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Penn anesthesiologists identify top 5 practices that could be avoided

Study supports 'choosing wisely' campaign to eliminate wasteful health care practices

PHILADELPHIA – A team of researchers led by Penn Medicine anesthesiologists have pinpointed the "top five" most common perioperative procedures that are supported by the least amount of clinical evidence, in an effort to direct providers to make more cost-effective treatment decisions. Their findings are published in the current issue of JAMA Internal Medicine.

The team surveyed anesthesiologists, many of them in academic practice, to identify the most common activities that should be questioned in the field, using practice parameters developed by the American Society of Anesthesiology (ASA) and other perioperative guidelines. Criteria for inclusion were common clinical practices that may be tied to poorer quality of care or increased costs, those for which there is little or no benefit to patients and that could easily be ceased in practice. Items were restricted to common preoperative and intraoperative practices, with the exclusion of postoperative and pain services.

The "Choosing Wisely" campaign is an ongoing effort by the American Board of Internal Medicine Foundation to help physicians become better stewards of health care resources. The Physician's Charter, a similar initiative, was issued in 2002 and outlined the physician's responsibilities to ensure access to high quality care by practicing evidence-based medicine, cost-effectively, and maintaining trust by minimizing conflicts of interest. This initiative was adopted by the ASA and more than 130 other organizations.

"The elimination of low-value services in low-risk patients represents a substantial savings, but we needed some consensus from our peers as to what the top least-valued services and procedures were," said the study's lead author, Onyi C. Onuoha, MD, MPH, assistant professor of Anesthesiology and Critical Care.

The researchers surveyed their peers on 18 items. First, practicing academic anesthesiologists affiliated with the Society of Academic Anesthesiology Associations responded, narrowing the list to 11 items. The list was then disseminated to the Association of University Anesthesiologists and to a subset of ASA members.

Respondents were overwhelmingly male, from academic practice settings, in practice for more than 20 years and working with the leadership of the ASA.

Survey respondents were asked to report their feedback on each clinical item across five domains, including frequency in practice, impact on quality of care, impact on cost of care, evidence supporting the activity, and the ease in implementation of avoidance.

The resulting top five activities the authors recommend questioning included:

Baseline laboratory studies in healthy patients without significant systemic disease when blood loss during surgery is expected to be minimal

Baseline cardiac testing or cardiac stress test in asymptomatic stable patients with known cardiac disease undergoing low-risk or moderate-risk noncardiac surgery

Routine use of PAC (pulmonary artery catheter) for cardiac surgery in patients with a low risk of blood pressure complications, especially if other diagnostic tools like an echocardiogram are being used during the procedure

Administration of blood in young healthy patients without ongoing blood loss and with a low-normal hemoglobin concentration, unless they show symptoms or blood pressure problems

Routine administration of specific types of intravenous fluids for replacement of blood or other fluids without appropriate indications.

These recommendations were then submitted to the Choosing Wisely campaign.

"We need buy-in from the other disciplines we work with, including the patient, primary care physician and surgeon, to implement many of these changes and still provide optimal preoperative and perioperative care. There is a lot of concern among those surveyed about attempting to implement these changes in isolation," says the study's senior author, Lee Fleisher, MD, chair of the Department of Anesthesiology and Critical Care. "Still, we believe that spreading awareness and encouraging physicians of varying disciplines to 'Choose Wisely' in delivering care is the first step in instituting change."

http://www.eurekalert.org/pub_releases/2014-06/waeh-gr061614.php

Gene 'switch' reverses cancer in common childhood leukaemia

Melbourne researchers have shown a type of leukaemia can be successfully 'reversed' by coaxing the cancer cells back into normal development.

The discovery was made using a model of B-progenitor acute lymphoblastic leukaemia (B-ALL), the most common cancer affecting children.

Researchers from the Walter and Eliza Hall Institute showed that switching off a gene called Pax5 could cause cancer in a model of B-ALL, while restoring its function could 'cure' the disease.

Institute researchers Dr Ross Dickins and Ms Grace Liu led the study with institute colleagues and collaborators in Vienna. The study was published today in the journal Genes & Development.

Ms Liu said the team used a newly developed 'genetic switch' technology to inhibit then reactivate Pax5 in the leukaemia model.

"Along with other genetic changes, deactivating Pax5 drives normal blood cells to turn into leukaemia cells, which has been shown before," Ms Liu said. "However

we showed for the first time that reactivating Pax5 enabled the cells to resume their normal development and lose their cancer-like qualities, effectively curing the leukaemia. What was intriguing for us was that simply restoring Pax5 was enough to normalise these cancer cells, despite the other genetic changes."

In leukaemia, immature white blood cells replicate abnormally and build up in the bone marrow, interfering with production of normal blood cells.

Ms Liu said Pax5 was a gene frequently 'lost' in childhood B-ALL. "Pax5 is essential for normal development of a type of white blood cells called B cells," she said. "When Pax5 function is compromised, developing B cells can get trapped in an immature state and become cancerous. We have shown that restoring Pax5 function, even in cells that have already become cancerous, removes this 'block', and enables the cells to develop into normal white blood cells."

Dr Dickins said the research shed light on the function of Pax5, which was one of about 100 genes known to 'suppress' human tumours. "When these tumour suppressor genes are inactivated by changes to the DNA, cancers start to develop," Dr Dickins said.

"This work shows how inactivating the tumour suppressor gene Pax5 contributes to B-ALL development and how leukaemia cells become 'addicted' to low Pax5 levels to continue proliferating. Even though the B-ALL cells have multiple genetic mutations, simply reactivating Pax5 causes tumour cells to resume normal development and lose their cancerous properties."

Dr Dickins said forcing B-ALL cells to resume their normal development could provide a new strategy for treating leukaemia. "While B-ALL has a relatively good prognosis compared with other cancers, current treatments can last years and have major side-effects. By understanding how specific genetic changes drive B-ALL, it may be possible to develop more specific treatments that act faster with fewer side-effects."

