patterns are unequivocal--these trends must reflect the evolutionary changes that occurred."

The results reinforce the importance of gene regulation as a mechanism through which evolution occurs on the molecular level, he said. The findings also provide the first indication of distinct phases in vertebrate molecular evolution, with changes in different types of biological processes dominating during different periods of evolutionary history.

Because regulatory elements are typically located near the genes they govern, the researchers assigned each conserved element to the closest gene. They classified the genes into broad categories, such as developmental genes or genes involved in communication between cells, using information on gene functions available through the UCSC Genome Browser.

In the first period of evolutionary innovation, in addition to changes affecting developmental genes, the study found a dramatic enrichment in conserved elements near genes for proteins known as "transcription factors," which bind to DNA and regulate whole groups of other genes. New regulatory elements affecting

transcription factors peaked in our early vertebrate ancestors 500 million years ago, then declined steadily to background levels by the time mammals evolved.

The next trend affected genes involved in cell-to-cell communication, such as genes for "receptor" proteins that sit in the cell membrane and receive signals from other cells. The increase in regulatory innovations near these genes occurred from about 300 million years ago to 100 million years ago and happened independently in the lineages of both fish and animals with "amniotic" eggs (birds, reptiles, and mammals).

A third trend showed up in placental mammals during the past 100 million years, when there was a rise in regulatory innovations for genes involved in signaling pathways within cells. These changes tweaked the complex cross-talk between molecules that coordinates all cellular activities.

Finally, the researchers took a close look at the well-studied set of genes associated with the development of body hair, a trait shared by all mammals. Several hundred genes are known to be involved in hair formation. "These genes have been around a long time, but if we look at the period about 250 million years ago when hair evolved in the predecessors of mammals, we see a bump in regulatory innovations near those genes," Haussler said. "It's not a stunning surprise, but it's a way of validating the method we used to measure regulatory innovation."

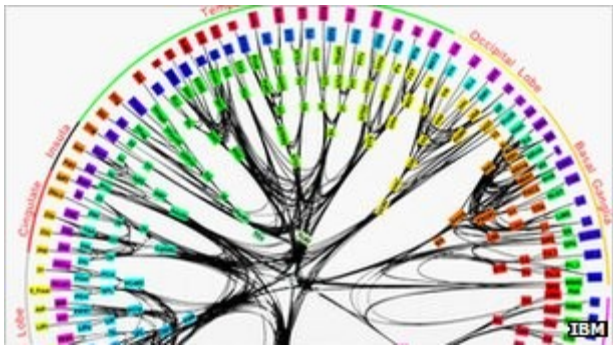
This method can be used to look for other evolutionary trends in particular lineages, especially as scientists sequence the genomes of more animals. Haussler, a Howard Hughes Medical Institute investigator and director of the Center for Biomolecular Science and Engineering at UCSC, is a cofounder of the Genome 10K Project, which aims to get genome sequences for 10,000 vertebrate species. With such a large number vertebrate genome sequences available for analysis, researchers will be in a position to discover the molecular basis for the evolutionary diversification of virtually all of the large animal species.

In addition to Haussler and Lowe, the coauthors of the Science paper include UCSC researchers Brian Raney and Sofie Salama; Manolis Kellis, Michele Clamp, and Kerstin Lindblad-Toh of the Broad Institute of MIT and Harvard; Adam Siepel of Cornell University, a former UCSC graduate student; and David Kingsley of Stanford University. This research was funded by the Howard Hughes Medical Institute, Sloan Foundation, and European Science Foundation.

http://www.bbc.co.uk/news/technology-14574747

IBM produces first 'brain chips'

IBM has developed a microprocessor which it claims comes closer than ever to replicating the human brain.



The system is capable of "rewiring" its connections as it encounters new information, similar to the way biological synapses work. Researchers believe that by replicating that feature, the technology could start to learn. Cognitive computers may eventually be used for understanding human behaviour as well as environmental monitoring.

Dharmendra Modha, IBM's project leader, explained that they were trying to recreate aspects of the mind such as emotion, perception, sensation and cognition by "reverse engineering the brain."

The SyNAPSE system uses two prototype "neurosynaptic computing chips". Both have 256 computational cores, which the scientists described as the electronic equivalent of neurons.

One chip has 262,144 programmable synapses, while the other contains 65,536 learning synapses.

Brain map graph IBM's processors replicate the system of synaptic connections found in the human brain

Man machine

In humans and animals, synaptic connections between brain cells physically connect themselves depending on our experience of the world. The process of learning is essentially the forming and strengthening of connections. A machine cannot solder and de-solder its electrical tracks. However, it can simulate such a system by "turning up the volume" on important input signals, and paying less attention to others.

IBM has not released exact details of how its SyNAPSE processor works, but Dr Richard Cooper, a reader in cognitive science at Birkbeck, University of London said that it likely replicated physical connections using a "virtual machine". Instead of stronger and weaker links, such a system would simply remember how much "attention" to pay to each signal and alter that depending on new experiences.

"Part of the trick is the learning algorithm - how should you turn those volumes up and down," said Dr Cooper. "There's a a whole bunch of tasks that can be done just with a relatively simple system like that such as associative memory. When we see a cat we might think of a mouse."

Some future-gazers in the cognitive computing world have speculated that the technology will reach a tipping point where machine consciousness is possible.

