http://phys.org/news/2013-09-diamonds-trees-millions-years.html

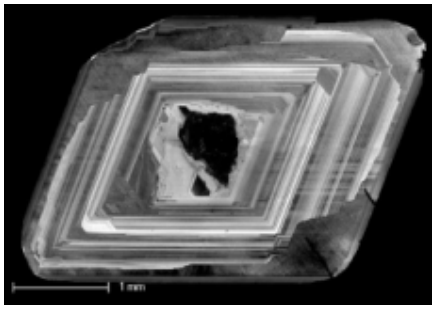
Diamonds grow like trees, but over millions of years

Diamonds consist of highly compressed carbon atoms and develop deep underground at relatively high pressures and temperatures of over 1000 degrees Celsius.

Earth scientists from VU University Amsterdam show that diamonds often have 'growth rings' similar to trees: due to changes in temperature and composition, the chemical composition in the growth zones changes, which leads to the development of 'rings' in the diamond. In addition, the scientists show that diamonds take millions of years to grow. Moreover, diamonds are often half as old as the Earth. Daphne Wiggers de Vries will defend her PhD thesis on this study on Thursday, September 19th.

Diamonds are billions of years old

The investigated diamonds are from Yakutia in Russia and show that in this region they formed in two important periods in the past: 1 billion years ago and 2 billion years ago. Many individual diamonds record growth in both periods proving for the first time that diamonds take millions of years to form. In both periods, major changes took place in the Earth's crust: tectonic plates in the region pushed together causing fluids rich in carbon to move enabling the diamonds to grow. Because diamonds are so old and grow very slowly, they are perfect to learn more about the history of the Earth.



Cross section of the cathodoluminescence of a diamond. Surrounding the core the 'growth rings' are clearly visible. The rings are different in color due to the varying conditions in which the diamond is formed.

Radioactive decay

Most diamonds contain microscopic mineral inclusions. The scientists used special equipment to determine the age of the rings in diamonds. Some mineral inclusions in diamonds contain the element rhenium, which slowly disintegrates into osmium, a process called radioactive decay. Mineral inclusions mostly contain rhenium at the time of inclusion of such a mineral in the diamond. Over time, the amount of rhenium decreases while the amount of osmium increases. When the diamond and the included mineral are formed simultaneously, the age of the mineral corresponds with the age of the diamond

http://www.eurekalert.org/pub_releases/2013-09/nrr-col091413.php

Can olive leaf extract attenuate lead-induced brain injury?

In recent years, neurotoxicity from exposure to low levels of lead in the environment has become increasingly prevalent. Therefore, the discovery of herbs that have lead-eliminating properties without harmful side effects is essential for the management of lead poisoning.

Preliminary studies by Yu Wang and colleagues from Longnan Teachers College have verified that olive leaf extract can protect the blood, spleen and hippocampus in lead-poisoned mice. However, little is known about the effects of olive leaf extract on lead-induced brain injury. A recent study from Yu Wang and colleagues investigated brain histological structure and antioxidant capacity in lead-poisoned mice as well as apoptotic factors in the cerebral cortex of mice using transmission electron microscopy, spectrophotometry and immunohistochemical staining. The researchers have confirmed that olive leaf extract can inhibit lead-induced brain injury by increasing antioxidant capacity and reducing apoptosis. These findings, published in the Neural Regeneration Research (Vol. 8, No. 22, 2013), initially reveal the action mechanism underlying olive leaf extract treatment for lead poisoning, and provide scientific evidence and theoretical basis for development and utilization of olive leaf in boosting the body antioxidant capacity and discharging foreign bodies.

Article: " Olive leaf extract inhibits lead poisoning-induced brain injury," by Yu Wang, Shengqing Wang, Wenhui Cui, Jiujun He, Zhenfu Wang, Xiaolu Yang (Department of Biology and Chemistry, Longnan Teacher's College, Chengxian 742500, Gansu Province, China)

Wang Y, Wang SQ, Cui WH, He JJ, Wang ZF, Yang XL. Olive leaf extract inhibits lead poisoning-induced brain injury. Neural Regen Res. 2013;8(22):2021-2029.

http://www.eurekalert.org/pub_releases/2013-09/osu-gmc091613.php

Gut microbes closely linked to proper immune function, other health issues

A new understanding of the essential role of gut microbes in the immune system may hold the key to dealing with some of the more significant health problems facing people in the world today, Oregon State University researchers say in a new analysis.

CORVALLIS, Ore. - Problems ranging from autoimmune disease to clinical depression and simple obesity may in fact be linked to immune dysfunction that begins with a "failure to communicate" in the human gut, the

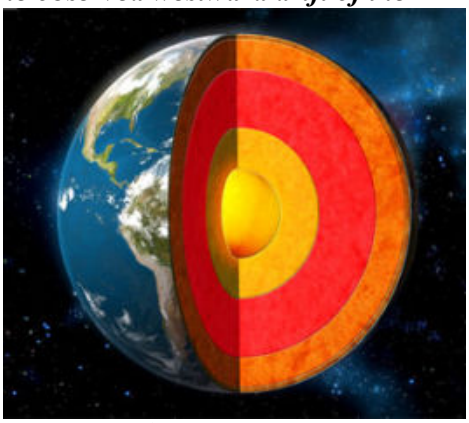
scientists say. Health care of the future may include personalized diagnosis of an individual's "microbiome" to determine what prebiotics or probiotics are needed to provide balance.
Appropriate sanitation such as clean water and sewers are good. But some erroneous lessons in health care may need to be unlearned -- leaving behind the fear of dirt, the love of antimicrobial cleansers, and the outdated notion that an antibiotic is always a good idea. We live in a world of "germs" and many of them are good for us.
"Asked about their immune system, most people might think of white blood cells, lymph glands or vaccines," said Dr. Natalia Shulzhenko, author of a new report in Clinical *Reviews in Allergy and Immunology*, and assistant professor and physician in the OSU Department of Biomedical Sciences. "They would be surprised that's not where most of the action is. Our intestines contain more immune cells than the entire rest of our body.
"The human gut plays a huge role in immune function," Shulzhenko said. "This is little appreciated by people who think its only role is digestion. The combined number of genes in the microbiota genome is 150 times larger than the person in which they reside. They do help us digest food, but they do a lot more than that."
An emerging theory of disease, Shulzhenko said, is a disruption in the "crosstalk" between the microbes in the human gut and other cells involved in the immune system and metabolic processes.
"In a healthy person, these microbes in the gut stimulate the immune system as needed, and it in turn talks back," Shulzhenko said. "There's an increasing disruption of these microbes from modern lifestyle, diet, overuse of antibiotics and other issues. With that disruption, the conversation is breaking down."
An explosion of research in the field of genomic sequencing is for the first time allowing researchers to understand some of this conversation and appreciate its significance, Shulzhenko said. The results are surprising, with links that lead to a range of diseases, including celiac disease and inflammatory bowel disease. Obesity may be related. And some studies have found relevance to depression, late-onset autism, allergies, asthma and cancer.
In the new review, researchers analyzed how microbe dysfunction can sometimes result in malabsorption and diarrhea, which affects tens of millions of children worldwide and is often not cured merely by better nutrition. In contrast, a high-fat diet may cause the gut microbes to quickly adapt to and prefer these foods, leading to increased lipid absorption and weight gain.
The chronic inflammation linked to most of the diseases that kill people in the developed world today -- heart disease, cancer, diabetes -- may begin with dysfunctional gut microbiota.
Understanding these processes is a first step to addressing them, Shulzhenko said. Once researchers have a better idea of what constitutes healthy microbiota in the gut, they may be able to personalize therapies to restore that balance. It should also be possible to identify and use new types of probiotics to mitigate the impact of antibiotics, when such drugs are necessary and must be used.
Such approaches are "an exciting target for therapeutic interventions" to treat health problems in the future, the researchers concluded.
The study, supported by OSU, included researchers from both the College of Veterinary Medicine and the College of Pharmacy.

http://scitechdaily.com/scientists-link-earths-westward-drift-magnetic-field-superrotation-inner-core/

Scientists Link Earth's Westward Drift of Magnetic Field and Superrotation of Inner Core

In a newly published study, researchers from the University of Leeds detail how they solved a 300-year-old riddle about which direction the center of the Earth spins, linking the observed westward drift of the magnetic field and superrotation of the inner core.

September 17, 2013 by Staff



Earth cross section showing its internal structure **from Shutterstock**

Scientists at the University of Leeds have solved a 300-year-old riddle about which direction the center of the Earth spins. The Earth's inner core, made up of solid iron, 'superrotates' in an eastward direction – meaning it spins faster than the rest of the planet – while the outer core, comprising mainly molten iron, spins westwards at a slower pace.
Although Edmund Halley – who also discovered the famous comet – showed the westward-drifting motion of the Earth's geomagnetic field in 1692, it is the first time that scientists have been able to link the way the inner core spins to the behavior of the outer core. The planet behaves in this way because it is responding to the Earth's geomagnetic field.
The findings, published in Proceedings of the National Academy of Sciences, help scientists to interpret the dynamics of the core of the Earth, the source of our planet's magnetic field.

In the last few decades, seismometers measuring earthquakes traveling through the Earth's core have identified an eastwards, or superrotation of the solid inner core, relative to Earth's surface. "The link is simply explained in terms of equal and opposite action", explains Dr Philip Livermore, of the School of Earth and Environment at the University of Leeds. "The magnetic field pushes eastwards on the inner core, causing it to spin faster than the Earth, but it also pushes in the opposite direction in the liquid outer core, which creates a westward motion."
The solid iron inner core is about the size of the Moon. It is surrounded by the liquid outer core, an iron alloy, whose convection-driven movement generates the geomagnetic field.
The fact that the Earth's internal magnetic field changes slowly, over a timescale of decades, means that the electromagnetic force responsible for pushing the inner and outer cores will itself change over time. This may explain fluctuations in the predominantly eastwards rotation of the inner core, a phenomenon reported for the last 50 years by Tkalčić et al. in a recent study published in Nature Geoscience.
Other previous research based on archeological artifacts and rocks, with ages of hundreds to thousands of years, suggests that the drift direction has not always been westwards: some periods of eastwards motion may have occurred in the last 3,000 years. Viewed within the conclusions of the new model, this suggests that the inner core may have undergone a westwards rotation in such periods.
The authors used a model of the Earth's core which was run on the giant super-computer Monte Rosa, part of the Swiss National Supercomputing Center in Lugano, Switzerland. Using a new method, they were able to simulate the Earth's core with an accuracy about 100 times better than other models.
The study was a collaboration between the University of Leeds and the Swiss Federal Institute of Technology, Zurich.
Publication: Philip W. Livermore, et al., "Electromagnetically driven westward drift and inner-core superrotation in Earth's core," PNAS, 2013; doi: 10.1073/pnas.1307825110

http://www.eurekalert.org/pub_releases/2013-09/uog-vca091613.php

Vaccinating cattle against E. coli O157 could cut human cases of infection by 85 percent, say scientists

Vaccinating cattle against the E. coli O157 bacterium could cut the number of human cases of the disease by 85%, according to scientists.

The bacteria, which cause severe gastrointestinal illness and even death in humans, are spread by consuming contaminated food and water, or by contact with livestock faeces in the environment. Cattle are the main reservoir for the bacterium. The vaccines that are available for cattle are rarely used, buc could be significant.
The research was lead by a team of researchers at the University of Glasgow in collaboration with the University of Edinburgh, the Royal Veterinary College, Scotland's Rural College, Health Protection Scotland, and the Scottish E. coli O157/VTEC Reference Laboratory.
The study, published in the online journal PNAS, used veterinary, human and molecular data to examine the risks of E. coli O157 transmission from cattle to humans, and to estimate the impact of vaccinating cattle.
The risk of E. coli O157 infection is particularly significant when the cattle are 'super-shedding' – excreting extremely high numbers of bacteria in their faeces for a limited period of time. Vaccines against the bacteria exist that can reduce super-shedding.
As a consequence, the researchers predict that vaccinating cattle could reduce human cases by nearly 85 percent, far higher than the 50 percent predicted by studies simply looking at the efficacy of current vaccines in cattle.
These figures provide strong support for the adoption of vaccines by the livestock industry, and work is now underway to establish the economic basis for such a programme of vaccination. In addition, research is continuing in Scotland by the same collaborative grouping to develop even more effective vaccines that would further reduce the impact on human disease.
Lead author, Dr Louise Matthews, Senior Research Fellow in the Institute of Biodiversity, Animal Health and Comparative Medicine, said: "E. coli O157 is a serious gastrointestinal illness. The economic impact is also serious – for instance studies in the US suggest that healthcare, lost productivity and food product recalls due to E. coli O157 can cost hundreds of millions of dollars each year.
"Treating cattle in order to reduce the number of human cases certainly makes sense from a human health perspective and, while more work is needed to calculate the cost of a vaccination programme, the public health justification must be taken seriously."
In Scotland, an average of 235 culture positive cases of E. coli O157 infection per year (i.e. people who had the organism in their stools) were notified to Health Protection Scotland from 2008 to 2012.
The vaccines that are available currently have poor take-up: one version in the US is not fully licensed because medicines for veterinary use must show that animal health is improved. This is problematic because E. coli

O157 does not harm cattle and assessing the impact of treatment involves coordination between human and veterinary health practitioners.
Senior author Professor Stuart Reid of the Royal Veterinary College added: "We increasingly recognise the fact that we share a common environment with the animals we keep – and inevitably the pathogens they harbour. This study is an excellent example the interface between veterinary and human medicine and of the concept of 'One Health' in action – controlling infections in animals can have a major impact on public health."
The study was funded by the Wellcome Trust International Partnership Award in Veterinary Epidemiology, BBSRC Institute Strategic Programmes at The Roslin Institute and The Pirbright Institute and the Foods Standards Agency Scotland.

http://www.eurekalert.org/pub_releases/2013-09/uoc--lcm091213.php

Lifestyle changes may lengthen telomeres, a measure of cell aging

Diet, meditation, exercise can improve key element of immune cell aging, UCSF scientists report

A small pilot study shows for the first time that changes in diet, exercise, stress management and social support may result in longer telomeres, the parts of chromosomes that affect aging.
It is the first controlled trial to show that any intervention might lengthen telomeres over time.
The study will be published online on Sept. 16, 2013 in The Lancet Oncology.
The study was conducted by scientists at UC San Francisco and the Preventive Medicine Research Institute, a nonprofit public research institute in Sausalito, Calif. that investigates the effect of diet and lifestyle choices on health and disease. The researchers say they hope the results will inspire larger trials to test the validity of the findings.
"Our genes, and our telomeres, are not necessarily our fate," said lead author Dean Ornish, MD, UCSF clinical professor of medicine, and founder and president of the Preventive Medicine Research Institute.
"So often people think 'Oh, I have bad genes, there's nothing I can do about it,'" Ornish said. "But these findings indicate that telomeres may lengthen to the degree that people change how they live. Research indicates that longer telomeres are associated with fewer illnesses and longer life."
Telomeres are the protective caps on the ends of chromosomes that affect how quickly cells age. They are combinations of DNA and protein that protect the ends of chromosomes and help them remain stable. As they become shorter, and as their structural integrity weakens, the cells age and die quicker.
In recent years, shorter telomeres have become associated with a broad range of aging-related diseases, including many forms of cancer, stroke, vascular dementia, cardiovascular disease, obesity, osteoporosis and diabetes.
For five years, the researchers followed 35 men with localized, early-stage prostate cancer to explore the relationship between comprehensive lifestyle changes, and telomere length and telomerase activity. All the men were engaged in active surveillance, which involves closely monitoring a patient's condition through screening and biopsies. Ten of the patients embarked on lifestyle changes that included: a plant-based diet (high in fruits, vegetables and unrefined grains, and low in fat and refined carbohydrates); moderate exercise (walking 30 minutes a day, six days a week); stress reduction (gentle yoga-based stretching, breathing, meditation). They also participated in weekly group support.
They were compared to the other 25 study participants who were not asked to make major lifestyle changes. The group that made the lifestyle changes experienced a "significant" increase in telomere length of approximately 10 percent. Further, the more people changed their behavior by adhering to the recommended lifestyle program, the more dramatic their improvements in telomere length, the scientists learned.
By contrast, the men in the control group who were not asked to alter their lifestyle had measurably shorter telomeres – nearly 3 percent shorter – when the five-year study ended. Telomere length usually decreases over time. The researchers say the findings may not be limited to men with prostate cancer, and are likely to be relevant to the general population. "We looked at telomeres in the participants' blood, not their prostate tissue," said Ornish.
The new study is a follow up to a similar, three-month pilot investigation in 2008 in which the same participants were asked to follow the same lifestyle program. After three months, the men in the initial study exhibited significantly increased telomerase activity. Telomerase is an enzyme that repairs and lengthens telomeres.
The new study was designed to determine if the lifestyle changes would affect telomere length and telomerase activity in these men over a longer time period. "This was a breakthrough finding that needs to be confirmed by larger studies," said co-senior author Peter R. Carroll, MD, MPH, professor and chair of the UCSF Department of Urology. "Telomere shortening increases the risk of a wide variety of chronic diseases," Carroll said. "We believe that increases in telomere length may help to prevent these conditions and perhaps even lengthen lifespan."

Other co-authors from UCSF include senior author and Nobel laureate Elizabeth H. Blackburn, PhD, professor of biochemistry and biophysics; Jue Lin, PhD, associate research biochemist; June M. Chan, DSc, associate professor of epidemiology & biostatistics; Elissa Epel, PhD, associate professor of psychiatry; Mark Jesus M. Magbanua, associate specialist; Jennifer Daubenmier and Nancy K. Hills, PhD, associate adjunct professors; and Nita Chainani-Wu, DMD, MPH, PhD, assistant clinical professor.

The research was supported by the U.S. Department of Defense; the National Institutes of Health and National Cancer Institute grant number RO1 R01CA101042; Furlotti Family Foundation; Bahna Foundation; DeJoria Foundation; Walton Family Foundation; Resnick Foundation; Greenbaum Foundation; Natwin Foundation; Safeway Foundation; and the Prostate Cancer Foundation.

Jue Lin, Elissa Epel and Elizabeth Blackburn were co-founders of Telome Health Inc., a diagnostic company that assess telomere biology – THI had no relationship to this study. Dean Ornish works with Healthways, Inc. to educate and support people in making healthier behaviors. The other authors declared no conflicts of interest.

http://www.eurekalert.org/pub_releases/2013-09/uoa-ost091613.php

Obese stomachs tell us diets are doomed to fail

The way the stomach detects and tells our brains how full we are becomes damaged in obese people but does not return to normal once they lose weight, according to new research from the University of Adelaide.

Researchers believe this could be a key reason why most people who lose weight on a diet eventually put that weight back on.

In laboratory studies, University of Adelaide PhD student Stephen Kentish investigated the impact of a high-fat diet on the gut's ability to signal fullness, and whether those changes revert back to normal by losing weight.

The results, published in the International Journal of Obesity, show that the nerves in the stomach that signal fullness to the brain appear to be desensitized after long-term consumption of a high-fat diet.

"The stomach's nerve response does not return to normal upon return to a normal diet. This means you would need to eat more food before you felt the same degree of fullness as a healthy individual," says study leader Associate Professor Amanda Page from the University's Nerve-Gut Research Laboratory.

"A hormone in the body, leptin, known to regulate food intake, can also change the sensitivity of the nerves in the stomach that signal fullness. In normal conditions, leptin acts to stop food intake. However, in the stomach in high-fat diet induced obesity, leptin further desensitizes the nerves that detect fullness.

"These two mechanisms combined mean that obese people need to eat more to feel full, which in turn continues their cycle of obesity."

Associate Professor Page says the results have "very strong implications for obese people, those trying to lose weight, and those who are trying to maintain their weight loss". "Unfortunately, our results show that the nerves in the stomach remain desensitized to fullness after weight loss has been achieved," she says.