However Dr Dickins said that genes that are lost in tumour cells are not traditionally drug targets. "It is very difficult to develop drugs that restore the function of genes that are lost during cancer development," Dr Dickins said. "However by understanding the mechanisms by which Pax5 loss causes leukaemia, we can begin to look at ways of developing drugs that could have the same effect as restoring Pax5 function."

The genetic switch technology used to study Pax5 could also be used to understand 'tumour suppressor' genes in other cancers, he said.

The work was supported by the Australian National Health and Medical Research Council, the Leukaemia Foundation of Australia, the Sylvia and Charles Viertel Charitable Foundation, VESKI and the Victorian Government. The Walter and Eliza Hall Institute is a partner in the Victorian Comprehensive Cancer Centre.

http://www.eurekalert.org/pub_releases/2014-06/uom-vad061614.php

Vitamin A derivative potentially treats type 2 diabetes and prevents its complications

Potential of retinoic acid demonstrated in treating obesity and type 2 diabetes and preventing their cardiovascular complications

At a time when obesity, type 2 diabetes, and their complications are a veritable epidemic worldwide, researchers at the University of Montreal and CHUM Research Centre (CRCHUM) recently demonstrated the potential of retinoic acid (RA), a derivative of Vitamin A, in treating obesity and type 2 diabetes and preventing their cardiovascular complications. The findings were presented June 6, 2014 at the Annual Conference of the Canadian Nutrition Society in Saint John's, Newfoundland.

"In obese and insulin resistant mice, retinoic acid reduces the risk of cardiac apoptosis, stimulates the expression of cardio-protective genes reduced by the disease, and protects against the accumulation of collagen in the cardiac muscle, thus avoiding the occurrence of fibrosis and possible associated future complications," says the first author of the study, Daniel-Constantin Manolescu.

Apoptosis is the process of programmed cell death. The discovery follows other research conducted by his team on the effects of RA on insulin resistance, diabetes, and obesity. "Blood glucose, insulin resistance, body weight, and adipocyte size were significantly decreased in treated animals, including abdominal fat, while dietary intake and physical activity were similar for treated or non-treated animals. This suggests an increase in basal energy expenditure," says Manolescu.

White fat is an energy reserve formed by the accumulation of fat in the form of triglycerides to meet unexpected increases in energy costs, but it is also a hormonal tissue with a delicate balance. Overall, if energy intake is greater than expenditures over an extended period, obesity increases, while hormonal balance and energy metabolism are disturbed. In this context, resistance to type 2 diabetes develops over time.

Brown fat also stores triglycerides, but it has the ability to produce heat. Abundant in babies, brown fat does not disappear completely in adulthood. It is irrigated by blood vessels and has many mitochondria, the energy factories of cells. Vitamin A derivatives stimulate a mitochondrial uncoupling protein (UCP1) that allows uncoupling of the mitochondria pathway (oxidative phosphorylation), which uses energy from the oxidation of nutrients for the production of adenosine triphosphate (ATP). For a period of time, they generate heat (thermogenesis) instead of ATP, which is traditionally the energy required for active metabolism. Exposure to cold leads to the stimulation of brown fat and white fat, promoting the conversion of

triglycerides to free fatty acids and glycerol. However, in brown adipocytes, these fatty acids are rapidly oxidized in the mitochondria and produce heat (under the influence of the UCP1 protein). Brown fat thus helps to increase basal energy metabolism. As a result, hibernating mammals fatten in the fall without developing diabetes and lose weight without moving too much in the winter (while warming their den). They are also the animals that accumulate the most Vitamin A in their livers. Retinoic acid (a Vitamin A derivative) is recognized for its involvement in cell maturation and differentiation and may guide pre-adipocytes to become brown (or beige) instead of white. It is as if "boilers" were installed directly in reserves of white fat to melt it on the spot and prevent it from over-accumulating.

"Vitamine A is a bioactive nutrient. The originality of our project is in addressing obesity and type 2 diabetes through the involvement of retinoids. We have attracted international attention and were named among 12 teams in the world to bring conclusive data in this regard," says Dr. Pangala V. Bhat.

"Our studies on animals show that retinoic acid induces normalization of blood glucose and reduction of obesity. It is an important contribution to understanding RA action on the liver, fat, muscles, and the heart, and on retinoid metabolism, energy metabolism, fatty acid oxidation, and insulin resistance. Our research identifies new metabolic effects of retinoids and may lead to anti-obesity and anti-diabetic medicines," says Dr. Jean-Louis Chiasson.

1) "Natriuretic peptide and other cardio-protective genes are stimulated by Vitamin A (retinoid acid), preventing apoptosis and fibrosis in obese-diabetic mice heart" (Manolescu et al., 2014). Collaboration between the laboratoires of Dr. Jean-Louis Chiasson and Dr. Jolanta Gutkowska, CRCHUM.http://www.nrcresearchpress.com/doi/abs/10.1139/apnm-2014-0005

2) "All-trans retinoic acid lowers serum retinol-binding protein 4 concentrations and increases insulin sensitivity in diabetic mice" (Manolescu et al. 2010). http://www.ncbi.nlm.nih.gov/pubmed/20032483

http://www.eurekalert.org/pub_releases/2014-06/uoc--sfe061214.php

Survey finds e-cigarette online market on fire

First comprehensive survey of e-cigarettes underscores the complexity in regulating the rapidly growing market

Researchers at the University of California, San Diego School of Medicine have completed the first comprehensive survey of e-cigarettes for sale online and the results, they believe, underscore the complexity in regulating the rapidly growing market for the electronic nicotine delivery devices.

The survey, published in a June 16 special supplement of the journal Tobacco Control, found that 10 new e-cigarette brands entered the Internet marketplace every month, on average, from 2012 to 2014, and that there are currently 466 e-cigarette brands online, offering more than 7,700 flavors, including candy flavors such as gummy bear and marshmallow, that may appeal to children. In contrast, traditional cigarettes sold in the United States can be marketed in just two flavors: tobacco and menthol.