However, Dr Mark Bishop, professor of cognitive computing at Goldsmiths, was more cautious. "[I] understand cognition to be something over and above a process simulated by the execution of mere computations, [and] see such claims as verging on the magical," he said.

IBM's work on the SyNAPSE project continues and the company, along with its academic partners, has just been awarded $21m (£12.7m) by the US Defense Advanced Research Projects Agency (DARPA).

http://www.eurekalert.org/pub_releases/2011-08/uob-dm081911.php

Deadly medication?

Bonn scientists shed light on the dark secret of Queen Hatshepsut's flacon

The corpus delicti is a plain flacon from among the possessions of Pharaoh Hatshepsut, who lived around 1450 B.C., which is on exhibit in the permanent collection of the Egyptian Museum of the University of Bonn. For three and a half millennia, the vessel may have held a deadly secret. This is what the Head of the collection, Michael Höveler-Müller and Dr. Helmut Wiedenfeld from the university's Pharmacology Institute just discovered. After two years of research it is now clear that the flacon did not hold a perfume; instead, it was a kind of skin care lotion or even medication for a monarch suffering from eczema. In addition, the pharmacologists found a strongly carcinogenic substance. Was Hatshepsut killed by her medicine?

When Michael Höveler-Müller became the curator of the Egyptian Museum of the University of Bonn in 2009, it occurred to him to examine the interior of the vessel that, according to an inscription, belonged to Pharaoh Hatshepsut. Its neck had been blocked with what was generally considered "dirt," but Höveler-Müller suspected that it might also be the original clay stopper. So possibly, some of the original contents might still be inside. In Dr. Helmut Wiedenfeld from the Pharmacy Institute, he found just the right partner, to get to the bottom of this question and of the flacon.

At the Radiology Clinic of the Bonn Universitätsklinikum, the flacon was subjected to a CAT scan. Here, the Egyptologist's suspicion was confirmed – not only was the closure intact, but the vessel also held residue of a dried-up liquid. In the summer of 2009, Professor Dr. Friedrich Bootz from the Klinik und Poliklinik für Hals-, Nasen- und Ohrenheilkunde (laryngology, rhinology and otology) of the University of Bonn took samples, using an endoscope.

Too greasy for perfume

This allowed Dr. Wiedenfeld and his team to analyze the old substances for their ingredients. And it became obvious very quickly that what they had found was not dried-up perfume. The mix contained large amounts of palm oil and nutmeg apple oil. "I didn't think anybody would put so much grease on her face," said Dr. Wiedenfeld. "That would make her look as greasy as a plate of ribs." Two additional components clued the pharmacologist in to the actual purpose of the mix, "We found a lot of unsaturated fatty acids that provide relief for people with skin diseases." And this is where the Egyptologist was able to add another piece of the puzzle, "It is indeed known that there were cases of skin disease in Hatshepsut's family." Inflammatory skin diseases such as psoriasis have a largely genetic component.

And the third group of ingredients also points to the fact that this substance was not about providing a nice fragrance, but instead, for fighting a big itch – the pharmacologists found a lot of hydrocarbons derived from creosote and asphalt. To this day, creams containing creosote are used to treat chronic skin diseases. Due to the potentially carcinogenic effects of some of its ingredients, creosote has meanwhile been banned from cosmetics completely, and medications containing creosote are now prescription-only.

What the pharmacologists detected in Hatshepsut's little bottle was in particular benzo(a)pyrene, a hazardous aromatic hydrocarbon consisting of several carbon rings. "Benzo(a)pyrene is one of the most dangerous carcinogenic substances we know," explained Dr. Wiedenfeld. For example, the risk of contracting lung cancer from cigarette smoke results essentially from this substance.

Did the lotion cause the Pharaoh's death from cancer?

Did Hatshepsut maybe poison herself without knowing it? "There is a lot that speaks for this hypothesis," Dr. Wiedenfeld said. "If you imagine that the Queen had a chronic skin disease and that she found short-term improvement from the salve, she may have exposed herself to a great risk over the years." The Egyptologist also thinks that this is very likely. "We have known for a long time that Hatshepsut had cancer and maybe even died from it," said Michael Höveler-Müller. "We may now know the actual cause."

But at this point, the Bonn scientists can only surmise how Hatshepsut obtained her lotion. "Egyptian physicians were general practitioners and good surgeons, but they were lousy internists," explained Dr. Wiedenfeld. "It is quite possible that they owe their knowledge of certain medications to their contacts with Persia and India where the healing arts were very advanced even in Antiquity."

http://www.guardian.co.uk/world/2011/aug/17/black-death-rats-off-hook

Black Death study lets rats off the hook

Plague of 1348-49 spread so fast in London the carriers had to be humans not black rats, says archaeologist

Maev Kennedy guardian.co.uk, Wednesday 17 August 2011 19.37 BST

Rats weren't the carriers of the plague after all. A study by an archaeologist looking at the ravages of the Black Death in London, in late 1348 and 1349, has exonerated the most famous animal villains in history.

"The evidence just isn't there to support it," said Barney Sloane, author of The Black Death in London. "We ought to be finding great heaps of dead rats in all the waterfront sites but they just aren't there. And all the evidence I've looked at suggests the plague spread too fast for the traditional explanation of transmission by rats and fleas. It has to be person to person – there just isn't time for the rats to be spreading it."