Associate Professor Page says they're not yet sure whether this effect is permanent or just long-lasting.

"We know that only about 5% of people on diets are able to maintain their weight loss, and that most people who've been on a diet put all of that weight back on within two years," she says.

"More research is needed to determine how long the effect lasts, and whether there is any way – chemical or otherwise – to trick the stomach into resetting itself to normal."

This study has been funded by the National Health and Medical Research Council (NHMRC).

http://www.eurekalert.org/pub_releases/2013-09/muhc-hai091613.php

Heart attacks in young women -- not all have chest pain

MUHC-led study evaluates gender and age differences

Montreal– Chest pain is recognized as a symptom of heart troubles, but one out of five women aged 55 years or less having a heart attack do not experience this symptom, according to a study led by the Research Institute of the McGill University Health Centre (RI-MUHC). The research findings, gathered from partner institutions across Canada including the University of British Columbia (UBC), are the first to describe this phenomenon in young women. The study, published in JAMA Internal Medicine, has implications for emergency room healthcare professionals and for at-risk individuals, as seconds matter when it comes to the accurate diagnosis and treatment of heart attack.

"We need to move away from the image of an older man clutching his chest, when we think about acute coronary syndrome (ACS - the umbrella term referring to heart attacks and angina), says senior author of the study, Dr. Louise Pilote, director of the Division of General Internal Medicine at the MUHC and McGill University and professor of medicine at McGill University. "The reality is that chest pain, age and gender are no longer the definers of a heart attack. Our study demonstrates that young people and women who come into the emergency without chest pain, but other telltale ACS symptoms such as weakness, shortness of breath and/or rapid heartbeats, are in crisis. We need to be able to recognize this and adapt to new standard assessments in previously unrecognized groups such as young women."

"Women less than 55 years old are more likely to have their ACS misdiagnosed in the ER than men, and they have higher risk of death," adds first author Dr. Nadia Khan, associate professor of Medicine, UBC. "The public and physicians need to be aware of this problem."

Pain not an indicator of disease severity

Drs. Pilote, Khan and colleagues evaluated more than 1,000 young patients who were hospitalized for ACS. Their findings showed that women were less likely to experience chest pain compared with men and that the absence of this pain did not correlate with less severe heart attacks. Patients without chest pain had fewer symptoms overall but their ACS was not less severe. The diagnosis of ACS, therefore, depended on detailed cardiological assessments.

"It is important to remember that chest pain is a main indicator of ACS, but not the only one," says Dr. Pilote. "We need to remind ourselves that even without chest pain, something serious could still be happening," adds Dr. Khan.

This research was made possible thanks to funding from the Heart and Stroke Foundation and the Canadian Institutes of Health Research (CIHR).

http://www.scientificamerican.com/article.cfm?id=concussions-lingering-effects-linked-hormone-deficiency

Concussions' Lingering Effects Linked to Hormone Deficiency

The finding may explain why even seemingly mild concussions can give rise to persistent maladies

By Stephani Sutherland | Monday, September 16, 2013 | 3

When a blast rattles the brain, the resulting concussion sometimes leads to unremitting psychological problems such as depression, anxiety, irritability, sleep disorders, pain, and learning and memory problems. Tens of thousands of American veterans are estimated to suffer from this postconcussive syndrome (PCS), formerly associated with shell shock. Now evidence suggests that a hormone imbalance may underlie the chronic symptoms—meaning hormone replacement therapy could spur a dramatic recovery.

At least since World War I, scientists have tried to figure out why about 10 percent of adults' concussions—from any cause, including accidents, falls and sports injuries—lead to persistent psychological and physical complaints. Endocrinologist Charles Wilkinson of the VA Puget Sound and the University of Washington and his colleagues were intrigued by studies that found pituary hormone deficiencies, which affect only 1 percent of the general population, in many people who had had a concussion. No one had investigated whether a blast concussion could disrupt hormones as well, so Wilkinson's team tested 35 soldiers who had been near a bomb explosion. They found that a whopping half of the soldiers had undergone a precipitous drop in growth and sex hormones compared with other deployed soldiers without any concussions. The data were presented in April at the Experimental Biology 2013 meeting in Boston.

The researchers hypothesize that the force of a blast physically disrupts the pituitary gland's ability to either produce or transport its hormones. Receptors for growth hormone and its by-product hormone IGF-1 are found throughout the brain. The receptors' locations—in areas such as the amygdala, prefrontal cortex, putamen and hippocampus—correspond with functions that are disturbed in PCS, including mood, sleep and memory. In addition, hormones are thought to affect plasticity, maintenance and protection of the brain. Wilkinson and his colleagues plan to test soon whether hormone replacement therapy could benefit patients with PCS—he is optimistic because such therapy has been shown to improve the same symptoms in people with hormone deficiencies from other causes. “There is considerable evidence that the cognitive and mood problems of growth hormone deficiency can be treated successfully with growth hormone replacement,” Wilkinson says.

http://www.sciencedaily.com/releases/2013/09/130916140500.htm

Extremely Potent, Improved Derivatives of Successful Anticancer Drug Created

Scientists at The Scripps Research Institute (TSRI) have found a way to make dramatic improvements to the cancer cell-killing power of vinblastine, one of the most successful chemotherapy drugs of the past few decades.

The team's modified versions of vinblastine showed 10 to 200 times greater potency than the clinical drug. Even more significantly, these new compounds overcome the drug resistance that emerges upon treatment relapse, which renders continued or subsequent vinblastine treatment ineffective in some patients.

The TSRI researchers expect that similar modifications will boost the effectiveness of vincristine, a closely related drug that is commonly used against childhood leukemias and Hodgkin's disease.

"These new compounds should improve on what are already superb anticancer drugs," said Dale L. Boger, who is the Richard and Alice Cramer Professor and Chair of the Department of Chemistry at TSRI. Boger and members of his laboratory reported the discovery in a paper recently published online ahead of print by the journal ACS Medicinal Chemistry Letters.

Anticancer Agents

Vinblastine and vincristine are natural products of a pink-flowered herb known as the Madagascar periwinkle. Although the leaves of the plant had been used in traditional medicines for a range of other conditions, from diabetes to hemorrhoids, drug researchers at Eli Lilly found in the 1960s that the two compounds showed excellent potential as anticancer agents.

Both were found to selectively kill cancer cells by a mechanism that many other cancer drugs, including taxol, epothilones, and colchicine, have followed since -- they bind a cellular protein called tubulin in a way that interferes with the buildup and breakdown of tubulin-containing chains called microtubules -- structural elements of cells that play a key role in cell division. When the normal dynamics of their microtubules are disrupted, fast-dividing cancer cells stop dividing and die.

Since the 1960s, vinblastine has been used successfully in combination with other chemotherapy drugs against lymphomas as well as testicular, ovarian, breast, bladder and lung cancers. Vincristine is routinely used in combination regimes against childhood acute lymphoblastic leukemia and non-Hodgkin lymphomas. Both compounds are presently isolated from cultivated fields of the plants that make them naturally, but in trace amounts (0.0001% of the dry leaf weight). Since they are plant-derived natural products, they cannot be accessed using existing biotechnology or genetic engineering methods and, prior to the TSRI efforts, they were viewed as far too complex to be prepare by laboratory organic chemistry techniques. The authors developed a remarkable three-step preparation from commercially available chemicals using chemistry that they invented specifically for this purpose.

A significant limitation of vinblastine and vincristine is that, with extended treatment, they may evoke a powerful form of drug resistance. This resistance comes from a doorkeeper-type molecule called P-glycoprotein (Pgp), which transports infiltrating drug molecules out of the cancer cells. As cancer cells evolve to produce more and more Pgp, drugs fail to reach effective concentrations in cells and cancerous growth resumes. For years, medicinal chemists have tried to find modified versions -- "analogues" -- of these drugs that would overcome Pgp-mediated resistance, but without success.

Developing Extraordinary Potency

Last year, however, in a landmark paper in Organic Letters, Boger and his colleagues described a broad new method for modifying organic compounds like vinblastine, and demonstrated the method by making previously inaccessible variants of the drug. "Although it is a versatile method, we developed it specifically so that we could start making these vinblastine analogues that couldn't be made before," Boger said.

As his team used the method to make more new vinblastine analogues, the scientists discovered a type of modification to the drug that limits its usual drop in potency against resistant, Pgp-overproducing cancer cells as compared to non-resistant cancer cells. For the new study, the team explored variations of that modification and eventually found several analogues that were as good at killing resistant cells as ordinary vinblastine is at killing non-resistant cancer cells.

These new analogues were also many times more potent than vinblastine against non-resistant cells -- which are the kinds of cancer cells almost all patients have at diagnosis. The laboratory of a major drug company, Bristol-Myers Squibb, was able to repeat these results in a larger set of clinically important human tumor cell lines, and Boger's team confirmed that the new analogues' greater potency corresponds to their greater ability to bind to tubulin.

"The potency of these analogues is extraordinary -- they show activity down at the 100 picomolar level [100 trillionths of a mole] against some cell lines," said Boger. "So we have something here that's really unique, and we discovered it only because of the novel chemistry we developed."

Timothy J. Barker, Katharine K. Duncan, Katerina Otrubova, Dale L. Boger. Potent Vinblastine C20' Ureas Displaying Additionally Improved Activity Against a Vinblastine-Resistant Cancer Cell Line. ACS Medicinal Chemistry Letters, 2013; : 130909155105008 DOI: 10.1021/ml400281w

http://www.wired.com/wiredscience/2013/09/cdc-amr-rpt1/

CDC Threat Report: 'We Will Soon Be in a Post-Antibiotic Era'

CDC's first-of-its-kind assessment ranking America's threat from antibiotic-resistant organisms by the yearly number of illnesses and deaths

By Maryn McKenna

The U.S. Centers for Disease Control and Prevention has just published a first-of-its-kind assessment of the threat the country faces from antibiotic-resistant organisms, ranking them by the number of illnesses and deaths they cause each year and outlining urgent steps that need to be taken to roll back the trend.

The agency's overall - and, it stressed, conservative - assessment of the problem:

Each year, in the U.S., 2,049,442 illnesses caused by bacteria and fungi that are resistant to at least some classes of antibiotics;

Each year, out of those illnesses, 23,000 deaths;

Because of those illnesses and deaths, $20 billion each year in additional healthcare spending;

And beyond the direct healthcare costs, an additional $35 billion lost to society in foregone productivity.

"If we are not careful, we will soon be in a post-antibiotic era," Dr. Tom Frieden, the CDC's director, said in a media briefing. "And for some patients and for some microbes, we are already there."

The report marks the first time the agency has provided hard numbers for the incidence, deaths and cost of all the major resistant organisms. (It had previously estimated illnesses and deaths from some families of organisms or types of drug resistance, but those numbers were never gathered in one place.) It also represents the first time the CDC has ranked resistant organisms by how much and how imminent a threat they pose, using seven criteria: health impact, economic impact, how common the infection is, how easily it spreads, how much further it might spread in the next 10 years, whether there are antibiotics that still work against it, and whether things other than administering antibiotics can be done to curb its spread.

Out of that matrix, their top three "urgent" threats:

Carbapenem-resistant Enterobacteriaceae or CRE, a set of ICU germs that are resistant to almost all antibiotics: 9,000 infections per year, 600 deaths

Antibiotic-resistant gonorrhea, which currently responds to only one drug: 246,000 infections per year

Clostridium difficile, which is growing in resistance to one class of drugs, but more important, serves as a marker for the use of other antibiotics: 250,000 illnesses, 14,000 deaths.

There are 12 resistant bacteria and fungal infections in a second category, which the agency dubs "serious" (requiring "prompt and sustained action"); they include the hospital infections Acinetobacter, Pseudomonas aeruginosa, and VRE; the foodborne organisms Campylobacter, Salmonella and Shigella; MRSA; Candida, a fungal infection; and TB, among others. The last category, "concerning" (requiring "careful monitoring and prevention") includes rare but potent vancomycin-resistant staph, VRSA, as well as strains of strep resistant to two different categories of drugs.

For each organism, the report explains why it is a public health threat, where the trends are headed, what actions the CDC is taking, and what it is important for health care institutions, patients and their families, and states and local authorities to do to help. It also makes explicit where the trend of increasing and more common resistance is taking the country, outlining the risks to people taking chemotherapy for cancer, undergoing surgery, taking dialysis, receiving transplants, and undergoing treatment for rheumatoid arthritis.

(The report also - and this is so important that I'll take it up in a separate post tomorrow - tackles the issue of how agriculture, as well as healthcare, contributes to the increase in resistant organisms nationwide.)

The report lists some serious concerns the CDC has regarding how well resistance is monitored: in "gaps in knowledge," it specifically names limited national and international surveillance, as well as the lack of data on agricultural use of antibiotics. And it calls for action in four areas: gathering better data; preventing infections, through vaccination, better protective behavior in hospitals, and better food handling; improving the way in which antibiotics are used, by not using them inappropriately in health care or agriculture; and developing not just new categories of antibiotics but better diagnostic tests so that resistant organisms can be identified and dealt with sooner, before they spread.

In an interview before the report became public, Frieden said that some of these actions are already happening. "My biggest frustration is the pace of change," he told me. "Hospitals are making progress, but it's single digits in terms of the number of hospitals that are being very proactive. The challenge is scaling up what we know works, and doing that fast enough so that we can close the door on drug resistance before it's too late."

I talked about the report's calls for action to Dr. Ed Septimus, who is a professor of internal medicine at Texas A&M Health Sciences Center in Houston and a frequent spokesperson for the Infectious Diseases Society of America, the professional group for the physicians who usually treat resistant infections.

"We have gotten some action in Congress," he said, mentioning the GAIN Act, which passed and offers incentives for drug development, and the STAAR Act, which aimed to improve surveillance and antibiotic conservation, but did not pass. "The FDA is considering regulations that would allow a special designation for drugs for unmet needs, and resistance would qualify; and the NIH has prioritized research on resistance higher than it did 10 years ago. So there is movement - but in terms of funding, it is a slow difficult process.

"Still, there are things we can do without funding," he said: hospitals could create their own stewardship programs, and could work with nursing homes, whose patients bring some of the most resistant organisms into hospitals but who usually lack the budget for infection prevention.

"It's up to us to make the recommendations in this report happen," Septimus said. "If we do nothing but say, 'Here's the problem,' then the problem will continue to grow."

Cite: Antibiotic Resistance Threats in the United States, 2013, Centers for Disease Control and Prevention. Sept. 16, 2013.

http://www.sciencedaily.com/releases/2013/09/130916162020.htm

'Vicious Cycle' Shields, Spreads Cancer Cells

A "vicious cycle" produces mucus that protects uterine and pancreatic cancer cells and promotes their proliferation, according to researchers at Rice University. The researchers offer hope for a therapeutic solution.

They found that protein receptors on the surface of cancer cells go into overdrive to stimulate the production of MUC1, a glycoprotein that forms mucin, aka mucus. It covers the exposed tips of the elongated epithelial cells that coat internal organs like lungs, stomachs and intestines to protect them from infection.

But when associated with cancer cells, these slippery agents do their jobs too well. They cover the cells completely, help them metastasize and protect them from attack by chemotherapy and the immune system.

Details of the new work led by biochemist Daniel Carson, dean of Rice's Wiess School of Natural Sciences, appear in the Journal of Cellular Biochemistry.

In the paper, Carson, lead author Neeraja Dharmaraj, a postdoctoral researcher, and graduate student Brian Engel described MUC1 overexpression as particularly insidious not only for the way it protects tumor cells and promotes metastasis, but also because the cells create a feedback loop in which epidermal growth factor receptors (EGFR) and MUC1 interact to promote each other.

Carson described EGFR as a powerful transmembrane protein that stimulates normal cell growth, proliferation and differentiation. "What hadn't been considered is whether this activated receptor might actually promote the expression of MUC1, which would then further elevate the levels of EGFR and create this vicious cycle. "That's the question we asked, and the answer is 'yes,'" he said.

Carson compared mucus to Teflon. "Things don't stick to it easily, which is normally what you want. It's a primary barrier that keeps nasty stuff like pathogenic bacteria and viruses from getting into your cells," he said. But cancer cells "subvert systems and find ways to get out of control," he said. "They auto-activate EGFR by making their own growth factor ligands, for example, or mutating the receptor so it doesn't require the ligand anymore. It's always on."

Mucin proteins can then cover entire surface of a cell. "That lets (the cell) detach and move away from the site of a primary tumor," while still preventing contact with immune system cells and cytotoxins that could otherwise kill cancer cells, Carson said.

Hope comes in the form of a controversial drug, rosiglitazone, in the thiazolidinedione class of medications used in diabetes treatment, he said. The drug is suspected of causing heart problems over long-term use by diabetes patients. But tests on cancer cell lines at Rice found that it effectively attenuates the activation of EGFR and reduces MUC1 expression. That could provide a way to weaken the mucus shield.

"Chronic use of rosiglitazone can produce heart problems in a subset of patients, but if you're dying of pancreatic cancer, you're not worried about the long term," Carson said. "If you can reduce mucin levels in just a few days by using these drugs, they might make cancer cells easier to kill by established methods."

He said more work is required to see if rosiglitazone or some variant is suitable for trials. "We think it's best to understand all the effects," he said. "That might give us a rational way to modify these compounds, to avoid unwanted side effects and focus on what we want them to do."

Neeraja Dharmaraj, Brian J. Engel, Daniel D. Carson. Activated EGFR stimulates MUC1 expression in human uterine and pancreatic cancer cell lines. Journal of Cellular Biochemistry, 2013; 114 (10): 2314 DOI: 10.1002/jcb.24580

http://www.nytimes.com/2013/09/17/science/dna-double-take.html?partner=rss&emc=rss&pagewanted=all

DNA Double Take

Scientists are discovering that - to a surprising degree - we contain genetic multitudes.

By CARL ZIMMER

From biology class to “C.S.I.,” we are told again and again that our genome is at the heart of our identity. Read the sequences in the chromosomes of a single cell, and learn everything about a person’s genetic information - or, as 23andme, a prominent genetic testing company, says on its Web site, “The more you know about your DNA, the more you know about yourself.”

But scientists are discovering that - to a surprising degree - we contain genetic multitudes. Not long ago, researchers had thought it was rare for the cells in a single healthy person to differ genetically in a significant way. But scientists are finding that it’s quite common for an individual to have multiple genomes. Some people, for example, have groups of cells with mutations that are not found in the rest of the body. Some have genomes that came from other people.

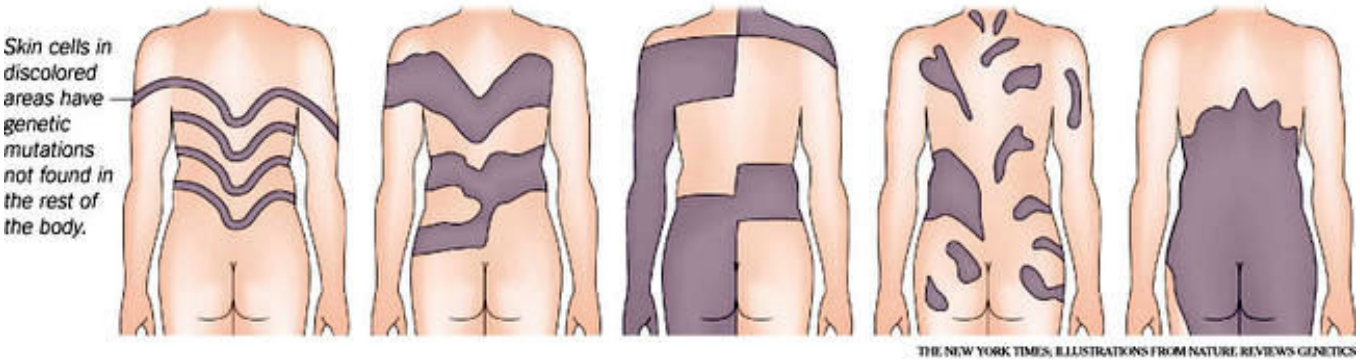
“There have been whispers in the matrix about this for years, even decades, but only in a very hypothetical sense,” said Alexander Urban, a geneticist at Stanford University. Even three years ago, suggesting that there was widespread genetic variation in a single body would have been met with skepticism, he said. “You would

have just run against the wall." But a series of recent papers by Dr. Urban and others has demonstrated that those whispers were not just hypothetical. The variation in the genomes found in a single person is too large to be ignored. "We now know it's there," Dr. Urban said. "Now we're mapping this new continent."
Dr. James R. Lupski, a leading expert on the human genome at Baylor College of Medicine, wrote in a recent review in the journal Science that the existence of multiple genomes in an individual could have a tremendous impact on the practice of medicine. "It's changed the way I think," he said in an interview.
Scientists are finding links from multiple genomes to certain rare diseases, and now they're beginning to investigate genetic variations to shed light on more common disorders.
Science's changing view is also raising questions about how forensic scientists should use DNA evidence to identify people. It's also posing challenges for genetic counselors, who can't assume that the genetic information from one cell can tell them about the DNA throughout a person's body.