The scientists also documented a shift in the marketing of e-cigarettes, with newer brands selling customizable e-cigarettes that might look nothing like an old-fashioned tobacco cigarette. For example, some resemble pens, flashlights, even a violin.

The marketing messages also change with the products: Older brands were more likely to claim that e-cigarettes were healthier or cheaper than smoking, or that e-cigarettes could help people quit smoking. Newer brands are less inclined to making these claims. Instead, their marketing is focused on consumer choice, such as flavors or models.

"It almost seems that newer brands don't want to be compared to cigarettes, which are associated with the image of cancer" said lead author Shu-Hong Zhu, PhD, professor of Family and Preventive Medicine and director of the Center for Research and Interventions in Tobacco Control at UC San Diego.

Smoking-related diseases are the leading cause of preventable death worldwide, estimated to be responsible for 6 million deaths annually. Although smoking rates among American adults have declined by more than half, from 42 percent in 1965 to 18 percent in 2012, tobacco use in the United States is still responsible for nearly one in five deaths, according to the American Cancer Society. E-cigarettes vaporize nicotine, the addictive ingredient in tobacco products. They first became available in the U.S. in 2007 and have since stoked controversy about whether they improve or worsen public health by reducing tobacco cigarette consumption or provide an alternative way to consume nicotine.

"Some consider them promising products to help smokers quit traditional cigarettes, while others believe they will re-normalize smoking, which will keep more people smoking," Zhu said.

Although the authors support the Food and Drug Administration's proposed rules that e-cigarette companies be required to list ingredients and nicotine strengths, follow good manufacturing practices to ensure product safety, require child proof e-liquid containers and ban sale to minors, they caution that overly stringent regulations could have unintended consequences. Regulations would likely favor brands with strong financial backing and most of these would be owned by tobacco companies.

"Obviously, tobacco companies would be more concerned with protecting cigarette market share than smaller e-cigarette companies," Zhu said.

Too many regulations run the risk of changing only the market share of different e-cigarette brands rather than reducing the prevalence of smoking, he said. The most

important goal in e-cigarette regulation should be to reduce the number of people smoking cigarettes. Cigarettes are the most deadly tobacco product, killing half its users.

Co-authors include Jessica Sun, Erika Bonnevie, Sharon Cummins, Anthony Gamst, Lu Yin and Madeleine Lee, UC San Diego Moores Cancer Center.

Funding for the study came from the National Cancer Institute under the State and Community Tobacco Control Initiative (grant UO1-CA154280).

http://www.eurekalert.org/pub_releases/2014-06/ifor-efl061614.php

E-cigarettes far less harmful than cigarettes, says researcher at INFORMS Conference

Rates alcohol most dangerous drug to public, heroin worst for individuals

A London School of Economics researcher examining the public and private dangers of drugs argues against demonizing e-cigarettes in a presentation being given at a conference of the Institute for Operations Research and the Management Sciences (INFORMS). He also calls on public officials to recognize that alcohol causes greater harm than other recreational drugs and more public attention should be paid to controlling its harmful effects.

Lawrence D. Phillips, an emeritus professor at the London School of Economics, will present his research group's findings about the relative risks of different drugs at Advances in Decision Analysis, a conference sponsored by the INFORMS Decision Analysis Society (DAS). The conference takes place June 16-18 at Georgetown University in Washington, DC.

A recent workshop facilitated by Prof. Phillips led a group of researchers to write a letter to the World Health Organization advocating against the classification of e-cigarettes as tobacco. They argued that e-cigarettes should be classified as a device for fighting nicotine addiction. . " It is well known that 'people smoke for the nicotine, but die from the smoke'," he says.

In his upcoming presentation, Prof. Philips draws on a study about drugs in the United Kingdom he co-authored in 2010 that was published in The Lancet. "Drug Harms in the UK: A Multi-Criteria Analysis" has lessons that can be applied in the U.S. and across the world, he says. The 2010 results are based on an expert panel that was called upon to use participants' judgment to assess the relative harm of 20 different drugs. Because the drugs are illegal and data is extremely difficult to obtain, the participants relied on their collective knowledge and experience to score the drugs and a decision analysis model to aggregate the judgments.

Similar results were obtained in 2013 among a group assessing drug risk in Europe. A .993 correlation between the two panels, which contained different sets of experts, is considered extremely high.

A 2013 expert panel about the relative harm of 12 nicotine products named cigarettes the most harmful but ranked e-cigarettes near the bottom, in ninth place. Prof. Phillips explores its results in his presentation at Georgetown University.

The various panels used decision analysis to determine psychological, physical and social harm to users and to those around them. Members of the United Kingdom's Independent Scientific Committee on Drugs scored 20 drugs on 16 criteria.

Decision analysis of the UK panel ratings showed that heroin, crack cocaine, and methamphetamine were the most harmful drugs to individuals (scoring 34, 37, and 32, respectively). Alcohol, heroin, and crack cocaine were the most harmful to others (46, 21, and 17).

Overall, alcohol was the most harmful drug (overall harm score 72), with heroin (55) and crack cocaine (54) in second and third places. Similar results were found when analyzing the continental Europe panel.

Several experts from the drug harm workshop, including The Lancet study's co-author David Nutt, the Edmund J Safra Professor of Neuropsychopharmacology and Head of the Department of Neuropsychopharmacology and Molecular Imaging at Imperial College London, have called on British and international health organizations like WHO to adjust their guidelines about dangerous drugs based on the findings.

In the 2010 paper, they write, "the present drug classification systems have little relation to the evidence of harm. They also accord with the conclusions of previous expert reports that aggressively targeting alcohol harms is a valid and necessary public health strategy."

Prof. Phillips notes the important role of judgment in quantitative models, particularly when there is limited data available, as was the case for these panels.

The 20 drugs rated in the studies were alcohol, amphetamines, anabolic steroids, benzodiazepines, buprenorphine, butane, cannabis, cocaine, crack, Ecstasy, GHB, heroin, ketamine, khat, LST, mephedrone, methadone, methamphetamine, mushrooms, and tobacco.