He added: "It was certainly the Black Death but it is by no means certain what that disease was, whether in fact it was bubonic plague."

Sloane, who was previously a field archaeologist with the Museum of London, working on many medieval sites, is now attached to English Heritage. He has concluded that the spread of the 1348-49 plague, the worst to hit the capital, was far faster, with an impact far worse than had been estimated previously.

While some suggest that half the city's population of 60,000 died, he believes it could have been as high as two-thirds. Years later, in 1357, merchants were trying to get their tax bill cut on the grounds that a third of all property in the city was lying empty.

Sloane spent nearly 10 years researching his book, poring over records and excavation reports. Many records have gone missing, while there was also a documentation shortfall as disaster overwhelmed the city. Names of those buried in three emergency cemeteries seem not to have been recorded.

However, Sloane found a valuable resource in records from the Court of Hustings, of wills made and then enacted during the plague years. As the disease gripped – in October 1348 rather than the late summer others suggested, reaching its height in April 1349 – the numbers of wills soared as panic-striken wealthy citizens realised their deaths were probably imminent.

On 5 February 1349 Johanna Ely, her husband already dead, arranged provision for her children, Richard and Johanna. She left them property, spelled out which beds and even pots and pans each was to receive, and placed them in the guardianship of her own mother. She was dead within 72 hours.

It appeared to the citizens that everyone in the world might die. Richard de Shordych left goods and money to his son Benedict when he died in early March: his son outlived him by a fortnight.

Money, youth, and formerly robust good health were no protection. Edward III's own daughter, Joan, sailed for Spain with her trousseau, her dowry and her bridesmaids, to marry Pedro, heir to the throne of Castile. She would never see her wedding day as she died of the plague within 10 days of landing.

John of Reading, a monk in Westminster, left one of the few witness accounts. He described deaths happening so fast there was "death without sorrow, marriage without affection, self-imposed penance, want without poverty, and flight without escape".

In Rochester, William of Dene wrote that nobody could be found to bury the dead, "but men and women carried the bodies of their own little ones to church on their shoulders and threw them into mass graves from which arose such a stink that it was barely possible for anyone to go past a churchyard".

Sloane estimates that people living near the cemetery at Aldersgate, which is now buried under Charterhouse Square, in Smithfield, would have seen a corpse carried past every five minutes at the height of the plague.

As many wills were being made in a week as in a normal year. Usually these would only be activated months or years later: in the worst weeks of the plague there was barely time to get them written down. Many, like Johanna Ely, probably made their wills when they felt the first dreaded sweats and cramps of the disease. Others left property and the care of their children to people who then barely outlived them.

The archaeology of the plague also reveals that most people, however, were buried with touching care, neatly laid out in rows, heads facing west, with far more bodies put in coffins than in most medieval cemeteries – but possibly through fear of infection.

Only a few jumbled skeletons hint at burials carried out some time after death and decomposition; those cases probably arose because bodies were found later on in buildings where every member of the household had died.

Sloane believes there was little difference in mortality rates between rich and poor, because they lived so closely packed together. The plague, he is convinced, spread from person to person in the crowded city.

Mortality continued to rise throughout the bitterly cold winter, when fleas could not have survived, and there is no evidence of enough rats.

Black rat skeletons have been found at 14th-century sites, but not in high enough numbers to make them the plague carriers, he said.

In sites beside the Thames, where most of the city's rubbish was dumped and rats should have swarmed, and where the sodden ground preserves organic remains excellently, few black rats have been found.

Sloane wants to dig up Charterhouse, where he believes 20,000 bodies lie under the ancient alms houses and modern buildings, including the Art Deco block where the fictional character Hercule Poirot lives in the television series. And, if anyone finds a mass medieval rat grave, he would very much like to know.

http://www.newscientist.com/blogs/shortsharpscience/2011/08/george-washington-biowarrior.html

British used bioweapon in US war of independence

Debora MacKenzie, consultant

A document has just gone on display; an order signed by Washington himself to send troops that had not been vaccinated for smallpox - or survived it - to Philadelphia to be vaccinated.

A document has just gone on display at Mount Vernon, Virginia - the museum in the former home of George Washington, first US President. It is an order dated 1777 and signed by Washington himself to send troops that had not been vaccinated for smallpox - or survived it - to Philadelphia to be vaccinated. These troops were then to join up with the main army, where the disease was raging.

It sounds like amazing foresight for its day. "Washington's careful handling of the smallpox epidemic at the beginning of the war was a significant reason for the disease not decimating his army", says Mount Vernon.

Not quite. Washington's order was likely a response, not just to a normal smallpox epidemic, but to a bioweapon wielded by the British enemy - a strategy that the redcoats had already used against the colonists to great effect earlier in the American revolutionary war.

Historically, disease was always the real enemy of armies - the First World War was the first in which enemy action killed more soldiers than disease did. In 1776, more than half of all people caught smallpox at some point, and a third of those died. Edward Jenner did not popularise the use of the related, milder "cowpox" virus for "vaccination" until 1798.

But pre-Jenner, smallpox itself was used to immunise - a practice called variolation widespread in the American colonies at the time of the revolution. That was what Washington sent his troops to Philly to get. He later set up special clinics to inoculate all new recruits.

While people were variolated in ways that reduced the severity of the infection - only 1 or 2 per cent died - if you caught smallpox from someone still experiencing this mild disease you often got full-blown smallpox. That made people who were recently-variolated a threat to anyone without immunity to smallpox.