Human Blueprint

When an egg and sperm combine their DNA, the genome they produce contains all the necessary information for building a new human. As the egg divides to form an embryo, it produces new copies of that original genome.
For decades, geneticists have explored how an embryo can use the instructions in a single genome to develop muscles, nerves and the many other parts of the human body. They also use sequencing to understand genetic variations that can raise the risk of certain diseases. Genetic counselors can look at the results of genetic screenings to help patients and their families cope with these diseases - altering their diet, for example, if they lack a gene for a crucial enzyme.
The cost of sequencing an entire genome has fallen so drastically in the past 20 years - now a few thousand dollars, down from an estimated $3 billion for the public-private partnership that sequenced the first human genome - that doctors are beginning to sequence the entire genomes of some patients. (Sequencing can be done in as little as 50 hours.) And they're identifying links between mutations and diseases that have never been seen before.

MOSAICISM Scientists have long known that genetic variations in different groups of skin cells can cause visible patterns on the body. But researchers are now finding such genetic variations, and even multiple genomes in a single person, are more common than previously thought.



THE NEW YORK TIMES; ILLUSTRATIONS FROM NATURE REVIEWS GENETICS

Mosaicism

Yet all these powerful tests are based on the assumption that, inside our body, a genome is a genome is a genome. Scientists believed that they could look at the genome from cells taken in a cheek swab and be able to learn about the genomes of cells in the brain or the liver or anywhere else in the body.
In the mid-1900s, scientists began to get clues that this was not always true. In 1953, for example, a British woman donated a pint of blood. It turned out that some of her blood was Type O and some was Type A. The scientists who studied her concluded that she had acquired some of her blood from her twin brother in the womb, including his genomes in his blood cells.
Chimerism, as such conditions came to be known, seemed for many years to be a rarity. But "it can be commoner than we realized," said Dr. Linda Randolph, a pediatrician at Children's Hospital in Los Angeles who is an author of a review of chimerism published in The American Journal of Medical Genetics in July.
Twins can end up with a mixed supply of blood when they get nutrients in the womb through the same set of blood vessels. In other cases, two fertilized eggs may fuse together. These so-called embryonic chimeras may go through life blissfully unaware of their origins.
One woman discovered she was a chimera as late as age 52. In need of a kidney transplant, she was tested so that she might find a match. The results indicated that she was not the mother of two of her three biological children. It turned out that she had originated from two genomes. One genome gave rise to her blood and some of her eggs; other eggs carried a separate genome.

Women can also gain genomes from their children. After a baby is born, it may leave some fetal cells behind in its mother's body, where they can travel to different organs and be absorbed into those tissues. "It's pretty likely that any woman who has been pregnant is a chimera," Dr. Randolph said.

Everywhere You Look

As scientists begin to search for chimeras systematically - rather than waiting for them to turn up in puzzling medical tests - they're finding them in a remarkably high fraction of people. In 2012, Canadian scientists performed autopsies on the brains of 59 women. They found neurons with Y chromosomes in 63 percent of them. The neurons likely developed from cells originating in their sons.

In The International Journal of Cancer in August, Eugen Dhimolea of the Dana-Farber Cancer Institute in Boston and colleagues reported that male cells can also infiltrate breast tissue. When they looked for Y chromosomes in samples of breast tissue, they found it in 56 percent of the women they investigated.

A century ago, geneticists discovered one way in which people might acquire new genomes. They were studying "mosaic animals," rare creatures with oddly-colored patches of fur. The animals didn't inherit the genes for these patches from their parents. Instead, while embryos, they acquired a mutation in a skin cell that divided to produce a colored patch.

Mosaicism, as this condition came to be known, was difficult to study in humans before the age of DNA sequencing. Scientists could only discover instances in which the mutations and the effects were big.

In 1960, researchers found that a form of leukemia is a result of mosaicism. A blood cell spontaneously mutates as it divides, moving a big chunk of one chromosome to another.

Later studies added support to the idea that cancer is a result of mutations in specific cells. But scientists had little idea of how common cases of mosaicism were beyond cancer. "We didn't have the technology to systematically think about them," said Dr. Christopher Walsh, a geneticist at Children's Hospital in Boston who recently published a review on mosaicism and disease in Science. "Now we're in the midst of a revolution."

Benign Differences

The latest findings make it clear that mosaicism is quite common - even in healthy cells.

Dr. Urban and his colleagues, for example, investigated mutations in cells called fibroblasts, which are found in connective tissue. They searched in particular for cases in which a segment of DNA was accidentally duplicated or deleted. As they reported last year, 30 percent of the fibroblasts carried at least one such mutation.

Michael Snyder of Stanford University and his colleagues searched for mosaicism by performing autopsies on six people who had died of causes other than cancer. In five of the six people they autopsied, the scientists reported last October, they found cells in different organs with stretches of DNA that had accidentally been duplicated or deleted.

Now that scientists are beginning to appreciate how common chimerism and mosaicism are, they're investigating the effects of these conditions on our health. "That's still open really, because these are still early days," Dr. Urban said.

Nevertheless, said Dr. Walsh, "it's safe to say that a large proportion of those mutations will be benign." Recent studies on chimeras suggest that these extra genomes can even be beneficial. Chimeric cells from fetuses appear to seek out damaged tissue and help heal it, for example.

But scientists are also starting to find cases in which mutations in specific cells help give rise to diseases other than cancer. Dr. Walsh, for example, studies a childhood disorder of the brain called hemimegalencephaly, in which one side of the brain grows larger than the other, leading to devastating seizures.

"The kids have no chance for a normal life without desperate surgery to take out half of their brain," he said.

Dr. Walsh has studied the genomes of neurons removed during those surgeries. He and his colleagues discovered that some neurons in the overgrown hemisphere have mutations to one gene. Two other teams of scientists have identified mutations on other genes, all of which help to control the growth of neurons. "We can get our hands on the mechanism of the disease," said Dr. Walsh.

Other researchers are now investigating whether mosaicism is a factor in more common diseases, like schizophrenia. "This will play itself out over the next 5 or 10 years," said Dr. Urban, who with his colleagues is studying it.

Moving Cautiously

Medical researchers aren't the only scientists interested in our multitudes of personal genomes. So are forensic scientists. When they attempt to identify criminals or murder victims by matching DNA, they want to avoid being misled by the variety of genomes inside a single person.

Last year, for example, forensic scientists at the Washington State Patrol Crime Laboratory Division described how a saliva sample and a sperm sample from the same suspect in a sexual assault case didn't match.

Bone marrow transplants can also confound forensic scientists. Researchers at Innsbruck Medical University in Austria took cheek swabs from 77 people who had received transplants up to nine years earlier. In 74 percent of the samples, they found a mix of genomes - both their own and those from the marrow donors, the scientists reported this year. The transplanted stem cells hadn't just replaced blood cells, but had also become cells lining the cheek.

While the risk of confusion is real, it is manageable, experts said. "This should not be much of a concern for forensics," said Manfred Kayser, a professor of Forensic Molecular Biology at Erasmus University in Rotterdam. In the cases where mosaicism or chimerism causes confusion, forensic scientists can clear it up by other means. In the Austrian study, for example, the scientists found no marrow donor genomes in the hair of the recipients.

For genetic counselors helping clients make sense of DNA tests, our many genomes pose more serious challenges. A DNA test that uses blood cells may miss disease-causing mutations in the cells of other organs. "We can't tell you what else is going on," said Nancy B. Spinner, a geneticist at the University of Pennsylvania, who published a review about the implications of mosaicism for genetic counseling in the May issue of Nature Reviews Genetics.

That may change as scientists develop more powerful ways to investigate our different genomes and learn more about their links to diseases. "It's not tomorrow that you're going to walk into your doctor's office and they're going to think this way," said Dr. Lupski. "It's going to take time."

http://www.eurekalert.org/pub_releases/2013-09/bmj-2ec091313.php

2008 economic crisis could be to blame for thousands of excess suicides worldwide

Researchers say 'urgent action' is needed to prevent further deaths

In a paper published today on bmj.com, researchers are suggesting that the 2008 global economic crisis could be to blame for the increase in suicide rates in European and American countries, particularly among males and in countries with higher levels of job losses.

In 2008, the International Labour Organization estimated that the number of jobless worldwide would reach approximately 212 million by 2009, an increase of 34 million compared with 2007. The World Health Organization raised concerns of the crisis' impact on global health and called for action to monitor and protect health, in particular amongst the poor and vulnerable.

Available studies only report data from a limited number of countries and there have been no systematic investigations into the broader international pattern or the sex/age groups and regions most affected.

In this study, the first to look at international trends in suicide, researchers from the universities of Hong Kong, Oxford and Bristol used the latest available data from 54 countries to assess changes in suicide rates following the 2008 crisis, as well as differential effects by sex, age, country and employment change.

Data were used from the World Health Organization (WHO) mortality database, the Centers for Disease Control and Prevention and the International Monetry Fund's World Economic Outlook database. Unemployment was used as the main economic indicator.

The researchers assessed time trends and estimated the expected numbers of suicides using former trends. They used the year 2000 as the starting point because suicide rates in some countries in the 1990s were affected by the recession in the early 1990s and the Asian economic crisis in the late 1990s. The main analysis focused on suicide rates in 2009.

Different age categories were used to determine whether the impact of the global economic crisis varied amongst ages: 15-24 (just entering labour market), 25-44 (early years of employment), 45-64 (later employment) and 65+ (post retirement).

In 2009, there was a 37% rise in unemployment and 3% falls in GDP per capita, reflecting the onset of the economic crisis in 2008. All European groups experienced rises in unemployment in 2009 and 2010. Unemployment rates started rising in USA and Canada in 2008 followed by dramatic increases in 2009-10.

In 2009, the overall male suicide rate rose 3.3%, with an excess of 5000 male suicides in all countries studied. These increases were mainly seen in the 27 European countries (4.2%) and 18 American countries (6.4%) studied. The largest increase in Europe was seen in 15-24 year old men and in 45-64 year old men in America. There was no change in suicide in European females and a small increase was seen in American females.

Also in 2009, new EU member states (Bulgaria, Czech Republic, Estonia, Hungary, Latvia, Lithuania, Malta, Poland, Romania and Solvenia) showed the largest increase in male suicide rates (13.3%) within Europe. USA and Canada showed an 8.9% increase and Caribbean and Central American countries showed a 6.4% increase in male suicides compared with a smaller increase in South American countries.

Rises seemed to be associated with the magnitude of increases in unemployment, particularly for males and in countries with low pre-crisis unemployment levels.
The researchers say that this study documents a "marked rise in suicide following the 2008 global economic crisis". The increases mainly occurred in men, with 5000 estimated excess suicides in 2009 compared with those expected based on previous trends.
The study adds to other evidence suggesting that the 2008 global economic crisis caused subsequent rises in suicide in affected countries. Several recent studies have shown an increased prevalence of depression or anxiety after the economic crisis, particularly in people who experienced unstable employment or financial problems. This is consistent with the documented increases in suicide during past recessions, such as the Great Depression in the 1930s.
The researchers say that their findings are "likely to be an underestimate of the true global impact of the economic crisis on suicide" as data were unavailable for a number of countries. Also, increases in suicide are likely to be the tip of the iceberg of recession-related emotional distress: for every suicide approximately 30-40 people make suicide attempts and for every suicide attempt about ten people experience suicidal thoughts. In the 20 European countries with available data for 2010, their analysis indicated an even larger increase in male suicide in 2010 (10.8%) than in 2009.
The researchers conclude that "urgent action is needed to prevent the economic crisis from further increasing suicides" and that labour market programmes may "help offset the impact of recession on suicide".
Research: Impact of 2008 global economic crisis on suicide: time trend study in 54 countries

http://www.sciencedaily.com/releases/2013/09/130917085606.htm

New Marker Identified for Early Diagnosis of Lung Cancer

A protein called isocitrate dehydrogenase (IDH1) is present at high levels in lung cancers and can be detected in the blood, making it a noninvasive diagnostic marker for lung cancers, according to a study published in Clinical Cancer Research, a journal of the American Association for Cancer Research.

"This study is the first to report identification of IDH1 as a novel biomarker for the diagnosis of non-small cell lung cancers (NSCLC) using a large number of clinical samples," said Jie He, M.D., Ph.D., director of the Laboratory of Thoracic Surgery at the Peking Union Medical College and Chinese Academy of Medical Sciences in Beijing. "Lung cancer has a high mortality rate, mostly because of late diagnosis. With an increase in aging population, we are likely to see an increase in lung cancer incidence and a need for better biomarkers for early diagnosis. We have identified IDH1 as an effective plasma biomarker with high sensitivity and specificity in the diagnosis of NSCLC, especially lung adenocarcinoma."
Lung cancer is the leading cause of cancer deaths in both men and women in the United States and worldwide. To detect lung cancer in blood, currently certain biomarkers including CEA, Cyfra21-1 and CA125 are used, but these markers are not very sensitive, according to He.
He and colleagues found that IDH1 could be detected in the blood of lung cancer patients with 76 percent sensitivity and 77 percent specificity. When they used a mathematical model to combine the detection of IDH1 with the detection of existing markers CEA, Cyfra21-1, and CA125, the sensitivity increased to 86 percent.
"Based on the present data, IDH1 can be used to detect stage 1 lung cancer; however, it is also possible that IDH1 could be used to detect precancer but further studies are required to address that possibility," said He.
He and colleagues used blood samples collected from 943 patients with NSCLC and 479 healthy controls, enrolled between 2007 and 2011 in the Cancer Institute and Hospital of the Chinese Academy of Medical Sciences. None of the study participants had a cancer diagnosis, nor were they treated for cancer in the three years prior to the study. Using methods called ELISA and ECL, they measured the levels of IDH1, CEA, Cyfra21-1, and CA125 in the participants' blood.
The researchers then divided the samples into a training set and a test set to validate the detection efficiency of IDH1. They found the data obtained from the test set were as good as those from the training set, demonstrating the robustness of IDH1 as a biomarker for lung cancer diagnosis.
The median IDH1 levels in patients with two types of lung cancer, adenocarcinoma and squamous cell carcinoma, were 2.7-fold and 2.2-fold higher, respectively, compared with healthy controls.
The researchers also found that combining the detection of all four markers -- IDH1, CEA, Cyfra21-1, and CA125 -- helped to better classify different types of adenocarcinoma, compared with detection with IDH1 alone.
He and colleagues are planning to conduct a multicenter clinical trial for further validation of IDH1.
"Our research also suggests IDH1 may be involved in the development of lung cancer, and it may be a good target for the treatment of NSCLC," said He. His team is currently studying the molecular mechanisms that increase IDH1 in lung cancer patients and its clinical implications.

Nan Sun, Zhaoli Chen, Fengwei Tan, Baihua Zhang, Ran Yao, Chengcheng Zhou, Jiagen Li, Yibo Gao, Ziyuan Liu, Xiaogang Tan, Fang Zhou, Max Y.f He, Kang Shao, Ning Li, Bin Qiu, Jian Sun, Yue Yu, Suya Wang, Yuda Zhao, Xuejiao Shi, and Jie He. Isocitrate Dehydrogenase 1 Is a Novel Plasma Biomarker for the Diagnosis of Non–Small Cell Lung Cancer. Clinical Cancer Research, September 2013 DOI: 10.1158/1078-0432.CCR-13-004

http://www.sciencedaily.com/releases/2013/09/130917124817.htm

Clean Energy Least Costly to Power America's Electricity Needs

Findings show carbon pollution from power plants can be cut cost-effectively by using wind, solar and natural gas

It's less costly to get electricity from wind turbines and solar panels than coal-fired power plants when climate change costs and other health impacts are factored in, according to a new study published in Springer's Journal of Environmental Studies and Sciences.

In fact -- using the official U.S. government estimates of health and environmental costs from burning fossil fuels -- the study shows it's cheaper to replace a typical existing coal-fired power plant with a wind turbine than to keep the old plant running. And new electricity generation from wind could be more economically efficient than natural gas.

The findings show the nation can cut carbon pollution from power plants in a cost-effective way, by replacing coal-fired generation with cleaner options like wind, solar, and natural gas.

"Burning coal is a very costly way to make electricity. There are more efficient and sustainable ways to get power," said Dr. Laurie Johnson, chief economist in the Climate and Clean Air Program at the Natural Resources Defense Council. "We can reduce health and climate change costs while reducing the dangerous carbon pollution driving global warming."

Johnson co-authored the study, "The Social Cost of Carbon: Implications for Modernizing our Electricity System," with Chris Hope of the Judge Business School, University of Cambridge; and Starla Yeh in NRDC's Center for Market Innovation. Power plants are the nation's single largest source of such pollution, accounting for 40 percent of our national carbon footprint.

"And yet, there are no federal limits on the amount of carbon pollution our power plants may release," said Johnson. "That's wrong. It doesn't make sense. It's putting our future at risk. We limit the amount of mercury, arsenic, soot, and other harmful pollution from these plants. It's time to cut this carbon pollution."

President Obama has vowed to do that, using his authority under the Clean Air Act to set the first federal limits on the amount of carbon pollution power plants may release. Critics claim that could raise costs. But, in fact, it can reduce the total cost of electricity generation, the new study finds.

Carbon pollution imposes economic costs by damaging public health and driving destructive climate change. Working together, the White House Office of Management and Budget, the Treasury Department, the Department of Energy and eight other federal agencies put a dollar value on those damages, in an official figure called the "social cost of carbon" (SCC).

The SCC is used to calculate the benefits (i.e., avoided climate damages) of carbon pollution reduction. The administration puts the best estimate at $33 per ton of carbon pollution emitted in 2010.

The study also included government damage estimates from sulfur dioxide, a pollutant released simultaneously with carbon. Every year, sulfur dioxide causes thousands of premature deaths, respiratory ailments, heart disease and a host of ecosystem damages.

"Already, climate change is contributing to record heat waves, floods, drought, wildfires and severe storms," Johnson said. Such extreme weather caused more than $140 billion in damages in 2012. American taxpayers picked up nearly $100 billion of those costs, according to an NRDC report released in May, 2013.

"These damages are only likely to increase if nothing is done to reduce carbon pollution," concluded Johnson.

Laurie T. Johnson, Starla Yeh, Chris Hope. The social cost of carbon: implications for modernizing our electricity system. Journal of Environmental Studies and Sciences, 2013; DOI: 10.1007/s13412-013-0149-5

http://bit.ly/15fCn6V

Pig-Manure Fertilizer Linked to Human MRSA Infections

Living near livestock farms and manure-treated fields are found to be associated with higher rates of antibiotic-resistant infection

By Sarah Zhang and Nature magazine | Tuesday, September 17, 2013 | 4

People living near pig farms or agricultural fields fertilized with pig manure are more likely to become infected with methicillin-resistant Staphylococcus aureus (MRSA) bacteria, according to a paper published today in JAMA Internal Medicine. Previous research has found that livestock workers are at high risk of carrying MRSA, compared to the general population. But it has been unclear whether the spreading of MRSA through livestock puts the public at risk of infection.