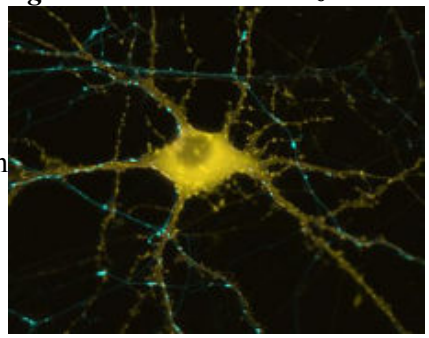
The combined research shows the important role for judgment in quantitative models, particularly when there is very little data available, as in research about illegal drugs. The decision science techniques provided new tools to reevaluate common perceptions and laws governing hazardous drugs.

This research was made public in conjunction with the INFORMS Decision Analysis Society (DAS).Formed in 1980 with a thousand current members, DAS promotes the development and use of logical methods for improving decision-making in public and private enterprise. For more information, visit http://www.informs.org/Community/DAS.

http://www.eurekalert.org/pub_releases/2014-06/uoc--hob061114.php

How our brains store recent memories, cell by single cell

Findings may shed light on how to treat neurological conditions like Alzheimer's and epilepsy



Confirming what neurocomputational theorists have long suspected, researchers at the Dignity Health Barrow Neurological Institute in Phoenix, Ariz. and University of California, San Diego School of Medicine report that the human brain locks down episodic memories in the hippocampus, committing each recollection to a distinct, distributed fraction of individual cells.

This is a human neuron showing actin formation in response to stimulation. **Michael A. Colicos, UC San Diego**

The findings, published in the June 16 Early Edition of PNAS, further illuminate the neural basis of human memory and may, ultimately, shed light on new treatments for diseases and conditions that adversely affect it, such as Alzheimer's disease and epilepsy.

"To really understand how the brain represents memory, we must understand how memory is represented by the fundamental computational units of the brain – single neurons – and their networks," said Peter N. Steinmetz, MD, PhD, program director of neuroengineering at Barrow and senior author of the study. "Knowing the mechanism of memory storage and retrieval is a critical step in understanding how to better treat the dementing illnesses affecting our growing elderly population."

Steinmetz, with first author John T. Wixted, PhD, Distinguished Professor of Psychology, Larry R. Squire, PhD, professor in the departments of neurosciences, psychiatry and psychology, both at UC San Diego, and colleagues, assessed nine patients with epilepsy whose brains had been implanted with electrodes to monitor seizures. The monitoring recorded activity at the level of single neurons.

The patients memorized a list of words on a computer screen, then viewed a second, longer list that contained those words and others. They were asked to identify words they had seen earlier, and to indicate how well they remembered them. The observed difference in the cell-firing activity between words seen on the first list and those not on the list clearly indicated that cells in the hippocampus were representing the patients' memories of the words.

The researchers found that recently viewed words were stored in a distributed fashion throughout the hippocampus, with a small fraction of cells, about 2 percent, responding to any one word and a small fraction of words, about 3 percent, producing a strong change in firing in these cells.

"Intuitively, one might expect to find that any neuron that responds to one item from the list would also respond to the other items from the list, but our results did not look anything like that. The amazing thing about these counterintuitive findings is that they could not be more in line with what influential neurocomputational theorists long ago predicted must be true," said Wixted.

Although only a small fraction of cells coded recent memory for any one word, the scientists said the absolute number of cells coding memory for each word was large nonetheless – on the order of hundreds of thousands at least. Thus, the loss of any one cell, they noted, would have a negligible impact on a person's ability to remember specific words recently seen.

Ultimately, the scientists said their goal is to fully understand how the human brain forms and represents memories of places and things in everyday life, which cells are involved and how those cells are affected by illness and disease. The researchers will next attempt to determine whether similar coding is involved in memories of pictures of people and landmarks and how hippocampal cells representing memory are impacted in patients with more severe forms of epilepsy.

Co-authors include Yoonhee Jang, University of Montana; Megan H. Papesh, Louisiana State University; Stephen D. Goldinger, Arizona State University; Joel Kuhn, UCSD; Kris A. Smith and David M. Treiman, Barrow Neurological Institute.

Funding for this research came, in part, from the Medical Research Service of the Department of Veterans Affairs, the National Institute of Mental Health (grant 24600), the National Institute for Deafness and Other Communications Disorders (grant 1R21DC009781), the Barrow Neurological Foundation, the Arizona Biomedical Research Council and the UC San Diego Kavli Institute for Brain and Mind.

http://www.eurekalert.org/pub_releases/2014-06/gsoa-doe061614.php

Discovery of Earth's northernmost perennial spring

A Canadian team lead by Stephen Grasby reports the discovery of the highest latitude perennial spring known in the world.

Boulder, Colo., USA – This high-volume spring demonstrates that deep groundwater circulation through the cryosphere occurs, and can form gullies in a region of extreme low temperatures and with morphology remarkably similar to those on Mars. The 2009 discovery raises many new questions because it remains uncertain how such a high-volume spring can originate in a polar desert environment.

Grasby and colleagues encountered the northernmost perennial spring in the world, which they have dubbed the Ice River Spring, on Ellesmere Island, Nunavut, Canadian High Arctic. The specific study area is north of Otto Fiord in a mountainous region underlain by carbonates of the Nansen Formation. The spring

discharges at 300 m elevation from colluvium on a south-facing (21° incline) mountain slope. The unnamed mountain rises 800 m above sea level.

Detailed recordings show that this spring flows year-round, even during 24 hours of darkness in the winter months, when air temperatures are as low as minus 50 degrees Celsius.

Detailed geochemistry shows that the waters originate from the surface and circulate down as deep as 3 km before returning through thick permafrost as a spring.

This points to a much more active hydrogeological system in polar regions than previously thought possible, which is perhaps driven by glacial meltwater.

Another intriguing feature of the Ice River site is the remarkable similarity to mid-latitude gullies observed on Mars. The discovery of these features on Mars has led to suggestions that recent groundwater discharge has occurred from confined aquifers.