Washington's army was largely composed of rural conscripts who were far less likely than city-dwellers to have been exposed to smallpox. And without being safely variolated, they were sitting ducks, not just to normal smallpox, but to other people who had recently been variolated themselves.

That's certainly what the wily British were counting on. In his admirable history of smallpox, biodefence expert Jonathan Tucker (tragically found dead recently at the age of 56) confirms that British troops in North America indeed deliberately spread smallpox to control restive Indians in the 1760s. By 1775, the British defending Boston from the rebels had already inoculated all their troops. When smallpox broke out in the town, they sent recently-variolated civilians among the besieging colonists, causing an outbreak that delayed the eventual American victory.

In 1776, the British did it again, as the Americans besieged Quebec City - the story goes that this time they variolated prostitutes and sent them among the troops. Half the 10,000 Americans fell ill, and after burying their dead in mass graves, Tucker reports, they retreated in disorder from the colony, which remained in British hands (as did their commander, Benedict Arnold, who infamously turned coat). I'm Canadian - I may well owe that to this spot of biowarfare.

So in 1777, Washington was hardly acting before time in getting his troops inoculated for smallpox. The irony, of course, is that now, 30 years after smallpox was eradicated in the wild, fears that someone will use remaining stocks of the virus as a weapon mean the US still vaccinates its troops, and others.

Because of Jenner it's now a safer procedure, though not risk-free. And unlike in 1777, risks from the vaccine now top the risk you'll get smallpox - unless some would-be bioterrorist really does have plans to emulate the British in 1776. If so, please take note: that time, the British lost.

http://www.bbc.co.uk/news/science-environment-14592547

Map tracks Antarctica on the move

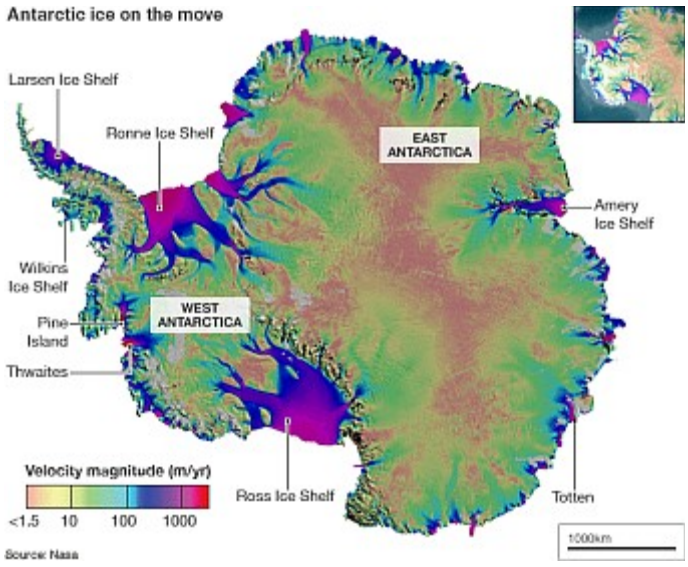
Jonathan Amos By Jonathan Amos Science correspondent, BBC News

Scientists have produced what they say is the first complete map of how the ice moves across Antarctica.

Built from images acquired by radar satellites, the visualisation details all the great glaciers and the smaller ice streams that feed them. The map has been published online by Science magazine. It should aid the understanding of how the White Continent might evolve in the warmer world being forecast by climatologists.

"This is like seeing a map of all the oceans' currents for the first time. It's a game changer for glaciology," said lead author Dr Eric Rignot. "We are seeing amazing flows from the heart of the continent that had never been described before," added the US space agency (Nasa) and University of California (UC), Irvine, researcher.

The map incorporates billions of radar data points collected between 1996 and 2009 by satellites belonging to Europe, Canada and Japan. It closes previous data omissions, especially in the east of the continent.



Click for animation

"We designed acquisition plans, switching on and off the satellites, in all the right desired geographic locations so we could fill the gaps we didn't have data in before," explained Dr Mark Drinkwater from the European Space Agency. "That was a mammoth effort," he told BBC News.

Dr Drinkwater praised in particular the contribution of Canadian company MacDonald, Dettwiler and Associates, which rolled its Radarsat-2 spacecraft every time it flew near the pole to get a view of the ice surface at the highest latitudes. "That was the only way we could fill the so-called 'data hole' that satellites traditionally don't see," he explained.

Ice movement is detected using a technique called Satellite Radar Interferometry (InSAR), which compares images from repeat passes over the same location. InSAR will pick up even subtle deformations in the ice sheet resulting from slow creep. That said, some areas of Antarctica are moving very fast.

Ice velocities on the new map range from just few cm/year near places where the ice divides into different paths, to km/year on fast-moving glaciers and the ice shelves that float out from the edges of the continent.

The sprinters are Pine Island and Thwaites glaciers in West Antarctica. This region, say the authors, is also the part of the continent "experiencing most rapid change at present, over the widest area, and with the greatest impact on total ice sheet mass balance". Recent survey work has revealed that Pine Island, for example, is thinning rapidly; its surface has been dropping by more than 15m per year.

Other fast-moving streams include the Larsen B glaciers on the Antarctic Peninsula. These glaciers experienced an eightfold increase in speed when the floating ice shelf that bounded them collapsed in 2002.