The study examined the incidence of infections in Pennsylvania, where manure from pig farms is often spread on crop fields to comply with state regulations for manure disposal. Researchers reviewed electronic health-care records from patients who sought care from the Pennsylvania-based Geisinger Health System (which helped to fund the study) in 2005–10.

The team analyzed cases of two different types of MRSA - community-associated MRSA (CA-MRSA), which affected 1,539 patients, and health-care-associated MRSA (HA-MRSA), which affected 1,335 patients. (The two categories refer to where patients acquire the infection as well as the bacteria's genetic lineages, but the distinction has grown fuzzier as more patients bring MRSA in and out of the hospital.) Then the researchers examined whether infected people lived near pig farms or agricultural land where pig manure was spread. They found that people who had the highest exposure to manure - calculated on the basis of how close they lived to farms, how large the farms were and how much manure was used - were 38% more likely to get CA-MRSA and 30% more likely to get HA-MRSA.

The researchers also analyzed 200 skin, blood, and sputum samples isolated from patients in the same health-care system in 2012. The MRSA strains found in those samples are commonly found in humans. Researchers did not find any evidence of bacteria belonging to clonal complex 398 (CC398), a MRSA strain classically associated with livestock and found in farms and farm workers in many previous studies.

However, there is little information about which MRSA strains are most common on US farms, so the absence of CC398 is not a sign that MRSA is not being transmitted from livestock to humans. "We've done studies in Iowa, we haven't always found CC398. That's not too shocking," says Tara Smith, a microbiologist at Kent State University in Ohio, who was not involved in the study.

Many researchers think that widespread use of antibiotics to encourage growth in farm animals fuels the proliferation of MRSA and other drug-resistant bacteria. The latest findings suggest that manure is helping antibiotic resistance to spread, says Joan Casey, an environmental-health scientist at the Johns Hopkins Bloomberg School of Public Health in Baltimore, Maryland, and a co-author of the study.

"We've certainly described a connection we think is plausible," she says. "We haven't described every step in the path."

"It's a pretty interesting and provocative observation," says Robert Daum, a pediatrician and the principal investigator of the MRSA Research Center at the University of Chicago in Illinois. He adds that he would like to see similar studies done in different geographic regions, and research to find out whether the MRSA strains carried in pig manure are the same as the MRSA strains found in nearby human infections.

Casey is at work on a follow-up genetics study to identify the most common MRSA strains in the region.

http://www.sciencenews.org/view/generic/id/353305/description/Poker_pros_arms_betray_their_hands

Poker pros' arms betray their hands

The way players move chips when betting signals card quality

By Bruce Bower

No bluff: In high-stakes matches, a poker face may not be good enough. Players may have to develop "poker arms" as well.

When shown two-second video clips of the arms and hands of top players making bets in the World Series of Poker, college students did well at judging who was playing a strong hand and who wasn't, say psychology graduate student Michael Slepian of Tufts University in Medford, Mass., and his colleagues.

But when viewing videos of only poker pros' upper bodies or faces during bets, students couldn't correctly predict whether players held good or bad cards, the researchers report Sept. 12 in Psychological Science.

In other words, experienced players' poker faces gave away nothing. "But professional poker players' arm movements enabled untrained observers to decode poker-hand quality," Slepian says.

Observers often rated poker players who held good cards as having moved their arms smoothly when pushing chips forward to make bets; bluffers moved their arms somewhat awkwardly. It's not known, though, whether poker players whose arm movements were rated as smooth really slid chips forward more gracefully than their opponents did.

Slepian's study adds to preliminary evidence that the ways in which people move provide clues to what they're thinking, remarks psychologist James Kilner of University College London.

A 2012 study coauthored by Kilner found that volunteers who responded to a lab task by moving a marble to one of two holes on a board did so more quickly when they were confident in their responses. In addition, observers consistently rated rapidly responding volunteers as confident in their decisions.

In the new study, Slepian and his colleagues divided 78 college students into three groups. Each group watched 20 video clips of big-time poker players placing bets. Clips showed the players' heads and arms, heads only or arms only. Study participants guessed at the quality of each poker hand on a scale from 1 to 7, from "very bad"

to “very good.” The researchers then compared these ratings with each player’s statistical likelihood of winning, as provided by the World Series of Poker.
None of the participants played poker regularly. Those who had played some poker, however, did best at using players’ arm movements to tell weak from strong hands.
Finally, the researchers found that a different group of 40 students rated poker players who were betting on strong hands as confident and as having smooth arm movements.
M. Slepian et al. Quality of professional players’ poker hands I perceived accurately from arm motions. Psychological Science. Published Sept. 12, 2013. doi:10.1177/0956797613487384. [Go to]

http://www.eurekalert.org/pub_releases/2013-09/ehs-dn091613.php

'Guns do not make a nation safer,' say doctors

Countries with lower gun ownership are safer than those with higher gun ownership, reports The American Journal of Medicine

Philadelphia, PA – A new study reports that countries with lower gun ownership are safer than those with higher gun ownership, debunking the widely quoted hypothesis that guns make a nation safer. Researchers evaluated the possible associations between gun ownership rates, mental illness, and the risk of firearm-related death by studying the data for 27 developed countries. Their findings are published in the current issue of The American Journal of Medicine.
Gun ownership in the US has been a hotly debated issue for more than 200 years. A popular notion in the US, where there are almost as many guns as people, is that "guns make a nation safer," although there has been little evidence either way. The shootings in Aurora, Tucson, Oak Creek, at Virginia Tech, among others in recent years, have demonstrated that there may be a relationship between mental illness and easy access to guns, and that lack of treatment for mental illness may be more of a pressing problem than mere availability of guns.
Ever since the second amendment stating "A well regulated militia being necessary to the security of a free State, the right of the people to keep and bear arms shall not be infringed" was passed in 1791, there has been a fierce debate over guns in the US. At one end is the argument that gun control laws are an infringement on the right to self-defense and on constitutional rights, and that there is no evidence that banning assault weapons would reduce crime. At the other end is the view that fewer firearms would reduce crime rates and overall lead to greater safety.
Sripal Bangalore, MD, MHA, of NYU Langone Medical Center, and Franz H. Messerli, MD, of St. Luke's Roosevelt Hospital, Columbia University College of Physicians and Surgeons, New York, examined data for 27 developed countries. The gun ownership data were obtained from the Small Arms Survey, and the data for firearm-related deaths were obtained from a European detailed mortality database (World Health Organization), the National Center for Health Statistics, and others. The crime rate was used as an indicator of safety of the nation and was obtained from the United Nations Surveys of Crime Trends.
"The gun ownership rate was a strong and independent predictor of firearm-related death," says Bangalore. "Private gun ownership was highest in the US. Japan, on the other end, had an extremely low gun ownership rate. Similarly, South Africa (9.4 per 100,000) and the US (10.2 per 100,000) had extremely high firearm-related deaths, whereas the United Kingdom (0.25 per 100,000) had an extremely low rate of firearm-related deaths. There was a significant correlation between guns per head per country and the rate of firearm-related deaths with Japan being on one end of the spectrum and the US being on the other. This argues against the notion of more guns translating into less crime. South Africa was the only outlier in that the observed firearms-related death rate was several times higher than expected from gun ownership."
The investigators also evaluated whether mental illness, and not merely the access to guns, is the driving force for criminal activities. They used age-standardized disability-adjusted life-year rates due to major depressive disorder per 100,000 inhabitants with data obtained from the World Health Organization database as a presumed indicator for mental illness burden in each country to assess whether there was a correlation between mental illness burden of a country and the crime rate in a country, but found no significant correlation between mental illness and crime rate.
Says Messerli and Bangalore, "Although correlation is not the same as causation, it seems conceivable that abundant gun availability facilitates firearm-related deaths. Conversely, high crime rates may instigate widespread anxiety and fear, thereby motivating people to arm themselves and give rise to increased gun ownership, which, in turn, increases availability. The resulting vicious cycle could, bit by bit, lead to the polarized status that is now the case with the US." They conclude that, "Regardless of exact cause and effect, the current study debunks the widely quoted hypothesis that countries with higher gun ownership are safer than those with low gun ownership."

http://www.eurekalert.org/pub_releases/2013-09/uab-ntf091813.php

Novel treatment for gonorrhea acts like a 'live vaccine,' prevents reinfection, animal study shows

Administered intravaginally, microsphere treatment reverses suppression of immunity and circumvents antibiotic resistance

BUFFALO, N.Y. -- A new gonorrhea treatment, based on an anti-cancer therapy developed by a Buffalo startup company, has successfully eliminated gonococcal infection from female mice and prevented reinfection, according to research published today by University at Buffalo scientists in the Journal of Infectious Diseases. Through TherapyX Inc., an early stage biotech company in Buffalo, the UB researchers have a $300,000 Small Business Innovation Research grant to develop the technology to treat and prevent gonorrhea infection. UB's Office for Science, Technology Transfer and Economic Outreach has filed for patent protection.

More than 100 million new gonococcal infections occur each year around the globe, according to the World Health Organization, which warns of a pending gonorrhea crisis due to soaring drug resistance rates. The infection can be asymptomatic but it also can cause extremely painful urination in men and pelvic inflammatory disease, which can lead to infertility and ectopic pregnancy in women. It may also make individuals more susceptible to infection with HIV/AIDS.

"We developed the concept that gonococcal infection seems to inhibit specific adaptive immune responses, which is, in part, why people can become infected with it multiple times," explains Michael W. Russell, PhD, professor of microbiology and immunology in the UB School of Medicine and Biomedical Sciences and senior author on the paper. "It turns out that gonococcal infection very cleverly controls the immune system, inducing responses the bacterium can fight and suppressing the responses that it cannot fight."

In considering how to modify the immune response to gonococcal infection, Russell became intrigued with an anti-cancer therapy being developed by a UB medical school colleague.

Nejat K. Egilmez, PhD, professor of microbiology and immunology at UB, and a co-author on the current paper, developed NanoCap, a sustained-release nanoparticle treatment that uses Interleukin-12, a cytokine or protein that helps stimulate an immune response against tumors that normally suppress immunity. Egilmez co-founded TherapyX Inc. to commercialize this and other drug formulations. "We had the idea that maybe these IL-12 microspheres that they were developing against tumors could be used to generate an immune response against gonococcal infection as well," says Russell. "This research proves that they can."

The current study describes how the IL-12 microspheres, administered intravaginally in mice, resulted in the development of a specific adaptive immune response -- development of antibodies specific to N. gonorrhoeae -- and clearance of the infection within days. One month later, attempts to reinfect these mice with the bacterium failed, demonstrating that the animals had retained the ability to fight reinfection.

"With this treatment, we have reversed the immunosuppression that gonococcal infection normally causes and allowed an effective immune response to develop," says Russell. "It could be argued that when the IL-12 microspheres are administered this way, they serve as an adjuvant that, in effect, converts the gonococcal infection into a live vaccine, thus essentially vaccinating the very population that is at risk for repeat infections." And because it may circumvent the growing resistance of this bacterium and others to antibiotics, this treatment method also may open up new approaches for the development of non-resistant treatments for other infectious diseases, Russell says.

"Here, we are delivering cytokines locally right to the site of infection," he says. "If we can use this method to teach the immune system to generate the right kind of response to other recalcitrant infections, then we could have a new approach to treat a range of infectious diseases without stimulating drug resistance."

The immunity developed in the mice lasted for one month. Russell plans to see if the immunity can last longer in mice and then ultimately, to test it in humans.

The first author on the paper is Yingru Liu, MD, PhD, UB research assistant professor in the Department of Microbiology and Immunology and a principal investigator at TherapyX. All three authors are researchers in UB's Witebsky Center for Microbial Pathogenesis and Immunology. The work was funded by the National Institute of Allergy and Infectious Diseases at the National Institutes of Health and a grant from The John R. Oishei Foundation of Buffalo.

http://www.eurekalert.org/pub_releases/2013-09/uoc--uds091713.php

UCLA doctors successfully 'vacuum' 2-foot blood clot out of patient's heart

First in state to perform minimally invasive alternative to open-heart surgery

Todd Dunlap, 62, arrived at Ronald Reagan UCLA Medical Center's emergency room on Aug. 8 suffering from shortness of breath, fatigue and extreme cold. When a CT scan revealed a 24-inch clot stretching from his legs into his heart, doctors feared the mass could break loose and lodge in his lungs, blocking oxygen and killing him instantly.

Dr. John Moriarty gave his patient a choice. Dunlap could have open-heart surgery or undergo a new minimally invasive procedure using a device called AngioVac to vacuum the massive clot out of his heart. The catch? The procedure had never been successfully performed in California.
A new grandfather, Dunlap didn't hesitate to choose the second option and underwent the procedure on Aug. 14. A week later, he was home, full of energy and eager to play on the floor with his 9-month-old grandson.
Here's how it worked: A team of UCLA interventional radiologists and cardiovascular surgeons slid a tiny camera down Dunlap's esophagus to visually monitor his heart. Next, they guided a coiled hose through his neck artery and plugged one end into his heart, against the clot. They threaded the other end through a vein at the groin and hooked the hose up to a powerful heart-bypass device in the operating room to create suction.
"Once in place, the AngioVac quickly sucked the deadly clot out of Mr. Dunlap's heart and filtered out the solid tissue," said Moriarty, a UCLA interventional radiologist with expertise in clot removal and cardiovascular imaging. "The system then restored the cleansed blood through a blood vessel near the groin, eliminating the need for a blood transfusion."
The procedure lasted three hours. Doctors observed Dunlap for three days in intensive care before transferring him to the hospital's cardiac ward and then discharging him four days later.
Open-heart surgery takes twice as long to perform and often requires the surgeon to divide the breastbone lengthwise down the middle and spread the halves apart to access the heart. After the heart is repaired, surgeons use wires to hold the breastbone and ribs in place as they heal. The procedure can necessitate extended rehabilitation before the patient makes a full recovery.
"Retrieving a clot from within the heart used to require open-heart surgery, resulting in longer hospitalization, recovery and rehabilitation times compared to the minimally invasive approach provided by the AngioVac system," said Dr. Murray Kwon, a UCLA cardiothoracic surgeon who collaborated on Dunlap's procedure.
Similarly, a clot-busting drug known as a tPA typically takes three to four days to work. In Dunlap's case, his physicians tried tPA first, but it failed due to the clot's large size and density.
"The AngioVac was the last resort for Mr. Dunlap," said Moriarty. "The clot clogged his heart chamber like a wad of gum in a pipe. Every moment that passed increased the risk that the clot would migrate to his lungs and kill him. We couldn't have asked for a better outcome."
"I'm thrilled that I didn't have to go through open-heart surgery," said Dunlap, a resident of Newbury Park, Calif., who is the father of two adult sons. "This procedure is a great option for the older, frail person who wouldn't survive open-heart surgery. Without an alternative like this, he's a goner."
Like Dunlap, roughly one in 500 Americans suffers from blood clots in the leg veins, a condition called deep vein thrombosis. Estimates double in people older than 80. Nearly 100,000 Americans die each year when a clot breaks away from the blood-vessel wall and lodges in the lungs or heart. In one of every four cases, sudden death is the only clue an individual is suffering from the condition.
"When you hear about new cutting-edge options, it gives you hope," said Cheryl Dunlap, who has been married to Todd for 32 years. "Without it, you run into a brick wall. If we'd consulted only with our community hospital and not a teaching facility like UCLA, we wouldn't have learned about all the treatment choices available to us."
"It takes a large team of experts to perform a potentially high-risk procedure like this for the first time," Moriarty said. "We couldn't have been successful without the collaboration of our colleagues in cardiac surgery, radiology, cardiology and anesthesia."
For more information about the AngioVac or removing clots from the heart, legs, veins and lungs, please call the UCLA Radiology Consultation Center at 310-481-7545 or go to http://www.radiology.ucla.edu.

http://www.eurekalert.org/pub_releases/2013-09/uom-cro091713.php

Coma: Researchers observe never-before-detected brain activity

Active brain state beyond the deep coma associated with a flat EEG

Researchers from the University of Montreal and their colleagues have found brain activity beyond a flat line EEG, which they have called Nu-complexes (from the Greek letter Nv). According to existing scientific data, researchers and doctors had established that beyond the so-called "flat line" (flat electroencephalogram or EEG), there is nothing at all, no brain activity, no possibility of life. This major discovery suggests that there is a whole new frontier in animal and human brain functioning.
The researchers observed a human patient in an extreme deep hypoxic coma under powerful anti-epileptic medication that he had been required to take due to his health issues. "Dr. Bogdan Florea from Romania contacted our research team because he had observed unexplainable phenomena on the EEG of a coma patient. We realized that there was cerebral activity, unknown until now, in the patient's brain," says Dr. Florin Amzica, director of the study and professor at the University of Montreal's School of Dentistry.

Dr. Amzica's team then decided to recreate the patient's state in cats, the standard animal model for neurological studies. Using the anesthetic isoflurane, they placed the cats in an extremely deep—but completely reversible—coma. The cats passed the flat (isoelectric) EEG line, which is associated with silence in the cortex (the governing part of the brain). The team observed cerebral activity in 100% of the cats in deep coma, in the form of oscillations generated in the hippocampus, the part of the brain responsible for memory and learning processes. These oscillations, unknown until now, were transmitted to the master part of the brain, the cortex. The researchers concluded that the observed EEG waves, or Nu-complexes, were the same as those observed in the human patient.

Dr. Amzica stresses the importance of understanding the implications of these findings. "Those who have decided to or have to 'unplug' a near-brain-dead relative needn't worry or doubt their doctor. The current criteria for diagnosing brain death are extremely stringent. Our finding may perhaps in the long term lead to a redefinition of the criteria, but we are far from that. Moreover, this is not the most important or useful aspect of our study," Dr. Amzica said.

From Nu-complexes to therapeutic comas

The most useful aspect of this finding is the therapeutic potential, the neuroprotection, of the extreme deep coma. After a major injury, some patients are in such serious condition that doctors deliberately place them in an artificial coma to protect their body and brain so they can recover. But Dr. Amzica believes that the extreme deep coma experimented on the cats may be more protective.

"Indeed, an organ or muscle that remains inactive for a long time eventually atrophies. It is plausible that the same applies to a brain kept for an extended period in a state corresponding to a flat EEG," says Professor Amzica. "An inactive brain coming out of a prolonged coma may be in worse shape than a brain that has had minimal activity. Research on the effects of extreme deep coma during which the hippocampus is active, through Nu-complexes. is absolutely vital for the benefit of patients."

"Another implication of this finding is that we now have evidence that the brain is able to survive a an extremely deep coma if the integrity of the nervous structures is preserved," said lead author of the study, Daniel Kroeger. "We also found that the hippocampus can send 'orders' to the brain's commander in chief, the cortex. Finally, the possibility of studying the learning and memory processes of the hippocampus during a state of coma will help further understanding of them. In short, all sorts of avenues for basic research are now open to us."

The study "Human brain activity patterns beyond the isoelectric line of extreme deep coma" by Daniel Kroeger and Florin Amzica of the University of Montreal's Department of Stomatology, and Bogdan Florea of the Medical Centre Regina Maria (Cluj-Napoca, Romania) was published in PLOS ONE. The University of Montreal is officially known as Université de Montréal. http://dx.plos.org/10.1371/journal.pone.0075257.

http://www.eurekalert.org/pub_releases/2013-09/aaon-cvb091113.php

Can vitamin B supplements help stave off stroke?