This is a view looking north at the Ice River spring, the highest latitude perennial spring known. Located in the polar desert of northern Ellesmere Island, Nunavut, the high discharge spring carves a gully remarkable similar to those observed on Mars. **Photo by Stephen Grasby**

Deep groundwater circulation through the High Arctic cryosphere forms Mars-like gullies Stephen E. Grasby et al., Geological Survey of Canada, Natural Resources Canada, 3303 33rd Street NW, Calgary, Alberta T2L 2A7, Canada, and Dept. of Geoscience, University of Calgary, 2500 University Drive NW, Calgary, Alberta T2N 1N4, Canada. Published online 9 June 2014; http://dx.doi.org/10.1130/G35599.1.

http://www.bbc.com/news/health-27866391

Microwave helmet 'can spot a stroke'

Scientists say they have devised a helmet that can quickly determine whether a patient has had a stroke.

By Michelle Roberts Health editor, BBC News online

It could speed diagnosis and treatment of stroke to boost chances of recovery, the scientists say. The wearable cap bounces microwaves off the brain to determine whether there has been a bleed or clot deep inside. The Swedish scientists who made the device plan to give it to ambulance crews to test after successful results in early studies with 45 patients.

Race against time

When a person has a stroke, doctors must work quickly to limit any brain damage. If it takes more than four hours to get to hospital and start treatment, parts of their brain tissue may already be dying.

But to give the best treatment, doctors first need to find out if the stroke is caused by a leaky blood vessel or one blocked by a clot.

A computerised tomography (CT) scan will show this, but it can take some time to organise one for a patient, even if they have been admitted as an emergency to a hospital that has one of these scanners.

Any delay in this "golden hour" of treatment opportunity could hamper recovery.

Vital window

To speed up the process, researchers in Sweden, from Chalmers University of Technology, Sahlgrenska Academy and Sahlgrenska University Hospital, have come up with a mobile device that could be used on the way to hospital.

The helmet uses microwave signals - the same as the ones emitted by microwave ovens and mobile phones but much weaker - to build a picture of what is going on throughout the brain.

Tests with an early prototype - a refashioned bicycle helmet - found it could accurately distinguish between bleeds (haemorrhagic stroke) and clots (ischaemic stroke), although not 100% of the time.

They have since built and tested a custom-made helmet to better fits skulls of different shapes and sizes, and they have tested it out with the help of nurses and patients at a local hospital ward.

Ultimately, they want to fit it into the pillow the patient rests their head on.

The researchers say their device needs more testing, but could be a useful aid in the future.

Doctors would probably still need to use other diagnostic methods too, they told Transactions on Biomedical Engineering journal.

Investigator Prof Mikael Persson said: "The possibility to rule out bleeding already in the ambulance is a major achievement that will be of great benefit in acute stroke care."

Dr Shamim Quadir, of the UK's Stroke Association, said: "When a stroke strikes, the brain is starved of oxygen, and brain cells in the affected area die. Diagnosing and treating stroke as quickly as possible is crucial.

"While this research is at an early stage, it suggests that microwave-based systems may become a portable, affordable, technology that could help rapidly identify the type of stroke a patient has had, and get them treated faster.

"By diagnosing and treating stroke as early as possible, we can minimise the devastating impact of stroke, secure better outcomes for patients and, ultimately, save lives. Time lost is brain lost."

http://www.eurekalert.org/pub_releases/2014-06/uoc--sdr061114.php

Single dose reverses autism-like symptoms in mice

Old drug used for sleeping sickness may point to new treatment in humans

In a further test of a novel theory that suggests autism is the consequence of abnormal cell communication, researchers at the University of California, San Diego School of Medicine report that an almost century-old drug approved for treating sleeping sickness also restores normal cellular signaling in a mouse model of autism, reversing symptoms of the neurological disorder in animals that were the human biological age equivalent of 30 years old.

The findings, published in the June 17, 2014 online issue of Translational Psychiatry, follow up on similar research published last year by senior author Robert K. Naviaux, MD, PhD, professor of medicine, pediatrics and pathology, and colleagues.

Naviaux said the findings fit neatly with the idea that autism is caused by a multitude of interconnected factors: "Twenty percent of the known factors associated with autism are genetic, but most are not. It's wrong to think of genes and the environment as separate and independent factors. Genes and environmental factors interact. The net result of this interaction is metabolism."

Naviaux, who is co-director of the Mitochondrial and Metabolic Disease Center at UC San Diego, said one of the universal symptoms of autism is metabolic disturbances. "Cells have a halo of metabolites (small molecules involved in metabolism, the set of chemical processes that maintain life) and nucleotides surrounding them. These create a sort of chemical glow that broadcasts the state of health of the cell."

Cells threatened or damaged by microbes, such as viruses or bacteria, or by physical forces or by chemicals, such as pollutants, react defensively, a part of the normal immune response, Naviaux said. Their membranes stiffen. Internal metabolic processes are altered, most notably mitochondria – the cells' critical "power plants." And communications between cells are dramatically reduced. This is the "cell danger response," said Naviaux, and if it persists, the result can be lasting, diverse impairment. If it occurs during childhood, for example, neurodevelopment is delayed.

"Cells behave like countries at war," said Naviaux. "When a threat begins, they harden their borders. They don't trust their neighbors. But without constant communication with the outside, cells begin to function differently. In the case of neurons, it might be by making fewer or too many connections. One way to look at this related to autism is this: When cells stop talking to each other, children stop talking."

Naviaux and colleagues have focused on a cellular signaling system linked to both mitochondrial function and to the cell's innate immune function. Specifically, they have zeroed in on the role of nucleotides like adenosine triphosphate (ATP) and other signaling mitokines – molecules generated by distressed mitochondria. These mitokines have separate metabolic functions outside of the cell where they bind to and regulate receptors present on every cell of the body. Nineteen types of so-called purinergic receptors are known to be stimulated by these extracellular nucleotides, and the receptors are known to control a broad range of biological characteristics with relevance to autism, such as impaired language and social skills.