Although the broad picture of how the ice drains from the centre of Antarctica to the edges has been reasonably well characterised for some time, the map throws up a number of previously unrecognised features. These include a new ridge that splits the 14 million square km landmass from east to west.

The map will be useful in monitoring change over time, by comparing it to past and future measurements.

It should also assist the calibration of the computer models that are used to forecast how the ice sheet will react to changes in the climate and the surrounding ocean. The models will need to reproduce the sort of behaviour seen in the map before scientists can have confidence in their ability to predict the future.

One aspect they need to simulate better is the length of some of the ice streams, which stretch much deeper into the interior of Antarctica than many people had acknowledged.

The map work was completed as part of the 2007-8 International Polar Year (IPY), a concerted programme of research to investigate Earth's far north and south. As part of that initiative, a lot of effort was also put into mapping the rock bed of Antarctica. Understanding conditions at the sheet's base, which can slide on liquid water, is a key part of the equation that describes how the ice mass above will move.

http://www.sciencenews.org/view/generic/id/333389/title/Financial_world_dominated_by_a_few_deep_pockets

Financial world dominated by a few deep pockets

Economic "superentity" controls more than one-third of global wealth

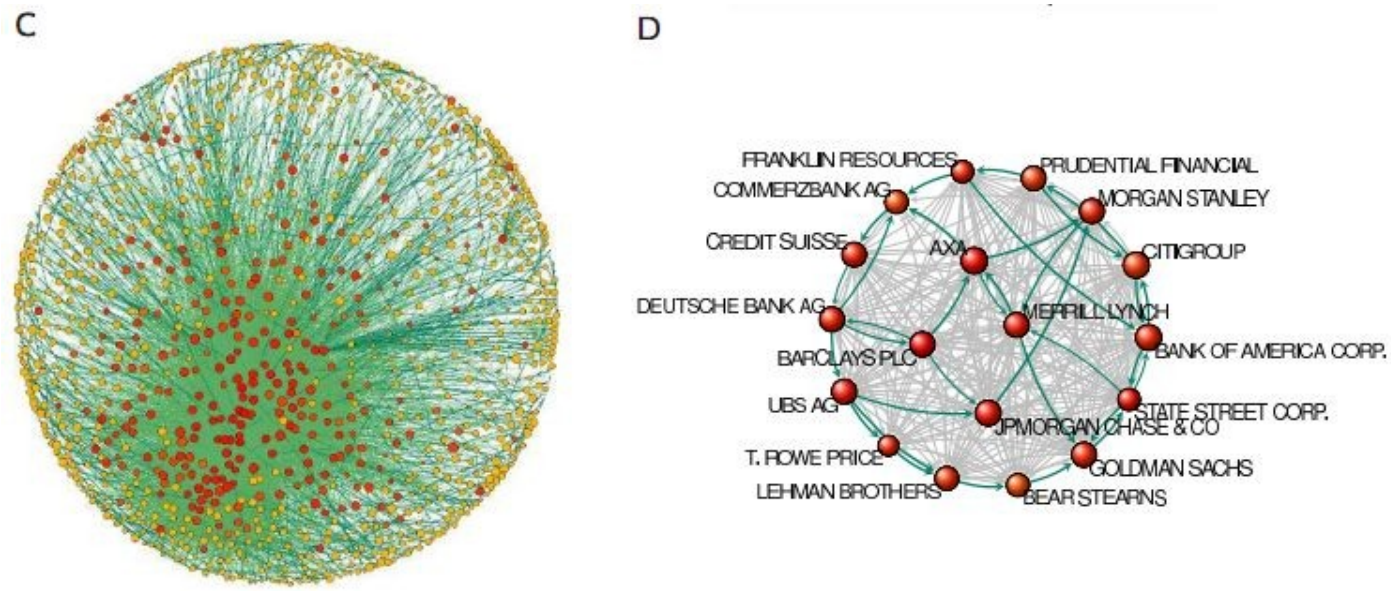
By Rachel Ehrenberg

Conventional wisdom says a few sticky, fat fingers control a disproportionate slice of the world economy's pie. A new analysis suggests that the conventional wisdom is right on the money.

Diagramming the relationships between more than 43,000 corporations reveals a tightly connected core of top economic actors. In 2007, a mere 147 companies controlled nearly 40 percent of the monetary value of all transnational corporations, researchers report in a paper published online July 28 at arXiv.org.

"This is empirical evidence of what's been understood anecdotally for years," says information theorist Brandy Aven of the Tepper School of Business at Carnegie Mellon in Pittsburgh.

The analysis is a first effort to document the international web of relationships among companies and to examine who owns shares - and how many - in whom. Tapping into the financial information database Orbis, scientists from ETH Zurich in Switzerland examined transnational companies, which they defined as having at least 10 percent of their holdings in more than one country. Then the team looked at upstream and downstream connections, yielding a network of 600,508 economic actors connected through more than a million ownership ties.



POWER BALL ***A central core of extremely powerful actors (red dots) dominates international corporate finance, a new mathematical analysis finds.Vitali et al, 2011***

This network takes on a bowtie shape, with a large number of diffuse actors in the wings and a few major players tangled up in the tie's knot. So while it's true that ownership of publicly held corporations is broadly distributed, says complex systems scientist James Glattfelder, a coauthor of the new work, "take a step back and it's all flowing into the same few hands." While any man on the street may have predicted this outcome, the economic literature portrays markets as so dynamic that they lack hot spots of control, Glattfelder says.