New evidence suggests that taking vitamin B supplements may help reduce the risk of stroke.

MINNEAPOLIS – The research appears in the September 18, 2013, online issue of Neurology®, the medical journal of the American Academy of Neurology.

"Previous studies have conflicting findings regarding the use of vitamin B supplements and stroke or heart attack," said author Xu Yuming, with Zhengzhou University in Zhengzhou, China.

"Some studies have even suggested that the supplements may increase the risk of these events."

For the research, scientists analyzed 14 randomized clinical trials with a total of 54,913 participants.

All of the studies compared B vitamin use with a placebo or a very low-dose B vitamin.

Participants were then followed for a minimum of six months.

There were 2,471 strokes throughout the studies, all of which showed some benefit of taking vitamin B.

Vitamin B lowered the risk of stroke in the studies overall by seven percent. However, taking supplements did not appear to affect the severity of strokes or risk of death from stroke.

Folic acid, a supplemental form of folate (vitamin B9), which is often found in fortified cereals, appeared to reduce the effect of vitamin B.

Researchers did not find a reduction in stroke risk for vitamin B12.

"Based on our results, the ability of vitamin B to reduce stroke risk may be influenced by a number of other factors such as the body's absorption rate, the amount of folic acid or vitamin B12 concentration in the blood, and whether a person has kidney disease or high blood pressure," said Yuming.

"Before you begin taking any supplements, you should always talk to your doctor."

http://www.eurekalert.org/pub_releases/2013-09/uoea-usr091713.php

UEA scientists reveal Earth's habitable lifetime and investigate potential for alien life

Habitable conditions on Earth will be possible for at least another 1.75 billion years – according to astrobiologists at the University of East Anglia.

Findings published today in the journal Astrobiology reveal the habitable lifetime of planet Earth – based on our distance from the sun and temperatures at which it is possible for the planet to have liquid water.

The research team looked to the stars for inspiration. Using recently discovered planets outside our solar system (exoplanets) as examples, they investigated the potential for these planets to host life.

The research was led by Andrew Rushby, from UEA's school of Environmental Sciences. He said: "We used the 'habitable zone' concept to make these estimates – this is the distance from a planet's star at which temperatures are conducive to having liquid water on the surface." "We used stellar evolution models to estimate the end of a planet's habitable lifetime by determining when it will no longer be in the habitable zone. We estimate that Earth will cease to be habitable somewhere between 1.75 and 3.25 billion years from now. After this point, Earth will be in the 'hot zone' of the sun, with temperatures so high that the seas would evaporate. We would see a catastrophic and terminal extinction event for all life.

"Of course conditions for humans and other complex life will become impossible much sooner – and this is being accelerated by anthropogenic climate change. Humans would be in trouble with even a small increase in temperature, and near the end only microbes in niche environments would be able to endure the heat.

"Looking back a similar amount of time, we know that there was cellular life on earth. We had insects 400 million years ago, dinosaurs 300 million years ago and flowering plants 130 million years ago. Anatomically modern humans have only been around for the last 200,000 years – so you can see it takes a really long time for intelligent life to develop.

"The amount of habitable time on a planet is very important because it tells us about the potential for the evolution of complex life – which is likely to require a longer period of habitable conditions.

"Looking at habitability metrics is useful because it allows us to investigate the potential for other planets to host life, and understand the stage that life may be at elsewhere in the galaxy.

"Of course, much of evolution is down to luck, so this isn't concrete, but we know that complex, intelligent species like humans could not emerge after only a few million years because it took us 75 per cent of the entire habitable lifetime of this planet to evolve. We think it will probably be a similar story elsewhere."

Almost 1,000 planets outside our solar system have been identified by astronomers.

The research team looked at some of these as examples, and studied the evolving nature of planetary habitability over astronomical and geological time. "Interestingly, not many other predictions based on the habitable zone alone were available, which is why we decided to work on a method for this.

Other scientists have used complex models to make estimates for the Earth alone, but these are not suitable for applying to other planets.

"We compared Earth to eight planets which are currently in their habitable phase, including Mars. We found that planets orbiting smaller mass stars tend to have longer habitable zone lifetimes. "One of the planets that we applied our model to is Kepler 22b, which has a habitable lifetime of 4.3 to 6.1 billion years. Even more surprising is Gliese 581d which has a massive habitable lifetime of between 42.4 to 54.7 billion years. This planet may be warm and pleasant for 10 times the entire time that our solar system has existed!

"To date, no true Earth analogue planet has been detected. But it is possible that there will be a habitable, Earth-like planet within 10 light-years, which is very close in astronomical terms. However reaching it would take hundreds of thousands of years with our current technology. "If we ever needed to move to another planet, Mars is probably our best bet. It's very close and will remain in the habitable zone until the end of the Sun's lifetime - six billion years from now."

'Habitable Zone Lifetimes of Exoplanets around Main Sequence Stars' by Andrew Rushby, Mark Claire, Hugh Osborne and Andrew Watson is published in the journal Astrobiology on Thursday, September 19, 2013.

http://bit.ly/15hRtsF

Mini drug factory churns out drugs from inside bone

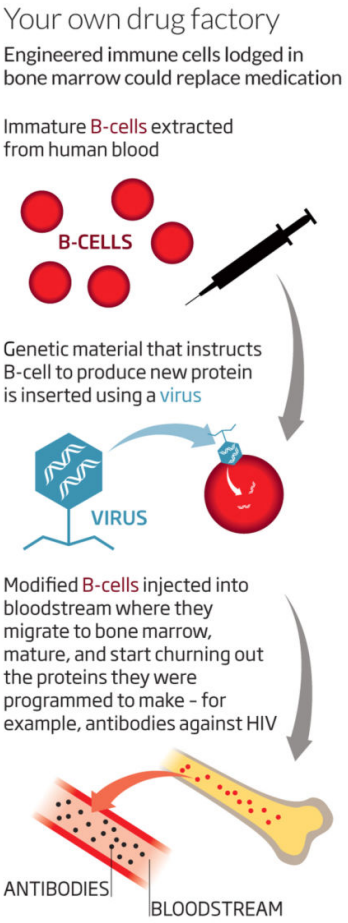
IMAGINE never having to take a pill again. Instead, mini drug factories hidden inside your bones, and made from your own immune cells, would churn out personalised drugs and other molecules designed to keep you fit and healthy.

Updated 12:07 20 September 2013 by Linda Geddes

Such a factory has been created in mice, and could soon be tested in humans to treat HIV.

"We want to turn people's cells into drug factories, giving them the genetic information they need to produce their own treatment," says Matthew Scholz of Immusoft in Seattle, which is developing the technique.

Immusoft is focusing on B-cells, a component of the immune system. One of their roles is to produce antibodies against infections or unwanted foreign substances such as toxins.



In 2009, David Baltimore and colleagues at the California Institute of Technology in Pasadena showed that stem cells from bone marrow could be engineered to develop into B-cells that made antibodies against HIV (Blood, doi.org/dwm53t).

The Immusoft researchers have built on that work, extracting immature B-cells from human blood and treating them with a virus that inserts the genetic code for a new protein (see diagram). When the modified cells were injected into the bloodstream of mice, some migrated to the bone marrow. Here they began making the proteins they had been engineered to make – a class of so-called broadly neutralising antibodies against HIV.

"I think this is potentially very important," says Carl June at the University of Pennsylvania, Philadelphia, who has been engineering another part of the immune system, T-cells, to fight HIV. "But whether this has clinical use depends on the efficiency of gene transfer, expression and secretion of the antibody, and most importantly the level of secreted protein in body fluids."

"If they can achieve therapeutic levels of antibody, this could be a real advance in cellular therapy," Baltimore says.

One of the remaining challenges is to ensure that the correct amount of antibody is produced. "The risk of too little or too much has to be considered," says Michel Sadelain of the Memorial Sloan-Kettering Cancer Center in New York, who has engineered T-cells to fight cancer.

It's also not clear how long the engineered B-cells can be made to survive. In the tests on mice, many of the cells died, but some were still alive 100 days later. "It's a very attractive idea to supply stable levels of antibodies from a cellular source in the body. The challenge is finding the right cell that would persist for the desired amount of time – days, or years in some cases – and that's a big challenge," Sadelain says.

The next step is to try injecting the modified B-cells into people, with Immusoft gearing up to do a trial in HIV-positive adults. If this succeeds, the approach could also be used in children with rare enzyme disorders like mucopolysaccharidosis. These children are unable to make certain enzymes, leading to the breakdown of various tissues, loss of vision and cognitive problems. Untreated, they usually die by the age of 12.

Hamster cells can be harnessed to produce replacement enzymes, but the treatment costs around $250,000 a year and involves regular injections. "For disorders like that, being able to produce the enzyme 24/7 in your body would be a big advantage," says Scholz, who presented the mouse results at the Strategies for Engineered Negligible Senescence meeting in Cambridge, UK, this month.

Ultimately, it might be possible to engineer B-cells to churn out any protein of choice. They could boost levels of hormones that fall as we age, or other substances that keep the body healthy, such as humanin. This protects brain cells against Alzheimer's disease, and is present at higher than average levels in people who live to be 100. "It might be possible to recreate the biochemical environment of youth," Scholz says.

http://www.sciencedaily.com/releases/2013/09/130918180425.htm

What's That Smell? Ten Basic Odor Categories Sniffed out With Math

Taste can be classified into five flavors that we sense, but how many odors can we smell?

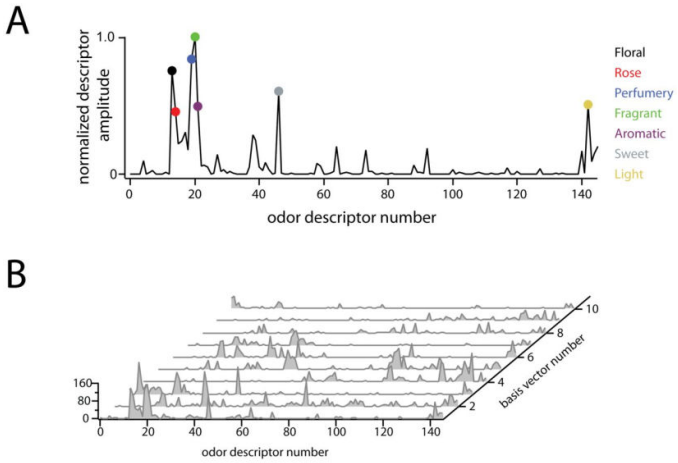
There are likely about 10 basic categories of odor, according to research published September 18th in the open access journal PLOS ONE by Jason Castro from Bates College, Chakra Chennubhotla from the University of Pittsburgh, and Arvind Ramanathan from Oak Ridge National Laboratory. The researchers used advanced statistical techniques to develop an approach for systematically describing smells.

Working with a standard set of data, Andrew Dravniek's 1985 Atlas of Odor Character Profiles, the researchers applied a mathematical method to simplify the olfactory information into coherent categories, similar to the way compressing a digital audio or image file reduces the file's size without, ideally, compromising its usefulness.

The team identified 10 basic odor qualities: fragrant, woody/resinous, fruity (non-citrus), chemical, minty/peppermint, sweet, popcorn, lemon and two kinds of sickening odors: pungent and decayed. Senses such as hearing and vision can be discussed in terms that most people understand and that are tied to measurable physical phenomena. But the sense of smell, or olfaction, has thus far not lent itself to such a systematic

understanding of what smells we perceive and how those perceptions relate to physical phenomena. "It's an open question how many fundamental types of odor qualities there are," says Castro. "This is in striking contrast to olfaction's 'sister sense,' taste, where we know that five basic qualities seem to organize sensations.

In ongoing work, the researchers are now approaching the problem from the other direction, applying the current research to a bank of chemical structures in an attempt to predict how a given chemical is going to smell. "That's something that nobody's really done with any kind of compelling accuracy," Castro says. "And obviously perfume companies, flavor and fragrance companies, are really interested in doing that well." He adds, "This study supports the idea that the world of smells is tightly structured, and organized by a handful of basic categories.



Properties of the perceptual basis set W. (A) Plot of normalized odor descriptor amplitude vs. odor descriptor number for the basis vector W1. Each point along the x-axis corresponds to a single odor descriptor, and the amplitude of each descriptor indicates the descriptor's relevance to the shown perceptual basis vector. Colored circles show the seven largest points in the basis vector, and descriptors corresponding to these points are listed to the right. (B) Waterfall plot of the 10 basis vectors constituting W, used in subsequent analyses. Note that each vector contains many values close to or equal to zero. **(Credit: Castro JB, Ramanathan A, Chennubhotla CS (2013) Categorical Dimensions of Human Odor Descriptor Space Revealed by Non-Negative Matrix Factorization. PLoS ONE 8(9): e73289. doi:10.1371/journal.pone.0073289, Creative Commons Attribution License)**

Jason B. Castro, Arvind Ramanathan, Chakra S. Chennubhotla. Categorical Dimensions of Human Odor Descriptor Space Revealed by Non-Negative Matrix Factorization. PLoS ONE, 2013; 8 (9): e73289 DOI: 10.1371/journal.pone.0073289

http://www.bbc.co.uk/news/health-24142695

Calls to give boys anti-cancer jab

Schoolboys should be given the HPV vaccine to help protect them from some cancers, according to public and sexual health bodies.

By James Gallagher Health and science reporter, BBC News

Human papillomavirus (HPV) is linked to a range of cancers and a vaccine is already given to girls in the UK to reduce the risk of cervical cancer.

The Faculty of Public Health and the British Association for Sexual Health and HIV said boys should be vaccinated. The Department of Health said there was no plan to extend the programme.

HPV infections are associated with cancer of the penis, vulva, vagina, anus, mouth and throat. It is spread by sexual contact. In the UK, girls aged 12-13 are offered the HPV jab. Australia is the only country to routinely offer the vaccination to boys and girls.

Prof John Ashton, the head of the Faculty of Public Health, told the BBC: "It seems oral sex has become a very common part of the repertoire in young people and it does seem a likely part of the story of increases in oral cancer.

"We really need to discuss oral sex as part of sex education in schools and to look closely at extending the vaccine to all men."

'Little benefit'

He said the reduced cancer risk would benefit all men, but the strongest case was in gay men.

Reducing the prevalence of the virus in women would have knock-on effects for some men, but not for those having sex only with other men.

Dr Janet Wilson, the president of the British Association for Sexual Health and HIV, said: "We need to take action to address the lack of protection men who have sex with men receive from the current all-girls HPV vaccination programme. "It is unfair that they remain unprotected."

However, a Department of Health official said there were "currently no plans to extend HPV vaccination to males, based on an assessment of currently available scientific evidence".

They added: "Vaccination of boys was not recommended by the Joint Committee on Vaccination and Immunisation because once 80% coverage among girls has been achieved, there is little benefit in vaccinating boys to prevent cervical cancer in girls."

http://www.bbc.co.uk/news/health-24142691

Alzheimer's brain scan detects tau protein

Pioneering brain imaging that can detect the build-up of destructive proteins linked to Alzheimer's has been developed by Japanese scientists.

By James Gallagher Health and science reporter, BBC News

It could lead to new ways of diagnosing the condition and of testing the effectiveness of new drugs.

The technology, reported in the journal Neuron, can identify inside a living brain clumps of a protein called tau that is closely linked to the disease. Alzheimer's Research UK said it was promising work.

Alzheimer's disease is a problem for researchers trying to come up with a cure. The brain starts to die years before any symptoms are detected, which means drugs are probably given too late. A diagnosis of Alzheimer's cannot be made with absolute certainty until a patient has died and their brain is examined. It is also not 100% clear what is the cause of the dementia and what are just symptoms. One protein, called tau, is very closely linked to the disease, with tangles of tau thought to be one way in which brain cells are killed.

The team, lead by the National Institute of Radiological Sciences in Chiba, used positron emission tomography to build a 3D picture of tau in the brain. They developed a chemical that could bind to tau and then be detected during a brain scan.

Tests on mice and people with suspected Alzheimer's showed the technology could detect tau.

Dr Makoto Higuchi, from the National Institute of Radiological Sciences in Japan, said: "Positron emission tomography images of tau accumulation... provide robust information on brain regions developing or at risk for tau-induced neuronal death."

The research is at an early stage, but it could eventually lead to an actual test for Alzheimer's disease.

It might also allow researchers to closely follow the impact drugs that affect tau have on the brain.

Another protein - beta amyloid - is also linked to Alzheimer's and can be detected in similar tests.

Dr Eric Karran, director of research at Alzheimer's Research UK, said: "This promising early study highlights a potential new method for detecting tau - a key player in both Alzheimer's and frontotemporal dementia - in the living brain. "With new drugs in development designed to target tau, scans capable of visualising the protein inside the brain could be important for assessing whether treatments in clinical trials are hitting their target. "If this method is shown to be effective, such a scan could also be a useful aid for providing people with an accurate diagnosis, as well as for monitoring disease progression."

http://www.sciencedaily.com/releases/2013/09/130918130504.htm

Interference With Cellular Recycling Leads to Cancer Growth, Chemo Resistance

Overactivity of a protein that normally cues cells to divide sabotages the body's natural cellular recycling process, leading to heightened cancer growth and chemotherapy resistance, UT Southwestern Medical Center researchers have found.

The epidermal growth factor receptor, or EGFR, is found at abnormally high levels on the surface of many types of cancer cells. The study, led by Dr. Beth Levine and published Sept. 12 in Cell, revealed that EGFR turns off autophagy, a process by which cells recycle unneeded parts, by binding to a protein, Beclin 1, which normally turns on the process. The researchers found that the deactivation of autophagy by EGFR led to more rapid tumor growth and chemotherapy resistance in mice implanted with non-small lung carcinoma cells.

"The fact that this type of cell surface receptor can directly interact with Beclin 1 and shut off autophagy provides fundamental insight into how certain oncogenes may cause cancer," said Dr. Levine, director of the Center for Autophagy Research and a Howard Hughes Medical Institute (HHMI) investigator at UT Southwestern. "Our findings suggest that inactivation of autophagy may be a critically important factor in the progression of lung cancer."

Earlier work in the laboratory of Dr. Levine identified beclin 1 as the first mammalian gene shown to function in autophagy. Defects in this gene may contribute not only to cancer, but also to aging, neurodegenerative diseases, and infectious diseases.

While the link between EGFR cell signaling action and cancer growth was known, with several pharmaceutical inhibitors of EGFR already on the market to combat cancer, exactly how this process worked was a mystery. This latest research uncovers Beclin 1 as one important way in which EGFR may derail the body's cancer-fighting autophagy machinery to increase tumor growth.

A second finding in the new study related to chemotherapy resistance. Several clinical trials are currently ongoing to test inhibitors of autophagy as a means of overcoming the resistance to chemotherapeutic drugs that many tumors develop. Unexpectedly, Dr. Levine's study found just the opposite: that autophagy inhibition may actually worsen chemotherapy outcomes for patients with specific cancer mutations. The researchers showed

that cancer cells with reduced autophagy grew faster and were more resistant to chemotherapy than cancer cells with normal autophagy. Dr. Levine noted that these findings may apply to many different types of cancers, especially those that rely on EGFR (or related signaling molecules) for their rapid growth.
About 10 percent of lung cancer patients have mutations in the EGFR oncogene, according to Dr. John Minna, one of the study authors and Director of the Nancy B. and Jack L. Hamon Center for Therapeutic Oncology Research and the W.A. "Tex" and Deborah Moncrief Jr. Center for Cancer Genetics at UT Southwestern. For those patients in particular, this finding could have significant impact in developing a personalized, targeted therapy.
"The EGFR protein is one of our most important targets for lung cancer therapy -- especially in patients whose tumors have certain EGFR gene mutations," Dr. Minna said. "We have oral medications that achieve dramatic clinical benefit and increase survival in this subset of patients, but even these successfully treated patients eventually become resistant to the treatment.
"These new findings are important for two reasons: First, they provide insight into how to extend EGFR-targeted therapy to a much larger group of lung cancer patients, including those whose tumors do not have mutations. Second, they provide a totally new approach to overcoming resistance to EGFR-targeted therapy."
Yongjie Wei, Zhongju Zou, Nils Becker, Matthew Anderson, Rhea Sumpter, Guanghua Xiao, Lisa Kinch, Prasad Koduru, Christhunesa S. Christudass, Robert W. Veltri, Nick V. Grishin, Michael Peyton, John Minna, Govind Bhagat, Beth Levine. EGFR-Mediated Beclin 1 Phosphorylation in Autophagy Suppression, Tumor Progression, and Tumor Chemoresistance. Cell, 2013; 154 (6): 1269 DOI: 10.1016/j.cell.2013.08.015

http://bit.ly/16iZL9O

Colorado Rainfall Was One for the Ages, Weather Service Says

The downpour that inundated parts of Colorado this month was a once-in-a-millennium event for those areas, according to an analysis by the National Weather Service.