In their latest work, Naviaux again tested the effect of suramin, a well-known inhibitor of purinergic signaling that was first synthesized in 1916 and is used to treat trypanosomiasis or African sleeping sickness, a parasitic disease. They found that suramin blocked the extracellular signaling pathway used by ATP and other mitokines in a mouse model of autism spectrum disorder (ASD), ending the cell danger response and related inflammation. Cells subsequently began behaving normally and autism-like behaviors and metabolism in the mice were corrected.

However, the biological and behavioral benefits of suramin were not permanent, nor preventive. A single dose remained effective in the mice for about five weeks, and then washed out. Moreover, suramin cannot be taken long-term since it can result in anemia and adrenal gland dysfunction.

Still, Naviaux said these and earlier findings are sufficiently encouraging to soon launch a small phase 1 clinical trial with children who have ASD. He expects the trial to begin later this year.

"Obviously correcting abnormalities in a mouse is a long way from a cure in humans, but we think this approach – antipurinergic therapy – is a new and fresh way to think about and address the challenge of autism.

"Our work doesn't contradict what others have discovered or done. It's another perspective. Our idea is that this kind of treatment – eliminating a basic, underlying metabolic dysfunction – removes a hurdle that might make other non-drug behavioral and developmental therapies of autism more effective. The discovery that a single dose of medicine can fundamentally reset metabolism for weeks means that newer and safer drugs might not need to be given chronically. Members of this new class of medicines might need to be given only intermittently during sensitive developmental windows to unblock metabolism and permit improved development in response to many kinds of behavioral and occupational therapies, and to natural play."

Co-authors are Jane C. Naviaux, Michael A. Schuchbauer and Susan B. Powell of the UCSD Department of Psychiatry; Kefeng Li and Lin Wang, UCSD Mitochondrial and Metabolic Disease Center and UCSD Department of Medicine; and Victoria B. Risbrough, UCSD

Department of Psychiatry and San Diego Veterans Affairs Center for Excellence in Stress and Mental Health.
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http://www.eurekalert.org/pub_releases/2014-06/ada-dn060214.php

Do 'walkable' neighborhoods reduce obesity, diabetes?

Studies show more sprawl associated with higher incidence of poor health outcomes

San Francisco – People who live in neighborhoods that are conducive to walking experienced a substantially lower rate of obesity, overweight and diabetes than those who lived in more auto-dependent neighborhoods, according to a pair of studies presented at the American Diabetes Association's 74th Scientific Sessions®. Researchers in Canada compared adults living in the most and least "walkable" metropolitan areas in southern Ontario and found a lower risk of developing diabetes over a 10-year period for those who lived in neighborhoods with less sprawl, more interconnectivity among streets, and more local stores and services within walking distance, among other measures used to determine a neighborhood's "walkability." The researchers controlled for variables, such as health at baseline, in order to rule out the probability that healthier people were choosing more walkable neighborhoods to begin with. A second study that compared neighborhoods, not individuals, found that the most walkable neighborhoods had the lowest incidence of obesity, overweight and diabetes.

"How we build our cities matters in terms of our overall health," said lead researcher Gillian Booth, MD, Endocrinologist and Research Scientist at St. Michael's Hospital and the Institute for Clinical Evaluative Sciences (ICES) in Toronto. "This is one piece of a puzzle that we can potentially do something about. As a society, we have engineered physical activity out of our lives. Every opportunity to walk, to get outside, to go to the corner store or walk our children to school can have a big impact on our risk for diabetes and becoming overweight."

Marisa Creatore, Epidemiologist with the Centre for Research on Inner City Health at St. Michael's Hospital, Toronto, added that the studies revealed the degree to which "your environment can influence your decisions about physical activity. When you live in a neighborhood designed to encourage people to be more active, you are in fact more likely to be more active."

Specifically, the studies found that people living in neighborhoods with greater walkability saw on average a 13 percent lower development of diabetes incidence over 10 years than those that were less walkable. However, walkability was only protective in those who were younger and middle aged; those who were age 65 or older saw no benefit from living in a walkable neighborhood.

Diabetes was lowest in the most walkable neighborhoods, where incidence fell 7 percent over 10 years, whereas neighborhoods rated least walkable saw a 6 percent rise in diabetes over the same time period. Overweight and obesity, as well, was lowest in the most walkable neighborhoods and fell by 9 percent over 10 years, whereas it rose 13 percent in neighborhoods with the least walkability during that time.

The researchers also noted that people who lived in the most walkable neighborhoods were three times more likely to walk or bicycle and half as likely to drive as a means of transportation. Solving the obesity pandemic, concluded Booth, "will require both policy changes as well as individual strategies. We have to take a more population-based approach to the problem, given the environment we live in."

http://www.eurekalert.org/pub_releases/2014-06/dumc-hds061114.php

Heparin derivative suppresses neuroblastoma tumor growth

New strategy for treating neuroblastoma using a modified version of heparin

DURHAM, N.C. -- Researchers at Duke Medicine have identified a new strategy for treating neuroblastoma using a modified version of heparin, a century-old injectable drug that thins the blood to prevent clots from forming.

The study, conducted in mice and published June 17, 2014, in the Journal of Clinical Investigation, found that when heparin is altered to remove its blood-thinning properties, it can suppress and shrink neuroblastoma tumors without causing severe bleeding.

"Our research translates mechanistic insights about heparin into a potential new therapy for neuroblastoma, and possibly other cancers," said senior author Gerard C. Blobe, M.D., Ph.D., professor of medicine, pharmacology and cancer biology at Duke.

Neuroblastoma is a cancer that arises in nerve tissue, and is typically seen in infants and children. While neuroblastoma is rare overall – 700 new cases in the United States each year, according to the American Cancer Society – it is the most common cancer in infants.

Despite numerous treatment options for neuroblastoma, survival rates in children with advanced cancer are less than 40 percent due to disease recurrence and the persistence of residual cancerous cells after chemotherapy. However, one difference between neuroblastoma and other solid tumors is the function of the stroma, or connective tissue around the tumor. "Most of the time we think of stroma in solid tumors as a bad thing that helps cancers become more invasive. In neuroblastoma, it's the opposite: having a lot of connective tissue around the tumor does something favorable for patients," Blobe said.