Researchers aren't sure what to make of the core's interconnectedness. On the one hand, it could expose the whole network to risk. "Imagine a disease spreading," says Aven. "If you have a high school where everyone's sleeping together and one person gets syphilis, then everyone gets syphilis." But on the flip side, she notes, interconnectedness can lead to better self-policing and positive behaviors, such as fair labor practices or environmentally friendly policies.

And even though the status of many players in the analysis has changed drastically since 2007 (now-defunct Lehman Brothers is a key element of the core), the analysis shows that ownership is becoming increasingly concentrated and increasingly transnational, says Gerald Davis of the University of Michigan in Ann Arbor.

Because interpreting and analyzing these kinds of data is difficult, he says, the analysis serves more as "an impression of the moon's surface you get with a telescope. It's not a street map."

Ownership can be difficult to study internationally because holding shares in a mutual fund doesn't necessarily mean the same thing in the U.S. as it does in communist China. And even within a single country ownership can be hard to tease out, says economist Matthew Jackson of Stanford University. For example, when an individual invests in a mutual fund or even purchases shares through an institution like Merrill Lynch,

the firm is often still the official owner of the assets. And even when shareholders do have voting rights, they may not exercise them.

"This becomes worrisome if everyone is like me and says I'll let Vanguard do the voting," says Jackson. "Maybe we should be a little bit worried. I don't know if we should be."

http://www.physorg.com/news/2011-08-amino-acid-alphabet-soup.html

Amino acid alphabet soup

August 19, 2011 by Clara Moskowitz

All life on Earth relies on a standard set of 20 amino acids to build the proteins that carry out life's essential actions. But did it have to be this way?

All living creatures on this planet use the same 20 amino acids, even though there are hundreds available in nature. Scientists therefore have wondered if life could have arisen based on a different set of amino acids. And what's more, could life exist elsewhere that utilizes an alternate collection of building blocks?

"Life has been using a standard set of 20 amino acids to build proteins for more than 3 billion years," said Stephen J. Freeland of the NASA Astrobiology Institute at the University of Hawaii. "It's becoming increasingly clear that many other amino acids were plausible candidates, and although there's been speculation and even assumptions about what life was doing, there's been very little in the way of testable hypotheses."

So Freeland and his University of Hawaii colleague Gayle K. Philip devised a test to try to learn if the 20 amino acids Earth's life uses were randomly chosen, or if they were the only possible ones that could have done the job. Amino acids are molecules built primarily from carbon, hydrogen, oxygen, and nitrogen. They assemble in particular shapes and patterns to form larger molecules called proteins that carry out biological functions.

"Technically there is an infinite variety of amino acids," Freeland told Astrobiology Magazine. "Within that infinity there are lots more than the 20 that were available [when life originated on Earth] as far as we can tell."

Testing the possibilities

The researchers defined a likely pool of candidate amino acids from which life drew its 20. They started with the amino acids that have been discovered within the so-called Murchison meteorite, a space rock that fell in Murchison, Victoria in Australia in September 1969. The rock is thought to date from the early solar system, and to represent a sample of which compounds existed in the solar system and on Earth before life began.

The scientists then used computers to estimate the fundamental properties of the 20 amino acids life uses, such as size, charge and hydrophilicity, or the extent to which the molecules are attracted to water.

"We know that these three are important to the ways they build proteins," Freeland said.

How did life end up selecting which amino acids in the primordial soup worked best? Image Credit: Peter Sawyer / Smithsonian Institution

Freeland and Philip analyzed whether these properties could have been achieved with as much coverage and efficiency with other combinations of 20 amino acids.

The researchers discovered that life seemingly did not choose its 20 building blocks randomly. "We found that chance alone would be extremely unlikely to pick a set of amino acids that outperforms life's choice," Freeland said.

Natural selection

In fact, the researchers think early life on Earth probably used a version of natural selection to choose these amino acids. Some combinations of other amino acids were likely tried, but none proved quite as fit, so no other combinations ended up producing the numbers of successful offspring that the existing set achieved.

"Here we found a very simple test that begins to show us that life knew exactly what it was doing," Freeland said. "This is consistent with the idea that there was natural selection going on."

Getting at the question of why nature chose the 20 amino acids it did is experimentally difficult, said Aaron Burton, a NASA Postdoctoral Program Fellow who works as an astrochemist at NASA's Goddard Space Flight Center in Greenbelt, Md.

"Although a number of experiments have shown that unnatural amino acids can be incorporated into the genetic alphabet of organisms, it may never be possible to experimentally simulate sufficient evolutionary time periods to truly compare alternate amino acid alphabets," said Burton, who was not involved in the new study. "As a result, studies such as those presented by Philip and Freeland offer interesting insights and provide a framework for formulating hypotheses that can actually be tested in the lab."

Amino acids in meteorites

Right now the race is on to directly find amino acids elsewhere in the solar system. Some hints that they abound have been found on meteorites that have landed on Earth from outer space, as well as from missions such as NASA's Stardust probe, which sampled the coma of comet Wild 2 in 2004.

"All signs are that amino acids are going to be found throughout the galaxy," Freeland said. "They are apparently obvious building blocks with which to construct life. What we're finding hints at a certain level of predictability in the way things turned out."