By Alex Dobuzinskis

(Reuters) - The downpour that inundated parts of Colorado this month was a once-in-a-millennium event for those areas, according to an analysis by the National Weather Service.
Colorado residents are coping with widespread destruction from floods unleashed by torrential rains that began on September 9 and lasted for several days. The flooding killed at least eight people, forced thousands from their homes and caused nearly $2 billion in property damage.
Towns at the base of Colorado's so-called Front Range in Larimer and Boulder counties, northwest of Denver, experienced the most damage.
"As it kept raining and kept raining and kept raining, this thing kept getting more and more rare, in terms that we use for evaluating," said Geoffrey Bonnin, chief of the Hydrologic Science and Modeling Branch of the National Weather Service.
To date, monthly rainfall for September in Boulder, Colorado, totaled 17.2 inches, the most for any month since official recordkeeping began in 1893, said Byron Louis, program manager for the National Weather Service in Colorado.
Rain in that area fell nearly continuously from September 9 to September 15, Louis said.
That amount of rainfall around Boulder is likely to occur less than once every 1,000 years, according to the weather service.
Two swaths of land experienced the once-in-a-millennium rainfall. One area extended south of Boulder to more than 40 miles north in the region of Estes Park, according to a map from the National Weather Service.
The second area that was swamped extended from the Denver suburb of Aurora to lands about 40 miles north, the map showed.
"There was quite a bit of area there that really got clobbered," Bonnin said.
The extent of flooding seen in Colorado has drawn comparisons to a 1976 flood in the state that killed nearly 150 people along the Big Thompson Canyon near Loveland.
Geologists have not been able to measure how the volume of floodwater that struck parts of Colorado this month compared to the flooding in 1976 because two key gauges along the Big Thompson River were swept away, said Robert Kimbrough, hydrologist for the U.S. Geological Survey in Denver.
"It's really frustrating both gauges were taken out. That's an indicator of how large an event this was," Kimbrough said.
The U.S. Geological Survey will dispatch teams of spotters to measure the high water mark from this month's flooding, which will allow the agency to determine how the flooding compares to past disasters, he said.
(Reporting by Alex Dobuzinskis; Editing by Cynthia Johnston and Stacey Joyce)

http://scitechdaily.com/genomic-test-accurately-distinguishes-viral-bacterial-infections/

Genomic Test Accurately Distinguishes between Viral and Bacterial Infections

A new genomic test developed by researchers at Duke University School of Medicine can accurately sort between viral and bacterial infections, possibly helping to limit the overuse of antibiotics and the emergence of bacterial resistance.

A blood test developed by researchers at Duke Medicine showed more than 90-percent accuracy in distinguishing between viral and bacterial infections when tested in people with respiratory illnesses.

The test, which detects a specific genetic "signature" that the sick person's immune system expresses as a response to the virus, demonstrates a potential new method for diagnosing the source of illnesses that have long been tough to pinpoint.

Reported in the Sept. 18, 2013, issue of the journal Science Translational Medicine, the finding moves the technology closer to clinical use, where it could help patients get quicker diagnoses and treatments, while curbing the unnecessary use of antibiotics that don't work on viral infections.

"In instances such as pandemic flu or the corona-virus that has erupted in the Middle East, it's extremely important to diagnose a viral illness far more accurately and speedier than can be done using traditional diagnostics," said co-senior author Geoffrey S. Ginsburg, M.D., Ph.D., director of Genomic Medicine and professor of medicine at Duke University School of Medicine. "Current tests require knowledge of the pathogen to confirm infection, because they are strain-specific. But our test could be used right away when a new, unknown pathogen emerges."

When infected by a virus, a person's immune system responds differently than when fighting a bacterial infection. These differences are evident at the genetic level, where certain genes are switched on during a viral attack, creating a fingerprint that broadly identifies the culpable pathogen.

In previous work, the Duke team described the development of a blood test, using a special assay, to identify some 30 genes involved in the immune response to viral infection among volunteers who had agreed to be infected with a series of common upper respiratory viruses. Unlike current tests that rely on evidence of the pathogen in the blood stream – requiring knowledge of that particular bug to detect it – the new approach could be used to detect unknown emerging diseases, including potential bioterrorism threats.

"This is important not only in viral pandemics where infection may be caused by unknown viruses but also in routine care where the decision to treat or not with antibiotics is paramount," said lead author Aimee K. Zaas, M.D., MHS, associate professor of infectious diseases and international health at Duke.

The current study was a trial run of the blood test in a "real-world" setting. Among 102 people arriving at a hospital's emergency department with fever, 28 had a viral infection, 39 had a bacterial infection and 35 were healthy controls. Using the test, the Duke researchers were able to accurately classify more than 90 percent of the patients as having viral infection or not. The assay provided true positive identifications of viral infection in 89 percent of the cases, and correctly ruled out the negative cases 94 percent of the time.

The researchers said larger studies are planned, and additional work is ongoing to trim the amount of time it takes for the test results to be reported. Ginsburg said the test currently takes 12 hours, and analyzes about 30 genes. He said both the time and the number of genes could be pared.

"We were very pleased that the assay could pick out those with viral infection with a high degree of accuracy," Zaas said. "This is perhaps the most important aspect of this effort – the accuracy of the new test in a real-world setting. It is a major step forward in the test becoming a useful diagnostic to help physicians and patients."

Co-senior author Christopher W. Woods, M.D., MPH, associate professor of medicine, pathology and global health at Duke, the Durham VA Medical Center and the Duke Global Health Institute, said the new test, if proven successful in additional studies, could help resolve some of the most pressing issues around infectious diseases.

"One of the big global threats at the moment is the emergence of bacterial resistance, and that is largely driven by overuse of antibiotics," Woods said. "This is a growing public health threat, creating infections that are increasingly difficult to manage. A tool that enables us to accurately identify viral infections could curb the indiscriminate use of antibiotics and reduce the development of resistant pathogens."

In addition to Ginsburg, Zaas and Woods, study authors include Thomas Burke, Minhua Chen, Micah McClain, Bradly Nicholson, Timothy Veldman, Ephraim L. Tsalik, Vance Fowler, Emanuel P. Rivers, Ronny Otero, Stephen F. Kingsmore, Deepak Voora, Joseph Lucas, Alfred O. Hero, and Lawrence Carin.

The study was supported in part by the Defense Advanced Research Projects Agency, the National Institutes of Allergy and Infectious Diseases (AI066569), and the Department of Veterans Affairs.

Ginsburg, Zaas, Woods, Hero, Carin and Lucas have filed for a provisional patent on the respiratory viral signature. Fuller disclosures are provided in the study.

Publication: A Host-Based RT-PCR Gene Expression Signature to Identify Acute Respiratory Viral Infection," Science Translational Medicine, 18 September 2013: Vol. 5, Issue 203, p. 203ra126; DOI: 10.1126/scitranslmed.3006280

http://www.medscape.com/viewarticle/811284?src=rss

Gastric Bypass 'Cures' Diabetes in Almost a Third of Patients

Publication of a new, albeit retrospective, study has shown that almost a third of obese patients with type 2 diabetes undergoing gastric bypass were effectively "cured" of their diabetes, being in complete remission as per the strictest definition possible that was maintained for 6 years after the surgery.

Lisa Nainggolan

The researchers also found that patients undergoing such surgery significantly reduced their cardiovascular risk factors according to the Framingham Risk Score and that diabetic nephropathy either improved or was completely resolved; the latter is "remarkable," said the surgeon who led the study, Stacy A. Brethauer, MD, from the Cleveland Clinic, Ohio.

Of the patients, "27% had complete remission maintained for 5 years, and that is the operational definition of a 'cure' by [American Diabetes Association] ADA standards. Hopefully this will help people understand that the effects we see after these procedures are durable. Even in the patients who had some of their diabetes come back, the disease came back milder; we have changed the trajectory," he told Medscape Medical News

Dr. Brethauer first reported the results of the study at the American Surgical Association meeting in Indianapolis in April, and they have been published online today in the Annals of Surgery. His colleague, Philip Schauer, MD, also from the Cleveland Clinic, presented the findings at the Prediabetes and the Metabolic Syndrome 2013 Congress in Vienna, Austria around the same time.

At the time of the presentation at the surgery meeting, the study generated many comments and questions, which are included, together with Dr. Brethauer's responses, in the article.

One noted that this study adds to the findings of STAMPEDE, a randomized controlled trial also carried out at the Cleveland Clinic: "Today's paper adds even more proof. Not only does it work, but it works 5 years later. It is not totally new. We reported good results at 10 years. The Swedish colleagues reported at 20 years. But your paper was far more elegant and far more detailed," observed Walter J. Pories, MD, from East Carolina University, Greenville, North Carolina. Why, then, he wonders, are there still "cries for more evidence, more evidence?"

Dr. Brethauer replies that the surgical community must continue "to provide data to support the concept that this is a surgically treated disease. It is a major paradigm shift for our endocrinology colleagues to accept. And I think it is going to require time and a new generation of endocrinologists before they fully embrace this."

Talking of Cure is "Controversial and Provocative"

Dr. Brethauer explained to Medscape Medical News that while his study is not unique in reporting longer-term outcomes, it is one of the few to have used the strictest definition of remission, as per ADA criteria, "that a patient has to have HbA1c of 6% or less, normal fasting blood glucose [<100 mg/dL], and be completely off diabetes medications for one year."

In their study, the Cleveland Clinic researchers went back and examined the clinical outcomes of 217 patients with type 2 diabetes who underwent bariatric surgery between 2004 and 2007 and had at least 5 years of follow-up. The majority of patients (n = 162) underwent Roux-en-Y gastric bypass (RYGB), with the remainder undergoing gastric banding (n = 32) or sleeve gastrectomy (n = 23).

At a median follow-up of 6 years (range, 5–9 years), a mean excess weight loss of 55% was associated with mean reductions in HbA1c from 7.5% to 6.5% ($P < .001$) and fasting blood glucose (FBG) from 155.9 mg/dL to 114.8 mg/dL ($P < .001$).

Long-term complete remission, as per the ADA criteria, occurred in 24% of patients, and partial remission (HbA1c 6%–6.4%, FBG of 100–125 mg/dL for 1 year in the absence of antidiabetic medications) was observed in 26% of patients. In addition, a further 34% of patients improved their long-term diabetes control compared with presurgery status. There were 16% of patients who remained unchanged.

When only the RYGB patients were considered, 31% of patients achieved complete remission; 27% of bypass patients continuously sustained this for more than 5 years, the ADA definition of a "cure," the researchers note, although Dr. Brethauer observed that the use of this term with respect to type 2 diabetes "is still quite controversial and somewhat provocative."

Realistic Expectations; Diabetes Recurs but Legacy Effect

Dr. Brethauer and colleagues go on to say that it's now obvious that the remission rates first seen in short-term studies of bariatric surgery, approaching 80%, are not sustained long term. For example, the 2-year diabetes remission rate seen in the Swedish Obese Subjects (SOS) study of 72% declined to a 36% remission rate after 10 years.

However, what must be appreciated, they say, is that despite recurrences - and "some would consider the recurrence of type 2 diabetes as a failure" - "our data and others must be measured against the known risks of poorly controlled diabetes in patients who do not undergo bariatric surgery."
Even experiencing remission for a few years or simply an improvement in diabetes should yield significant reductions in terms of micro- and macrovascular end points, a "legacy effect," similar to that seen with tight glycemic control in type 2 diabetes in the United Kingdom Prospective Diabetes Study (UKPDS) or with type 1 diabetes in Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Study (DCCT/EDIC), they note.
For example, in the subgroup of patients with diabetic nephropathy at baseline in the current study, the condition "either improved or was completely resolved," noted Dr. Brethauer, with 53% of patients seeing regression, 47% remaining stable, and no cases of progression.
And long-term control of cardiovascular risk factors in the study exceeded that of the general population, he points out, with 62% meeting blood-pressure goals after surgery and 72% meeting LDL-cholesterol goals (compared with figures of 51% and 56% in the National Health and Nutrition Examination Survey [NHANES]), for example.
SOS has already demonstrated a reduction in all-cause mortality, cardiovascular deaths, and first-time cardiovascular events after bariatric surgery, he noted, and among those with type 2 diabetes in that trial, "there was a significant reduction in the number of myocardial infarctions at 13 years in the surgery group compared with the standard-treatment group."

Predictors of Remission; Gastric Bypass the Best Tool

His team also found, consistent with other studies, that shorter duration of type 2 diabetes is associated with a higher rate of remission after surgery, as is greater long-term weight loss. But it did not show that poor preoperative glycemic control and insulin use were predictors of remission, as has other research. However, this was probably because relatively few participants had very poor glycemic control in this study, he noted.
All said, the findings indicate that gastric bypass "should be considered as an earlier treatment option for patients with uncontrolled type 2 diabetes," he urged.
And the current results also support prior findings that "gastric bypass is the best metabolic and diabetes operation we can offer," giving the best chance of diabetes remission, he concluded.
The authors have reported no relevant financial relationships. Ann Surg. 2013;258:628-637.

http://www.sciencedaily.com/releases/2013/09/130919140624.htm

Considering a Three-Year Medical Degree

Academic medical centers can help adapt to dramatic changes in health care by offering accelerated study to selected students so that they receive a doctor of medicine degree (MD) in three years rather than the traditional four, according to educational leaders at NYU School of Medicine.

In a Perspective essay in this week's New England Journal of Medicine (NEJM), the educators describe how a three-year medical degree, a radical rethinking of medical education, can increase the number of productive years clinicians and physician scientists spend in the workforce, as well as reduce student debt.
The educators argue that for highly qualified applicants, the three-year degree can be an effective counterbalance to ever more extensive training periods, which now average 10 years for some sub-specialties, without sacrificing quality. NYU School of Medicine began offering a three-year medical degree pathway for selected students this year.
"This article presents a compelling argument that a more effective medical education process is needed," said lead author Steven B. Abramson, MD, vice dean for education, faculty, and academic affairs and chair of the Department of Medicine at NYU School of Medicine. "We are hopeful that this article will encourage continued discussions focused on restructuring medical education to meet the changing health care needs of the population." In addition to Dr. Abramson, authors include Robert I. Grossman, MD, dean and CEO, NYU Langone Medical Center, and Dianna Jacob, RPA, MBA, vice president, Faculty and Academic Affairs.
"A four-year program for all graduates made sense when postgraduate training lasted two or three years. Now, residencies and fellowships routinely extend the postgraduate period to six years or more, which means that many physicians don't enter practice until their early or mid-30s," says Dr. Abramson. "Indeed, data from the American Medical Association show that since 1975, the percentage of physicians under the age of 35 has decreased from 28 percent to 15 percent."
The essay describes how it has been more than 100 years since Abraham Flexner proposed the current model for medical education in North America -- two years of basic science instruction followed by two years of clinical experience. Cutting the average duration of medical training, they note, by approximately 30 percent -- partly by eliminating one year of medical school -- can be accomplished without compromising physicians'

competence or the quality of care provided. While the three-year pathway can also reduce student debt by 25 percent, the educators acknowledge the challenges that a three-year pathway poses and say that shortening medical school education is "just one approach to address the need for change in the Post-Flexnerian era." "We are at a point of inflection whereby alignment of training across the levels of medical education with regulatory standards and our health care delivery system is critical to train physicians and physician scientists who are passionate care providers across specialties," said Robert I. Grossman, MD, dean and CEO at NYU Langone. "In the years ahead, developing a uniform set of milestones and competencies that assess physicians throughout medical school, residency, fellowship and ultimately clinical practice will be a major task for medical educators."

Steven B. Abramson, Dianna Jacob, Melvin Rosenfeld, Lynn Buckvar-Keltz, Victoria Harnik, Fritz Francois, Rafael Rivera, Mary Ann Hopkins, Marc Triola, Robert I. Grossman. A 3-Year M.D. - Accelerating Careers, Diminishing Debt. New England Journal of Medicine, 2013; 369 (12): 1085 DOI: 10.1056/NEJMp1304681

http://bit.ly/1aW8HjQ

How Many Die from Medical Mistakes in U.S. Hospitals?

An updated estimate says it could be at least 210,000 patients a year, more than twice the number in a frequently quoted Institute of Medicine report

By Marshall Allen and ProPublica | Friday, September 20, 2013 | 6

It seems that every time researchers estimate how often a medical mistake contributes to a hospital patient's death, the numbers come out worse.

In 1999, the Institute of Medicine published the famous "To Err Is Human" report, which dropped a bombshell on the medical community by reporting that up to 98,000 people a year die because of mistakes in hospitals. The number was initially disputed, but is now widely accepted by doctors and hospital officials 2014 and quoted ubiquitously in the media.

In 2010, the Office of Inspector General for Health and Human Services said that bad hospital care contributed to the deaths of 180,000 patients in Medicare alone in a given year.

Now comes a study in the current issue of the Journal of Patient Safety that says the numbers may be much higher 2014 between 210,000 and 440,000 patients each year who go to the hospital for care suffer some type of preventable harm that contributes to their death, the study says.

That would make medical errors the third-leading cause of death in America, behind heart disease, which is the first, and cancer, which is second.

The new estimates were developed by John T. James, a toxicologist at NASA's space center in Houston who runs an advocacy organization called Patient Safety America. James has also written a book about the death of his 19-year-old son after what James maintains was negligent hospital care.

Asked about the higher estimates, a spokesman for the American Hospital Association said the group has more confidence in the IOM's estimate of 98,000 deaths. ProPublica asked three prominent patient safety researchers to review James' study, however, and all said his methods and findings were credible.

What's the right number? Nobody knows for sure. There's never been an actual count of how many patients experience preventable harm. So we're left with approximations, which are imperfect in part because of inaccuracies in medical records and the reluctance of some providers to report mistakes.

Patient safety experts say measuring the problem is nonetheless important because estimates bring awareness and research dollars to a major public health problem that persists despite decades of improvement efforts. "We need to get a sense of the magnitude of this," James said in an interview.

James based his estimates on the findings of four recent studies that identified preventable harm suffered by patients 2013 known as "adverse events" in the medical vernacular 2013 using use a screening method called the Global Trigger Tool, which guides reviewers through medical records, searching for signs of infection, injury or error. Medical records flagged during the initial screening are reviewed by a doctor, who determines the extent of the harm.

In the four studies, which examined records of more than 4,200 patients hospitalized between 2002 and 2008, researchers found serious adverse events in as many as 21 percent of cases reviewed and rates of lethal adverse events as high as 1.4 percent of cases.

By combining the findings and extrapolating across 34 million hospitalizations in 2007, James concluded that preventable errors contribute to the deaths of 210,000 hospital patients annually.

That is the baseline. The actual number more than doubles, James reasoned, because the trigger tool doesn't catch errors in which treatment should have been provided but wasn't, because it's known that medical records are missing some evidence of harm, and because diagnostic errors aren't captured.