Studying the stroma's biology, the researchers determined that the connective tissue produces and releases receptors involved in nervous system signaling called heparan sulfate proteoglycans. The heparan sulfate proteoglycans had a differentiating effect on the cancer cells, making immature cancer cells act more like mature neurons and keeping them from proliferating.

Heparan sulfate proteoglycans are structurally similar to the anticoagulant heparin, which led the researchers to hypothesize that heparin might recreate the function naturally occurring in the stroma. They administered heparin to human neuroblastoma cells in laboratory cultures as well as to mice with neuroblastoma, and found that it could differentiate cancer cells and cause tumors to regress in mice. However, the heparin caused severe bleeding, rendering it an unusable treatment.

"That's what caused the 'eureka' moment. Heparin was effective, but caused serious bleeding," said Erik H. Knelson, an M.D./Ph.D. candidate at Duke University School of Medicine and the study's lead author. "If we could find a modified heparin that still promoted differentiation but did not cause anticoagulation, we might have a successful treatment."

The researchers studied the structure of heparin, and determined that although certain properties were necessary for anticoagulation, only some were important for the signaling that promoted differentiation. Using this finding, they identified a derivative of heparin that would still allow for differentiation but not cause anticoagulation. When tested in lab cultures, the heparin derivative suppressed neuroblastoma growth. In mice, it slowed the cancer's progression, shrunk the tumors and extended the animals' lives.

"The study illustrates the benefit of listening to the cancer and its biology," Knelson said. "We started out with an investigation of tumor stroma and its mechanism, and designed a potential therapy using this new understanding."

The team is now working to move the research into clinical trials in humans to determine whether the heparin derivative offers the same benefit in humans as it did in mice. "We want to repurpose a drug that's already out there for the benefit of patients," Blobe said. "What's exciting about that is there are other tumors in which differentiation is useful, so there's the potential to apply these insights to other cancers."

In addition to Blobe and Knelson, study authors include Angela L. Gaviglio of the Department of Pharmacology and Cancer Biology at Duke; Jasmine C. Nee, Mark D. Starr and Andrew B. Nixon of the Department of Medicine at Duke; and Stephen G. Marcus of Cantex Pharmaceuticals.

The study was supported by the National Institutes of Health (F30 CA168043-01, R01-CA136786 and R01-CA135006) as well as a Reach Award from Alex's Lemonade Stand.

Knelson and Blobe have submitted a U.S. provisional patent application describing the development of heparin derivatives as therapeutic agents in neuroblastoma.

http://www.eurekalert.org/pub_releases/2014-06/uomh-ant061614.php

A new twist on neuro disease: Discovery could aid people with dystonia, Parkinson's and more

Persistence pays off with new mouse model of primary dystonia

ANN ARBOR, Mich. -- Twist and hold your neck to the left. Now down, and over to the right, until it hurts. Now imagine your neck – or arms or legs – randomly doing that on their own, without you controlling it. That's a taste of what children and adults with a neurological condition called dystonia live with every day – uncontrollable twisting and stiffening of neck and limb muscles.

The mystery of why this happens, and what can prevent or treat it, has long puzzled doctors, who have struggled to help their suffering dystonia patients. Now, new research from a University of Michigan Medical School team may finally open the door to answering those questions and developing new options for patients.

In a new paper in the Journal of Clinical Investigation, the researchers describe new strains of mice they've developed that almost perfectly mimic a human form of the disease. They also detail new discoveries about the basic biology of dystonia, made from studying the mice.

They'll soon make the mice available for researchers everywhere to study, to accelerate understanding of all forms of dystonia and the search for better treatments. The lack of such mice has held back research on dystonia for years.

The U-M team's success in creating a mouse model for the disease came only after 17 years of stubborn, persistent effort – often in the face of setbacks and failure.

Led by U-M neurologist William Dauer, M.D., the team tried to figure out how and why a gene defect leads to an inherited form of dystonia that, intriguingly, doesn't start until the pre-teen or teen years, after which it progresses for many years but then stops getting worse after the person reaches their mid-20s.

The gene defect responsible, called DYT1, causes brain cells to make a less-active form of a protein called torsinA. But despite more than a decade of effort by Dauer's team and many others around the world, no one has been able to translate this information into an animal model with dystonia's characteristic movements.

Using the childhood onset as a clue, Dauer and his team used cutting-edge genetic technology to severely impair torsinA function during early brain development. This novel twist caused the new mice to closely mimic the human disease: they don't develop dystonia until they reach preteen age in "mouse years," and their symptoms stop getting worse after a while.

With this powerful tool in hand, Dauer's team were now able to peer into the brains of these animals to begin to unravel the mysteries of the disease.

In an unexpected development, they found that the lack of torsinA in the brains of dystonic mice led to the death of neurons – a process called neurodegeneration – in just a few highly localized parts of the brain that control movement. Like the dystonic movements, this neurodegeneration began in young mice, progressed for a time, and then became fixed.

"We've created a model for understanding why certain parts of the brain are more vulnerable to prob-lems from a certain genetic insult," says Dauer, an associate professor in the U-M departments of Neu-rology and Cell & Developmental Biology.

"In this case, we're showing that in dystonia, the lack of this particular protein during a critical window of time is causing cell death. Every disease is telling us something about biology -- one just has to listen carefully."

More discoveries to come

Dauer and his team don't yet know why only one-third of human DYT1 gene mutation carriers develop primary dystonia during their school years, and why those who don't develop the disease before their early 20s will never go on to develop it. They believe some critical events during the brain's development in infancy and childhood may have to do with it - and they're already working to explore that question in mice.