The question of life's amino acid toolbox is interesting not just in trying to trace the origin of the life on Earth, but in wondering whether life exists on other planets, and if so, what form it takes. Scientists are particularly curious about how a different set of amino acid building blocks would result in different characteristics in the life it creates.

"That is the biggest question of all," Freeland said. "We're trying to find a way to ask, if you change the set of amino acids with which we're building, what effect does that have on the proteins you can build. The most interesting thing is, nobody knows." Philip and Freeland reported their findings in a paper published in the April 19 issue of the journal Astrobiology.

http://www.eurekalert.org/pub_releases/2011-08/dumc-dmn081911.php

At last, a reason why stress causes DNA damage

DURHAM, N.C. – *For years, researchers have published papers that associate chronic stress with chromosomal damage.*

Now researchers at Duke University Medical Center have discovered a mechanism that helps to explain the stress response in terms of DNA damage.

"We believe this paper is the first to propose a specific mechanism through which a hallmark of chronic stress, elevated adrenaline, could eventually cause DNA damage that is detectable," said senior author Robert J. Lefkowitz, M.D., James B. Duke Professor of Medicine and Biochemistry and a Howard Hughes Medical Institute (HHMI) investigator at Duke University Medical Center.

The paper was published in the Aug. 21 online issue of Nature.

In the study, mice were infused with an adrenaline-like compound that works through a receptor called the beta adrenergic receptor that Lefkowitz has studied for many years. The scientists found that this model of chronic stress triggered certain biological pathways that ultimately resulted in accumulation of DNA damage. "This could give us a plausible explanation of how chronic stress may lead to a variety of human conditions and disorders, which range from merely cosmetic, like graying hair, to life-threatening disorders like malignancies," Lefkowitz said. P53 is a tumor suppressor protein and is considered a "guardian of the genome" – one that prevents genomic abnormalities.

"The study showed that chronic stress leads to prolonged lowering of p53 levels," said Makoto Hara, Ph.D., a postdoctoral fellow in the Lefkowitz laboratory. "We hypothesize that this is the reason for the chromosomal irregularities we found in these chronically stressed mice."

Lefkowitz earlier had proved the existence of isolated, and characterized the G-protein-coupled receptors (GPCRs) such as the beta adrenergic receptor. These receptors, which are located on the surface of the membranes that surround cells, are the targets of almost half of the drugs on the market today, including beta blockers for heart disease, antihistamines and ulcer medications.

Now he is continuing studies along another pathway,stemming from the GPCRs, that was discovered in his lab, which is known as the beta-arrestin pathway. At first, the theory was that beta-arrestin proteins turned off or desensitized the G-protein pathways, but evidence is accumulating that these proteins are also responsible for causing certain biochemical activities in their own right.

In the current study, the scientists found a molecular mechanism through which adrenaline-like compounds acted through both G-protein and the beta-arrestin pathways to trigger DNA damage.

The Nature publication showed that the infusion of an adrenaline-like compound for four weeks in the mice caused degradation of p53, which was present in lower levels over time.

The study also showed that the DNA damage was prevented in mice lacking beta-arrestin 1. Loss of beta-arrestin 1 stabilized cellular levels of p53 both in the thymus, an organ that strongly responds to acute or chronic stress, and in the testes, where paternal stress might affect an offspring's genome.

Future studies planned by the Lefkowitz laboratory include studying mice that are placed under stress (restrained), thus creating their own adrenaline or stress reaction to learn whether the physical reactions of stress, rather than an influx of adrenaline in the lab as was done in the current study, also leads to accumulation of DNA damage.

Other authors include Jeffrey J. Kovacs, Erin J. Whalen, Sudarshan Rajagopal, Ryan T. Strachan, Seungkirl Ahn, Barbara Williams, Christopher M. Lam, Kunhong Xiao, and Sudha K. Shenoy, all of the Duke Department of Medicine; Aaron J. Towers and Simon G. Gregory of the Department of Medicine and the Center for Human Genetics at Duke; and Wayne Grant and Derek R. Duckett of the Translational Research Institute, The Scripps Research Institute, Jupiter, Fla..

The study was supported by the Howard Hughes Medical Institute.

http://www.eurekalert.org/pub_releases/2011-08/nu-mab081711.php

Major ALS breakthrough

Researchers discover common cause of all forms of ALS

CHICAGO --- The underlying disease process of amyotrophic lateral sclerosis (ALS and Lou Gehrig's disease), a fatal neurodegenerative disease that paralyzes its victims, has long eluded scientists and prevented development of effective therapies. Scientists weren't even sure all its forms actually converged into a common disease process.

But a new Northwestern Medicine study for the first time has identified a common cause of all forms of ALS.

The basis of the disorder is a broken down protein recycling system in the neurons of the spinal cord and the brain. Optimal functioning of the neurons relies on efficient recycling of the protein building blocks in the cells. In ALS, that recycling system is broken. The cell can't repair or maintain itself and becomes severely damaged.

The discovery by Northwestern University Feinberg School of Medicine researchers, published in the journal Nature, provides a common target for drug therapy and shows that all types of ALS are, indeed, tributaries, pouring into a common river of cellular incompetence.

"This opens up a whole new field for finding an effective treatment for ALS," said senior author Teepu Siddique, M.D., the Les Turner ALS Foundation/Herbert C. Wenske Professor of the Davee Department of Neurology and Clinical Neurosciences at Northwestern's Feinberg School and a neurologist at Northwestern Memorial Hospital. "We can now test for drugs that would regulate this protein pathway or optimize it, so it functions as it should in a normal state."