An estimate of 440,000 deaths from care in hospitals "is roughly one-sixth of all deaths that occur in the United States each year," James wrote in his study. He also cited other research that's shown hospital reporting systems and peer-review capture only a fraction of patient harm or negligent care.
"Perhaps it is time for a national patient bill of rights for hospitalized patients," James wrote. "All evidence points to the need for much more patient involvement in identifying harmful events and participating in rigorous follow-up investigations to identify root causes."
Dr. Lucian Leape, a Harvard pediatrician who is referred to the "father of patient safety," was on the committee that wrote the "To Err Is Human" report. He told ProPublica that he has confidence in the four studies and the estimate by James.
Members of the Institute of Medicine committee knew at the time that their estimate of medical errors was low, he said. "It was based on a rather crude method compared to what we do now," Leape said. Plus, medicine has become much more complex in recent decades, which leads to more mistakes, he said.
Dr. David Classen, one of the leading developers of the Global Trigger Tool, said the James study is a sound use of the tool and a "great contribution." He said it's important to update the numbers from the "To Err Is Human" report because in addition to the obvious suffering, preventable harm leads to enormous financial costs.
Dr. Marty Makary, a surgeon at The Johns Hopkins Hospital whose book "Unaccountable" calls for greater transparency in health care, said the James estimate shows that eliminating medical errors must become a national priority. He said it's also important to increase the awareness of the potential of unintended consequences when doctors perform procedure and tests. The risk of harm needs to be factored into conversations with patients, he said.
Leape, Classen and Makary all said it's time to stop citing the 98,000 number.
Still, hospital association spokesman Akin Demehin said the group is sticking with the Institute of Medicine's estimate. Demehin said the IOM figure is based on a larger sampling of medical charts and that there's no consensus the Global Trigger Tool can be used to make a nationwide estimate. He said the tool is better suited for use in individual hospitals.
The AHA is not attempting to come up with its own estimate, Demehin said.
Dr. David Mayer, the vice president of quality and safety at Maryland-based MedStar Health, said people can make arguments about how many patient deaths are hastened by poor hospital care, but that's not really the point. All the estimates, even on the low end, expose a crisis, he said. "Way too many people are being harmed by unintentional medical error," Mayer said, "and it needs to be corrected."

http://phys.org/news/2013-09-neandertals-modern-humans-specialized-bone.html

Research finds Neandertals, not modern humans, made first specialized bone tools in Europe

Bone fragment turns out to be a part of an early specialized bone tool used by a Neandertal before the first modern humans appeared in Europe

One day in 2011, undergraduate student Naomi Martisius was sorting through tiny bone remnants in the University of California, Davis, paleoanthropology lab when she stumbled across a peculiar piece.
The bone fragment, from a French archaeological site, turned out to be a part of an early specialized bone tool used by a Neandertal before the first modern humans appeared in Europe.
"At the time, I had no idea about the impact of my discovery," said Martisius, who is now pursuing her doctoral degree in anthropology at UC Davis.
Martisius' opportunity was the result of a decade of excavation and research by two international teams. Their findings were published in the Proceedings of the National Academy of Science in August.
"Previously these types of bone tools have only been associated with modern humans," said Teresa E. Steele, associate professor of anthropology at UC Davis, who also served as a co-author on the article and adviser to Martisius at UC Davis and at archaeological excavations in France.
"However, our identification of these pieces in secure Neandertal contexts leaves open the possibility that we have found, for the first time, evidence that Neandertals may have influenced the technology of modern humans," she said.
Three bone tools (lissoirs) were recently discovered at the Neandertal excavation site at Abri Peyrony, France. Used to smooth tough animal hides, the tools were made about 50,000 years ago by Neandertals—not just the humans who came after them, as researchers had earlier theorized. The specialized tools are still used today, in similar form, to smooth and refine leather made into high-end purses and jackets.
The bone tools were found in deposits containing typical Neandertal stone tools and the bones of hunted animals including reindeer, red deer and bison. Three of the four pieces were from the site of Abri Peyrony,

France. The animal bones from that site had been exported to UC Davis for analysis in Steele's lab where Martisius worked with her to study the material.
Now in her second year of doctoral studies at UC Davis, Martisius will carry on her research of these pieces. She, Steele and their colleagues will use resources available at UC Davis to conduct experimental studies to manufacture—and use—new, similar animal bone tools for comparison.
Using sophisticated imaging techniques, Martisius will examine the pieces made by the Neandertals, comparing those with the ones first made by the first modern humans in Europe and the ones she manufactures at UC Davis. She said she also will look at animal bones from nearby sites to see if she can identify additional pieces made by Neandertals.
The tools described in their current work were recovered in archaeological sites in the French countryside that had been explored for more than 100 years, but modern archaeological techniques enabled researchers to recognize these smaller pieces now identified as pieces of once-sophisticated tools, Steele said.
The article, "Neandertals made the first specialized bone tools in Europe," is available online.
More information: *www.pnas.org/content/110/35/14186.full*

http://www.eurekalert.org/pub_releases/2013-09/uoo-oe091813.php

'Cascade of events' caused sudden explosion of animal life

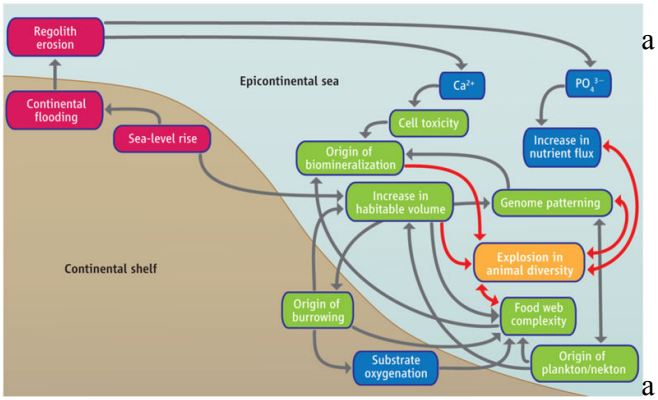
The explosion of animal life on Earth around 520 million years ago was the result of a combination of interlinked factors rather than a single underlying cause, according to a new study.

Dozens of individual theories have been put forward over the past few decades for this rapid diversification of animal species in the early Cambrian period of geological time.
But a paper by Professor Paul Smith of Oxford University and Professor David Harper of Durham University suggests a more holistic approach is required to discover the reasons behind what has become known as the Cambrian Explosion.
Theories for the Cambrian Explosion fall into three main categories – geological, geochemical and biological – and most have been claimed as standalone processes that were the main cause of the explosion.
Whatever the cause, this major evolutionary event led to a wide range of biological innovation, including the origin of modern ecosystems, a rapid increase in animal diversity, the origin of skeletons and the first appearance of specialist modes of life such as burrowing and swimming.
Among the weird and wonderful creatures to emerge in the early Cambrian was Anomalocaris, the free-swimming, metre-long top predator of the time with a mouth composed of 32 overlapping plates that could constrict to crush prey. It is distantly related to modern arthropods, including crabs and lobsters.
Vertebrate animals also made their first appearance in the Cambrian Explosion, the distant ancestors of modern fish, reptiles, birds and mammals.
Professor Smith, Professor Harper and a team of scientists have spent four years working on data from a site in northernmost Greenland, facing the Arctic Ocean.
The site, at Siriuspasset, is located at 83°N, just 500 miles from the North Pole in a remote part of north Greenland. Although logistically very difficult to reach, Siriuspasset attracted the team because of the high quality of its fossil material and the insights it provides.
Professor Smith and Professor Harper's findings are published in the latest edition of the journal Science.
Professor Smith, lead author of the report and Director of the Oxford University Museum of Natural History, said: 'This is a period of time that has attracted a lot of attention because it is when animals appear very abruptly in the fossil record, and in great diversity. Out of this event came nearly all of the major groups of animals that we recognise today.
'Because it is such a major biological event, it has attracted much opinion and speculation about its cause.'
Described by the researchers as a 'cascade of events', the interacting causes behind the explosion in animal life are likely to have begun with an early Cambrian sea level rise. This generated a large increase in the area of habitable seafloor, which in turn drove an increase in animal diversity. These early events then translate into the complex interaction of biological, geochemical and geological processes described in individual hypotheses.
Professor Harper, Professor of Palaeontology in the Department of Earth Sciences at Durham University, said: 'The Cambrian Explosion is one of the most important events in the history of life on our planet, establishing animals as the most visible part of the planet's marine ecosystems.
'It would be naïve to think that any one cause ignited this phenomenal explosion of animal life. Rather, a chain reaction involving a number of biological and geological drivers kicked into gear, escalating the planet's diversity during a relatively short interval of deep time.

'The Cambrian Explosion set the scene for much of the subsequent marine life that built on cascading and nested feedback loops, linking the organisms and their environment, that first developed some 520 million years ago.'

Professor Smith said: 'Work at the Siriuspasset site in north Greenland has cemented our thinking that it wasn't a matter of saying one hypothesis is right and one is wrong. Rather than focusing on one single cause, we should be looking at the interaction of a number of different mechanisms.

'Most of the hypotheses have at least a kernel of truth, but each is insufficient to have been the single cause of the Cambrian explosion. What we need to do now is focus on the sequence of interconnected events and the way they related to each other – the initial geological triggers that led to the geochemical effects, followed by a range of biological processes.'



Interconnected causes. Earth system, developmental, and ecological processes have been hypothesized as isolated, singular causes of the major diversification of marine taxa early in the Cambrian. Instead, many of these processes sit within a series of cascading and nested feedback loops that together generated the Cambrian explosion. Each box corresponds broadly to a stand-alone hypothesis or suite of related hypotheses (red, geological; blue, geochemical; green, biological). The figure represents a narrow interval of time at the beginning of the Cambrian (541 million to 521 million years ago). **M P Smith, and D A T Harper Science 2013;341:1355-1356**

The paper 'Causes of the Cambrian Explosion' by M. Paul Smith and David A. T. Harper is published in Science, Vol. 341, 20 September 2013. It will be available to view online at http://www.sciencemag.org/lookup/doi/10.1126/science.1239450
The research was funded by the Agouron Institute, the Carlsberg Foundation and Geocenter Danmark.

http://www.eurekalert.org/pub_releases/2013-09/jhm-cdf091913.php

Could dog food additive prevent disabling chemotherapy side effect?

Johns Hopkins researchers find, in mice, that common preservative may thwart pain and damage of peripheral neuropathy

Working with cells in test tubes and in mice, researchers at Johns Hopkins have discovered that a chemical commonly used as a dog food preservative may prevent the kind of painful nerve damage found in the hands and feet of four out of five cancer patients taking the chemotherapy drug Taxol. The Food and Drug Administration-approved preservative, an antioxidant called ethoxyquin, was shown in experiments to bind to certain cell proteins in a way that limits their exposure to the damaging effects of Taxol, the researchers say. The hope, they say, is to build on the protective effect of ethoxyquin's chemistry and develop a drug that could be given to cancer patients before taking Taxol, in much the same way that anti-nausea medication is given to stave off the nausea that commonly accompanies chemotherapy. While half of Taxol users recover from the pain damage, known as peripheral neuropathy, the other half continue to have often debilitating pain, numbness and tingling for the rest of their lives.

"Millions of people with breast cancer, ovarian cancer and other solid tumors get Taxol to treat their cancer and 80 percent of them will get peripheral neuropathy as a result," says Ahmet Höke, M.D., Ph.D., a professor of neurology and neuroscience at the Johns Hopkins University School of Medicine and director of the Neuromuscular Division. "They're living longer thanks to the chemotherapy, but they are often miserable. Our goal is to prevent them from getting neuropathy in the first place."

A report on Höke's research is published online in the Annals of Neurology.

Höke and his team knew from previous experiments that adding Taxol to a nerve cell line growing in a petri dish would cause neurodegeneration. In a series of experiments, they set out to hunt for compounds that might interrupt the degenerative process by adding Taxol to nerve cells along with some 2,000 chemicals - one at a time - to see which, if any, could do that.

Ethoxyquin did so, Höke says, apparently by making the cells resistant to the toxic effects of the Taxol.

Once they identified ethoxyquin's effects, they gave intravenous Taxol to mice, and saw nerves in their paws degenerate in a couple of weeks. But when they gave ethoxyquin to the mice at the same time as the Taxol, it prevented two-thirds of the nerve degeneration, which Höke says would have a big impact on quality of life if the same effects were to occur in humans.

Specifically, Höke and his team discovered that molecules of ethoxyquin were binding to Hsp90, one of the so-called heat shock proteins that cells defensively make more of whenever they are stressed. Hsp90 acts as a cell's

quality control officer, determining whether a protein is properly formed before sending it out where it is needed. When ethoxyquin binds to Hsp90, two other proteins - ataxin-2 and Sf3b2 - can't bind to Hsp90. When they can't bind, the cell senses that these two proteins are flawed, so they are degraded and their levels in the cell diminished.

Höke says his team is not certain why too much of those two proteins appears to have a negative effect on nerves, but reducing their levels clearly appears in their studies to make cells more resistant to Taxol toxicity.

Höke and his colleagues are looking into whether this medication could also make nerves more resistant to damage in peripheral neuropathy caused by HIV and diabetes, two other major causes of the pain. A previous study, Höke says, showed that ataxin-2 may cause degeneration in motor neurons in a rare form of ALS, commonly known as Lou Gehrig's disease, suggesting that ethoxyquin or some version of it might also benefit people with this disorder.

Twenty to 30 million Americans suffer from peripheral neuropathy. Höke says it's a "huge public health issue" that doesn't get much attention because it is not fatal.

Höke's team is hoping to conduct safety studies with ethoxyquin in animals in advance of possible testing in people. He says that while too much ethoxyquin is thought to be potentially harmful to dogs, the needed dose for humans would likely be 20-to-30-fold lower than what is found in dog food. Ethoxyquin was developed in the 1950s as an antioxidant, a compound to prevent pears and other foods from becoming discolored and spoiling.

Other Johns Hopkins researchers involved in the study include Jing Zhu, Ph.D.; Weiran Chen, M.D.; Ruifa Mi, M.D., Ph.D.; Chunhua Zhou, B.S.; and Nicole Reed, M.S.

The research was supported by the Foundation for Peripheral Neuropathy, Dr. Miriam and Sheldon G. Adelson Medical Research Foundation, the National Institutes of Health's National Institute of Neurological Disorders and Stroke (R01 NS43991) and Johns Hopkins Brain Science Institute.

http://www.eurekalert.org/pub_releases/2013-09/tos-frd091913.php

First real-time detector for IV delivered drugs may help eliminate life-threatening medical errors

New optical device can identify the contents of the fluid in an intravenous (IV) line in real-time

WASHINGTON -Today, computerized smart systems can deliver drugs intravenously in exact volumes to hospital patients. However, these systems cannot recognize which medications are in the tubing nor can they determine the concentration of the drug in the tubing. This lack of precise information can lead to medication errors with serious consequences.

Now, a new optical device developed by a team of electrical and computer engineering students at the University of Illinois at Urbana-Champaign (UIUC) can identify the contents of the fluid in an intravenous (IV) line in real-time, offering a promising way to improve the safety of IV drug delivery. The team, led by Prof. Brian T. Cunningham, interim director of the Micro and Nanotechnology Laboratory at UIUC, will present its work at The Optical Society's (OSA) Annual Meeting, Frontiers in Optics (FiO) 2013, being held Oct. 6-10 in Orlando, Fla.

The vulnerability of IV drug-delivery systems due to human error is a chief concern in hospital safety, Cunningham said. Errors can include incorrect dosage, unintentional substitution of one drug for another, and co-delivery of incompatible drugs.

"Up to 61 percent of all life-threatening errors during hospitalization are associated with IV drug therapy," Cunningham said, citing a recent report. "So for all the really good things hospitals can do, the data shows that mistakes can occasionally happen."

To approach this problem, Cunningham and colleagues turned to the very small – to structures and processes at the nanoscale (one-billionth of a meter), where novel physical and chemical properties arise. The researchers use a technology called Surface-Enhanced Raman Scattering (SERS), a powerful analytical tool prized for its extreme sensitivity in obtaining molecular signals that can be used to identify chemicals. To determine the identity of a particular IV medication, researchers shine laser light onto a nanostructured gold surface that contains millions of tiny "nano-domes" that are separated from each other by as little as 10 nanometers. The nano-domes are incorporated into the inner surface of IV tubing, where they are exposed to drugs that are dispersed in liquid. They capture the light scattered from drug molecules that are in contact with the nano-domes and use SERS to determine the drug's molecular signature. Finally, they match the signature to known signatures for the drug in order to confirm the presence of a specific medication in the IV line.

While other groups have demonstrated excellent nanostructured surfaces for SERS, those developed by the Cunningham group are unique because they are inexpensively produced on flexible plastic surfaces by a replica molding process with nanometer scale accuracy.

Early data show that the Cunningham group's system can identify medications including morphine, methadone, phenobarbital, the sedative promethazine, and mitoxantrone, which is used to treat multiple sclerosis. The system is extremely sensitive: it can detect drugs in amounts 100 times lower than the clinically delivered drug concentrations commonly used. So far, the researchers have also shown their system can sense a two-drug combination, which has its own unique signature.
The next step is further evaluation for combinations of up to ten drugs being delivered at the same time. Computer algorithms are also being developed to automatically interpret the SERS spectra, and Cunningham's team reports that the system is now being evaluated for possible commercialization.
Presentation LTh3H.4 "Enhanced Contrast in Chemical and Biological Sensors" takes place Thursday, Oct. 10 at 1:30 p.m. EDT at the Hilton Bonnet Creek.

http://www.sciencedaily.com/releases/2013/09/130919201256.htm

Paracetamol Improves Exercise Endurance in the Heat

Paracetamol has a significant effect on exercise performance and the body's ability to cope with the thermal challenge of exercise in the heat, shows a study published in Experimental Physiology.

The research team have previously shown that paracetamol can improve endurance performance through a reduction in exercise-induced pain. This study suggests, for the first time, that paracetamol can also improve the length of time someone can exercise for in hot conditions. The data suggests that this is achieved by reducing the body's temperature during exercise, which subsequently improves their tolerance to exercise in the heat.
To perform the research, a group of healthy, male participants ingested single doses of paracetamol or a placebo, before cycling at a fixed intensity for as long as they could in hot conditions. During the exercise, measures of core and skin temperature were recorded alongside the participants' perception of the heat.
As the study was performed in humans, with a common over-the counter drug, the implications are far reaching. Dr Lex Mauger, who led the study at The University of Kent's School of Sport and Exercise Sciences, explains: "Firstly, consideration by the World Anti-Doping Agency (WADA) and local anti-doping authorities should be made about the use of non-steroidal anti-inflammatory drugs (NSAIDs) in sport -- on both health and performance grounds. Secondly, the utility of paracetamol as a first-response drug to exertional heat illness should be investigated."
The research gives a new insight into the effects of paracetamol on endurance exercise, and further studies hope to determine by which mechanisms this takes place.
Dr Mauger says: "Whilst we have found that paracetamol improves the time someone can exercise in the heat, and that this occurs alongside a reduced body temperature, we did not measure the specific mechanisms by which this may have occurred. It is important now to try and isolate how paracetamol reduced participants' body temperature during exercise."
Alexis R Mauger, Lee Taylor, Christopher Harding, Ben Wright, Josh Foster, and Paul Castle. Acute acetaminophen (paracetamol) ingestion improves time to exhaustion during exercise in the heat. Experimental Physiology, September 2013 DOI: 10.1113/expphysiol.2013.075275

http://www.eurekalert.org/pub_releases/2013-09/kp-dua091913.php

Digoxin use associated with higher risk of death for patients diagnosed with heart failure

Digoxin was associated with a 72 percent higher rate of death among adults with newly diagnosed systolic heart failure

OAKLAND, Calif - Digoxin, a drug commonly used to treat heart conditions, was associated with a 72 percent higher rate of death among adults with newly diagnosed systolic heart failure, according to a Kaiser Permanente study that appears in the current online issue of Circulation: Cardiovascular Quality and Outcomes.
Digoxin is a drug derived from digitalis, a plant that has been used for more than 200 years to treat heart failure. "These findings suggest that the use of digoxin should be reevaluated for the treatment of systolic heart failure in contemporary clinical practice" said Alan S. Go, MD, senior author of the study and research scientist at the Kaiser Permanente Division of Research.
The results of this study contrast with the findings of a randomized trial by the Digitalis Investigation Group conducted between 1991 and 1993, which showed that digoxin did not lower mortality among therapy patients with systolic heart failure, or, a malfunction in the way the left ventricle of the heart pumps blood. Following the group's study, professional societies issued clinical guidelines endorsing the use of digoxin for patients with systolic dysfunction.
The current study was conducted among 2,891 adults within Kaiser Permanente in Northern California who had newly diagnosed systolic heart failure between 2006 and 2008 and no prior digoxin use. Eighteen percent of the participants initiated digoxin during the study period.

Researchers followed the patients through December 31, 2010, to evaluate the effectiveness and safety of digoxin therapy. They found that digoxin use was associated with higher mortality but no significant difference in the risk of heart failure hospitalization.
There were a total of 801 deaths (737 off digoxin and 64 on digoxin). After adjustment for potential confounders, digoxin use was associated with a 72 percent higher relative rate of death.
There were 1,723 hospitalizations for heart failure overall (1,596 off digoxin, 127 on digoxin). However, after adjustment for potential confounders, digoxin use was not significantly associated with hospitalization for heart failure.
"Our community-based study population is more likely to represent patients with systolic heart failure in the modern era with regard to pathogenesis and treatment patterns," Dr. Go said. "Therefore, our results may more accurately represent the outcomes expected with digoxin for patients with systolic heart failure in typical present-day practices. As with all medication, treatment, or therapy plans, care decisions should always be made by physicians and their patients working together, with the patient's particular care needs and goals in mind."
Other authors on the study include James V. Freeman, MD, MPH of Yale University School of Medicine; Mark A. Hlatky, MD of the Stanford University School of Medicine; and Jingrong Yang, MA, and Sue Hee Sung, MPH, of the Division of Research, Kaiser Permanente Northern California.

http://www.sciencedaily.com/releases/2013/09/130920143322.htm

Don't Let Fear of Falling Freeze You in Your Tracks

Older adults need to maintain their strength, function and activity to the level they are able.

A Saint Louis University School of Nursing faculty member is going to mark the first day of fall with a simple warning to senior adults: Don't let fear of falling stand in the way of being active and engaged with the world around you.
Helen Lach, Ph.D., associate professor of nursing at SLU, serves on the executive lead team of the Missouri Show Me Falls Free Coalition, a group that works to prevent falls. She specializes in gerontological nursing, and has studied ways to prevent falls for more than 20 years.
"While falls can cause problems, we want people to be both cautious and still maintain an active quality of life," Lach said. "You can't get rid of all of the risk in your life. But older adults need to maintain their strength, function and activity to the level they are able."
Lach most recently wrote a review article that appeared in JAMDA (Journal of the American Medical Directors Association) that showed fear of falling is a significant problem in nursing homes.
"People in nursing homes tend to be frailer and have more health problems and physical limitations than older adults who are in the community," Lach said.
"The fear of falling can stop some nursing home residents from doing anything, even participating in their own daily care. They become frozen in inactivity, which makes them depressed and bored. They get more out of shape, which creates more health problems that actually increase their risk of falling."
Lach noted that the fear of falling is part of a cycle that can lead to a frailty and a downward spiral in health.
"As people do less, they become less able to engage in activities. They have difficulty moving around, and their gait and balance deteriorates. This puts them at an increased risk of falling, which unfortunately means the fear of falling actually becomes a self-fulfilling prophesy."
It's important that nursing home staff members recognize that about half of residents have such a deep fear of falling that they limit their activities, and develop a way to assuage those fears. Exercise programs offered in a safe and supportive environment can be valuable in helping residents feel better – both physically and psychologically, Lach said.
Senior adults who aren't in long term care facilities also may need to confront their fear of falling, she added. Tai Chi, walking, weight training and simple exercises to increase muscle strength – such as practicing sitting and standing to strengthen leg muscles or standing on one foot with a chair at arm's reach – make a world of difference.
"Falls can cause problems," Lach said, "but so can the fear of falling."
Founded in 1928, Saint Louis University School of Nursing has achieved a national reputation for its innovative and pioneering programs. Offering bachelor's, master's, and doctoral nursing programs, its faculty members are nationally recognized for their teaching, research and clinical expertise.
Helen W. Lach, Jill L. Parsons. Impact of Fear of Falling in Long Term Care: An Integrative Review. Journal of the American Medical Directors Association, 2013; 14 (8): 573 DOI: 10.1016/j.jamda.2013.02.019

http://wapo.st/1bsqOAw

Weight bias in cancer care? Obese cancer patients often shorted on chemo, hurting survival

Obese people are less likely to survive cancer, and one reason may be a surprising inequality: The overweight are undertreated.

Doctors often short them on chemotherapy by not basing the dose on size, as they should. They use ideal weight or cap the dose out of fear about how much treatment an obese patient can bear. Yet research shows that bigger people handle chemo better than smaller people do.

Even a little less chemo can mean worse odds of survival, and studies suggest that as many as 40 percent of obese cancer patients have been getting less than 85 percent of the right dose for their size.

Now, the largest organization of doctors who treat cancer, the American Society of Clinical Oncology, aims to change that. The group has adopted guidelines urging full, weight-based doses for the obese.

Don't call it supersizing; it's right-sizing cancer care, said Dr. Gary Lyman, a Duke University oncologist who led the panel that wrote the advice. "There's little doubt that some degree of undertreatment is contributing to the higher mortality and recurrence rates in obese patients," he said.

The Food and Drug Administration's cancer drug chief, Dr. Richard Pazdur, agrees. "By minimizing the dose, or capping the dose, we have been undertreating patients," he said.

The dosing issue applies to all types of cancer treated with chemo - breast, colon, lung, ovarian and even blood diseases such as leukemia. It affects a lot of people. Big isn't healthy but it's the new "normal" - 60 percent of Americans are overweight and more than one-third of them are obese.

Giving too little chemo "could make it as if they didn't even get treated at all ... so they go through the whole ordeal with no benefit, in the extreme case ," said Dr. Jennifer Griggs, a University of Michigan breast cancer specialist who also worked on the guidelines. So why do doctors limit dose?

Sometimes it's for good reason - the patient has diabetes, heart problems or other illnesses that interfere with how much chemo they can stand. Usually, though, it's because doctors are afraid to follow a standard weight-based formula because the dose seems so huge and they're afraid of harming the heart and blood system, Lyman said.

"You're three times the size of the average person, but it doesn't mean your heart is," Griggs explained.

Yet studies show that heavier patients are less likely to develop dangerous, low blood counts from cancer treatment, and that they clear chemo drugs more quickly from the body than thinner people do.

A paper Lyman published in the journal Nature in August said that a 20 percent reduction in chemo doses lowered remission and cure rates by half in animal experiments and helped the tumors develop resistance to the drugs. Other research in people found lower survival among those getting less chemo as well.

Even if a patient develops a problem from a chemo treatment and doctors have to dial it back, it's important to try a full dose the next time around so the patient gets all the treatment intended, Lyman said.

That happened to Tracy Smith, a 46-year-old Durham, N.C., woman treated at Duke in 2011 for breast cancer that had spread to more than a dozen lymph nodes. Doctors gave her full chemo doses based on her weight, which at 285 pounds classified her as obese. Three times, high fevers put her in the hospital, and one treatment was cut short because doctors thought it was causing wheezing and possible lung damage. But she resumed and finished the intended treatment and has been cancer-free since then. After hearing you have cancer, "you're just kind of in a fog" and don't think to ask about doses of the drugs you need, she said. "I trusted my doctor. Doctors should be well aware of what you can tolerate. You should do whatever you can to fight this beast."

Smith's tumor was fueled by estrogen - a hormone made in abundance by fat tissue. Robin McRath, a floral designer who helps run a women's shelter in Ludington, Mich., had the same type.

"It's like a playground, an amusement park, for cancer cells when you're fat," she said. She was only was 41 when her cancer was diagnosed five years ago, and her oncologist, Dr. Carol Peterson, treated her with full doses based on her weight - about 240 pounds, which put her in the obese category.

"We didn't discuss dosage. That didn't matter to me - I just wanted to get it out of my system," she said of the cancer, and praised the treatments to prevent one of chemo's most feared side effects. "There are fantastic anti-nausea medicines. I was never sick one day."

McRath is active in the Obesity Action Coalition, an education and advocacy group. A spokesman said the group was unaware of the dosing issue for obese patients.

Not all doctors are aware either. Luckily for McRath, hers was. Peterson said she uses full doses unless a patient has other health issues. "If that's their only problem - if they're just overweight or obese - they can do quite well" with full weight-based doses, she said.

Duke's Lyman agreed, and offered this advice to patients: "Ask your doctor how they plan to treat you and whether you're going to get the full dosing. The doctor may have a good reason not to, but you should have that discussion." *AP National Writer Allen G. Breed in Raleigh, N.C., contributed to this report.*

http://www.eurekalert.org/pub_releases/2013-09/icl-sct092013.php

Scientists closer to universal flu vaccine after pandemic 'natural experiment'

Scientists have moved closer to developing a universal flu vaccine after using the 2009 pandemic as a natural experiment to study why some people seem to resist severe illness.

Researchers at Imperial College London asked volunteers to donate blood samples just as the swine flu pandemic was getting underway and report any symptoms they experienced over the next two flu seasons.
They found that those who avoided severe illness had more CD8 T cells, a type of virus-killing immune cell, in their blood at the start of the pandemic.
They believe a vaccine that stimulates the body to produce more of these cells could be effective at preventing flu viruses, including new strains that cross into humans from birds and pigs, from causing serious disease.
The findings are published in Nature Medicine.
Professor Ajit Lalvani from the National Heart and Lung Institute at Imperial College London, who led the study, said: "New strains of flu are continuously emerging, some of which are deadly, and so the Holy Grail is to create a universal vaccine that would be effective against all strains of flu."
Today's flu vaccines make the immune system produce antibodies that recognise structures on the surface of the virus to prevent infection with the most prevalent circulating strains. But they are usually one step behind as they have to be changed each year as new viruses with different surface structures evolve.
Previously, experimental models had suggested that T cells may protect against flu symptoms but until now this idea has not been tested in humans during a pandemic.
Professor Lalvani's team rapidly recruited 342 staff and students at Imperial to take part in their study in autumn 2009. The volunteers donated blood samples and were given nasal swabs. They were sent emails every three weeks asking them to fill in a survey about their health. If they experienced flu symptoms, they took a nasal swab and sent it back to the lab.
They found that those who fell more severely ill with flu had fewer CD8 T cells in their blood, and those who caught flu but had no symptoms or only mild symptoms had more of these cells.
Professor Lalvani said, "The immune system produces these CD8 T cells in response to usual seasonal flu. Unlike antibodies, they target the core of the virus, which doesn't change, even in new pandemic strains. The 2009 pandemic provided a unique natural experiment to test whether T cells could recognise, and protect us against, new strains that we haven't encountered before and to which we lack antibodies.
"Our findings suggest that by making the body produce more of this specific type of CD8 T cell, you can protect people against symptomatic illness. This provides the blueprint for developing a universal flu vaccine.
"We already know how to stimulate the immune system to make CD8 T cells by vaccination. Now that we know these T cells may protect, we can design a vaccine to prevent people getting symptoms and transmitting infection to others. This could curb seasonal flu annually and protect people against future pandemics."
Professor Lalvani is a Wellcome Trust Senior Research Fellow in Clinical Science and a National Institute for Health Research Senior Investigator. Other members of the team received support from Imperial College Healthcare NHS Trust, the Medical Research Council and Public Health England.
1. S Sridhar et al. 'Cellular immune correlates of protection against symptomatic pandemic influenza.' Nature Medicine 2013.

http://bit.ly/18OaEAu

Wild Animals of All Stripes Are Adapting to the Cityscape and Thriving

The new science of urban ecology reveals a surprising trend of wildlife adapting to the cityscape

By Jesse Greenspan

Cities are often viewed as environmental wastelands, where only the hardiest of species can eke out an existence. But as scientists in the fledgling field of urban ecology have found, more and more native animals are now adjusting to life on the streets.
Take America's biggest metropolis. As recently as a few decades ago, New York City lacked white-tailed deer, coyotes and wild turkeys, all of which have now established footholds. Harbor seals, herons, peregrine falcons and ospreys have likewise returned in force, and red-tailed hawks have become much more common.
Meanwhile the first beaver in more than two centuries turned up in 2007; river otters last year ended a similar exile.
What's happening in New York is by no means an anomaly. Experts say that the adaptation of wildlife to urban areas is ramping up worldwide, in part because cities are turning greener, thanks to pollution controls and an increased emphasis on open space.

In North America, the phenomenon is perhaps best exemplified by the coyote, which colonized cities roughly 15 to 20 years ago. A recent study of the Chicago area found that urban pups had survival rates five times higher than their rural counterparts. “Coyotes can absolutely exist in even the most heavily urbanized part of the city, without a problem,” says Stan Gehrt, a wildlife ecologist at Ohio State University. “They learn the traffic patterns, and they learn how stoplights work.”

Other studies have found animals from hawks to opossums reaping benefits from urban life. “We need to be careful about thinking of cities as places that don't really have interesting biodiversity,” says Seth Magle, director of the Urban Wildlife Institute at the Lincoln Park Zoo in Chicago. “Our urban areas are ecosystems, with just as many complex interactions as the Serengeti or the outback of Australia.”

http://www.eurekalert.org/pub_releases/2013-09/uom-wdy092013.php

Why do you want to eat the baby?

'Odour is a means of chemical communication between mother and child' -- Johannes Frasnelli, University of Montreal

What woman has not wanted to gobble up a baby placed in her arms, even if the baby is not hers? This reaction, which everyone has noticed or felt, could have biological underpinnings related to maternal functions. For the first time, an international team of researchers has found evidence of this phenomenon in the neural networks associated with reward. "The olfactory -- thus non-verbal and non-visual -- chemical signals for communication between mother and child are intense," explains Johannes Frasnelli, a postdoctoral researcher and lecturer at the University of Montreal's Department of Psychology. "What we have shown for the first time is that the odour of newborns, which is part of these signals, activates the neurological reward circuit in mothers. These circuits may especially be activated when you eat while being very hungry, but also in a craving addict receiving his drug. It is in fact the sating of desire."

Reward circuit

For their experiment, the researchers presented two groups of 15 women with the odours of others' newborns while the women were subjected to brain imaging tests. The first group was composed of women who had given birth 3-6 weeks prior to the experiment, and the other group consisted of women who had never given birth. All the women were non-smokers. The odours of the newborns were collected from their pyjamas two days after birth.

Although the women in both groups perceived the odour of newborns with the same intensity, brain imaging showed greater activation in the dopaminergic system of the caudate nucleus of mothers compared to the women who had never given birth. Located in the centre of the brain, the caudate nucleus is a double structure straddling the thalamus in both hemispheres. "This structure plays a role in reward learning," explains Frasnelli. "And dopamine is the primary neurotransmitter in the neural reward circuit."

This system reinforces the motivation to act in a certain way because of the pleasure associated with a given behaviour. "This circuit makes us desire certain foods and causes addiction to tobacco and other drugs," says the researcher. "Not all odours trigger this reaction. Only those associated with reward, such as food or satisfying a desire, cause this activation."

Dopamine is also associated with sexual pleasure and other forms of gratification. Laboratory rats whose dopamine levels are stimulated by electrodes become so addicted that they stop eating.

For the research team, these results show that the odour of newborns undoubtedly plays a role in the development of motivational and emotional responses between mother and child by eliciting maternal care functions such as breastfeeding and protection. The mother-child bond that is part of the feeling of maternal love is a product of evolution through natural selection in an environment where such a bond is essential for the newborn's survival.

Questions remain

The experiment, however, did not allow determining whether the greater activation of the dopaminergic system in mothers is due to an organic response related to childbirth itself or whether it is a consequence of the olfactory experience developed by mothers with their own babies. "It is possible that childbirth causes hormonal changes that alter the reward circuit in the caudate nucleus, but it is also possible that experience plays a role," says Frasnelli.

It is also not known whether this reaction is specific to mothers, since men were not part of the experiment. "What we know now and what is new is that there is a neural response linked to the status of biological mother," he says.

The University of Montreal is officially known as Université de Montréal. The experiment was conducted at the Department of Obstetrics at the Technical University of Dresden, Germany, and Johannes Frasnelli participated in the study design and data analysis. Other researchers from France, Sweden, and the United States also participated. The results are published in the

September 5, 2013 issue of the journal Frontiers in Psychology. This document is a translation from a text originally written in French by Daniel Baril, Université de Montréal.

http://www.eurekalert.org/pub_releases/2013-09/afps-drd092013.php

Disaster relief donations track number of people killed, not number of survivors

People pay more attention to the number of people killed in a natural disaster than to the number of survivors when deciding how much money to donate to disaster relief efforts

People pay more attention to the number of people killed in a natural disaster than to the number of survivors when deciding how much money to donate to disaster relief efforts, according to new research published in Psychological Science, a journal of the Association for Psychological Science. The donation bias can be reversed, however, with a simple change in terminology.

"While fatalities have a severe impact on the afflicted community or country, disaster aid should be allocated to people affected by the disaster – those who are injured, homeless, or hungry," says lead researcher Ioannis Evangelidis of Rotterdam School of Management, Erasmus University (RSM) in the Netherlands. "Our research shows that donors tend not to consider who really receives the aid."

This discrepancy leads to a "humanitarian disaster," say Evangelidis and colleague Bram Van den Bergh, where money is given disproportionately toward the natural disasters with the most deaths, instead of the ones with the most people in desperate need of help.

The researchers began by examining humanitarian relief data for natural disasters occurring between 2000 and 2010. As they expected, they found that the number of fatalities predicted the probability of donation, as well as the amount donated, by private donors in various disasters. Their model estimated that about $9,300 was donated per person killed in a given disaster. The number of people affected in the disasters, on the other hand, appeared to have no influence on the amount donated to relief efforts.

Evangelidis and Van den Bergh believe that donors are more likely to pay attention to a death toll when deciding how much to give because the term "affected" is ambiguous. In many cases, though, fatalities don't correlate with the number of actual people in need.

To find a way to combat this donation bias, the researchers brought participants into the laboratory and presented them with several scenarios, involving various types of disasters and different numbers of people killed and affected.

Overall, participants allocated more money when a disaster resulted in a high death toll – even when the number of people affected was low – mirroring the data from the real natural disasters.

The bias was reversed, however, when participants had to compare two earthquakes – one that killed 4,500 and affected 7,500 versus one that claimed 7,500 and affected 4,500 – before allocating funds.

The act of comparing the two disasters seems to have forced the participants to think critically about which group actually needed the aid more. Notably, the effect carried over when the participants were asked to allocate funds for a third disaster

But the easiest, and most realistic, way to reduce the donation bias may involve a simple change in terminology. When the researchers swapped the term "affected" with the much less ambiguous term "homeless," participants believed that money should be allocated according to the number of homeless people following a disaster.

"Above all, attention should be diverted from the number of fatalities to the number of survivors in need," Evangelidis and Van den Bergh conclude. "We are optimistic that these insights will enhance aid to victims of future disasters."

For more information about this study, please contact: Ioannis Evangelidis at ievangelidis@rsm.nl.
The article abstract is available online: http://pss.sagepub.com/content/early/2013/09/19/0956797613490748.abstract
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