The discovery of the breakdown in protein recycling may also have a wider role in other neurodegenerative diseases, specifically the dementias. These include Alzheimer's disease and frontotemporal dementia as well as Parkinson's disease, all of which are characterized by aggregations of proteins, Siddique said. The removal of damaged or misfolded proteins is critical for optimal cell functioning, he noted. This breakdown occurs in all three forms of ALS: hereditary, which is called familial; ALS that is not hereditary, called sporadic; and ALS that targets the brain, ALS/dementia. In related research, Feinberg School researchers also discovered a new gene mutation present in familial ALS and ALS/dementia, linking these two forms of the disease.

Siddique has been searching for the causes and underlying mechanism of ALS for more than a quarter century. He said he was initially drawn to it because, "It was one of the most difficult problems in neurology and the most devastating, a disease without any treatment or known cause." Siddique's efforts first showed in 1989 that molecular genetics techniques were applicable to ALS, then described the first ALS gene locus in 1991, which led to the discovery of SOD1 and engineering of the first genetic animal model for ALS.

ALS affects an estimated 350,000 people worldwide, including children and adults, with about 50 percent of people dying within three years of its onset. In the motor disease, people progressively lose muscle strength until they become paralyzed and can no longer move, speak, swallow and breathe. ALS/dementia targets the frontal and temporal lobes of the brain, affecting patients' judgment, the ability to understand language and to perform basic tasks like planning what to wear or organizing their day.

"These people in the prime of their lives and the peak of their productivity get this devastating illness that kills them," Siddique said. "The people who get ALS/dementia, an even more vicious disease, have a double whammy."

BROKEN DOWN RECYCLING SYSTEM

Feinberg School scientists found the cause of ALS by discovering a protein, ubiquilin2, whose critical job is to recycle damaged or misfolded proteins in motor and cortical neurons and shuttle them off to be reprocessed.

In people with ALS, Feinberg researchers found ubiquilin2 isn't doing its job. As a result, the damaged proteins and ubiquilin2 loiter and accumulate in the motor neurons in the spinal cord and cortical and hippocampal neurons in the brain. The protein accumulations resemble twisted skeins of yarn -- characteristic of ALS -- and cause the degeneration of the neurons. Researchers found ubiquilin2 in these skein-like accumulations in the spinal cords of ALS cases and in the brains of ALS/dementia cases.

The scientists also discovered mutations in ubiquilin2 in patients with familial ALS and familial ALS/dementia. But the skein-like accumulations were present in people's brains and spinal cords in all forms of ALS and ALS/dementia, whether or not they had the gene mutation.

"This study provides robust evidence showing a defect in the protein degradation pathway causes neurodegenerative disease," said Han-Xiang Deng, M.D., lead author of the paper and associate professor of neurology at the Feinberg School. "Abnormality in protein degradation has been suspected, but there was little direct evidence before this study." The other lead author is Wenjie Chen, senior research technologist in neurology.

About 90 percent of ALS is sporadic, without any known cause, until this study. The remaining 10 percent is familial. To date, mutations in about 10 genes, several of which were discovered at Northwestern, including SOD1 and ALSIN, account for about 30 percent of classic familial ALS, noted Faisal Fecto, M.D., study co-author and a graduate student in neuroscience at Feinberg.

http://news.discovery.com/earth/man-made-mountain-netherlands-110821.html

Man-Made Mountain Closer to Reality in Netherlands

A push to construct a mile-high mountain in the flat Netherlands is catching on and could become reality.

A mile-high mountain in the flatlands of the Netherlands is no longer just a pipe dream, the idea's main supporter, a newspaper columnist, said this weekend.

"The idea is not new but it's the first time that it is taken seriously by so many people," Thijs Zonneveld, a former athlete and writer for free daily De Pers said.

According to Zonneveld, engineers, construction firms, investors and experts are already busy discussing the feasibility of a man-made mountain that would include ski slopes, bobsleigh tracks, an ice rink, hiking trails, cliffs for climbing and scenic mountain roads with hairpin turns.

wine

"It seems like that my plea -- a joke at first -- has clicked," the columnist said.

During these socially difficult times, "people want to get excited about a big project and say: yes we can do that. Companies see a chance to create innovation platforms," he said.

The project website receives hundreds of hits every day and more than 8,200 people follow Zonneveld on Twitter.

A former semi-professional cyclist, Zonneveld decried his country's "boring" flatness in a column in July.

"Flat is ideal for growing beetroot, raising cows or building straight roads, but it's a catastrophe from a sports point-of-view," he wrote.

"I want a mountain, a real one. In the Netherlands."

Beyond being a tourist attraction, Zonneveld said his mountain would allow athletes to train in high altitudes and finally be on equal footing with competitors from countries with heights.

On Thursday, the cycling, skiing and climbing federations agreed.

"The hills in Limburg can no longer be compared to the Alps and Pyrenees. For this reason, we support the mountain," the federations said in a joint statement.

The ideal site would be the central Flevoland province, Zonneveld said.

"There's room and land is not expensive," he said adding that the province had already launched a study.

"I'm living a dream right now. It would be marvelous if the mountain became reality by 2018," the columnist said.