They also believe their mouse model will help them and other researchers understand how dystonia occurs in people who have Parkinson's disease, Huntington's disease, or damage caused by a stroke or brain injury. Some people develop dystonia without either a known gene defect or any of these other diagnoses – a condition called idiopathic dystonia. In all these cases, as in people with DYT1 mutations, dystonia's twisting and curling motions likely arise from problems in the area of the brain that controls the body's motor control system. In other words, something's going wrong in the process of sending signals to the nerves that control muscles involved in movement. Studying a "pure" form of dystonia using the mice will allow researchers to understand just what's going on.

The team's ultimate goal is to find new treatments for all kinds of dystonia. Currently, children, teens and young adults who develop it can take medications or even opt for a form of neurosurgery called deep brain stimulation. But the drugs carry major side effects and are only partially effective – and brain surgery carries its own risks. Dauer and his team are working to screen drug candidates.

In addition to Dauer, the research team included first author Chun-Chi Liang, and laboratory members Lauren M. Tanabe, Stephanie Jou, and Frank Chi.

Patients interested in learning more about dystonia care at the U-M Health System should visit http://www.uofmhealth.org/medical-services/dystonia .

Funding: The research was funded by the Bachmann-Strauss Dystonia and Parkinson Disease Foun-dation, the Robert P. Apkarian Integrated Electron Microscopy Core of Emory University, and a grant from the National Institute of Neurological Disorders and Stroke, NS077730.

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http://bit.ly/1m3vH7t

Russian Meteor Crash Linked to Mass Extinction

New evidence implicates one of Earth's biggest impact craters in a mass extinction that occurred 33.7 million years ago, according to research presented here Wednesday (June 11) at the annual Goldschmidt geochemistry conference.

By Becky Oskin, Senior Writer | June 13, 2014 07:35am ET

SACRAMENTO, Calif. - Researchers from the University of California, Los Angeles precisely dated rocks from beneath the Popigai impact crater in remote Siberia to the Eocene epoch mass extinction that occurred 33.7 million years ago. Popigai crater is one of the 10 biggest impact craters on Earth, and in 2012, Russian scientists claimed the crater harbors a gigantic industrial diamond deposit.

The new age, which is later than other estimates, means the Eocene extinction - long blamed on climate change - now has another prime suspect: an "impact winter." Meteorite blasts can trigger a deadly global chill by blanketing the Earth's atmosphere with tiny particles that reflect the sun's heat. [Crash! 10 Biggest Impact Craters on Earth]

"I don't think this will be the smoking gun, but it reopens the door to Popigai being involved in the mass extinction," said lead study author Matt Wielicki, a UCLA graduate student.

This isn't the first time flying space rocks have been implicated in the Eocene's mass die-offs. Other possible culprits besides Popigai crater include three smaller Earth-meteorite smashups between 35 million and 36 million years ago: Chesapeake Bay crater offshore Virginia, Toms Canyon crater offshore New Jersey and Mistastin crater in Labrador, Canada.

Previously, all four craters were ruled out because of their ages. Earlier dating attempts had pinned Popigai's impact age at 35.7 million years ago, Wielicki said. And 2 million years is too much of a time lag between a meteorite blast and disappearing species, he said. The cosmic impact that killed the dinosaurs 65 million years ago coincides in time with its extinction by just 33,000 years, according to the most precise dating techniques available.

With no meteorite to blame for the Eocene mass extinction, scientists focused on climate change. In this case, global cooling killed off many species, researchers think.

Here's how they can tell: By measuring isotopes of oxygen, carbon and other elements in Eocene-age rocks, researchers can estimate Earth's past temperature and greenhouse-gas levels. (Isotopes are elements with different numbers of neutrons in their nuclei.)

The signal from the Eocene shows the epoch started off extremely warm and then swung toward colder, drier conditions before the big extinction event. However, a sharp spike in these climate signals at the end of the Eocene hints at short-lived but extreme global cooling, followed by a rebound to warmer temperatures. "The age of the crater matches perfectly with that [short-term] global change," Wielicki said. Wielicki thinks the Popigai impact created a global icehouse, similar to the climate disasters seen after enormous volcanic eruptions or the dinosaur-killing impact. The meteoritic crash could have pumped massive amounts of sunlight-reflecting sulfur droplets into the atmosphere, he said. The planet's "quick" recovery, in geologic time, set plants and animals on an evolutionary path to modern species.

The end of the Eocene was the last big mass extinction in Earth's history. More than 90 percent of snails disappeared, sea urchins were hard hit and the earliest toothed whales died off, which eventually would be replaced by modern whales. The dramatic shift of European mammals, called the "Grand Coupure," occurred soon afterward, following Eocene-Oligocene transition.

http://bit.ly/1nSzNPt

The Gory New York City Riot that Shaped American Medicine

Back before medical school was a respected place to be, New Yorkers raised up in protest over the doctors' preference for cadavers for study

By Bess Lovejoy

For most Americans, being a physician is a respectable profession, held in high esteem and relatively untarnished by the constant health care debates. But that wasn't always the case, and one of the first major riots in the post-revolution United States was caused by popular anger against doctors. The so-called "Doctors' Riot," which began on April 16, 1788, and killed as many as 20 people, influenced both the perception of American medicine and the way it was carried out for decades to come, even though it has been mostly forgotten today.

In the closing years of the 18th century, New York was home to only one medical school: Columbia College. At the time, those looking to practice medicine didn't have to graduate from a professional school, and this led to some students attending private, not-for-credit classes at New York Hospital, taught by Richard Bayley, a Connecticut-born doctor who had studied in London with the famous Scottish surgeon John Hunter.

Anatomical dissections were a central component of these classes, and medical training in general, but they were offensive, even seen as sacrilegious, to early New Yorkers. In the winter of 1788, the city was abuzz with newspaper stories about medical students robbing graves to get bodies for dissection, mostly from the potter's field and the cemetery reserved for the city's blacks, known as the Negroes Burial Ground. While some of those reports may have been based on rumor, they pointed to an underlying truth: with no regulated source of bodies for dissection, the medical students had taken matters into their hands and begun plundering the local graveyards.

In February, a group of the city's free and enslaved blacks submitted a petition to the Common Council complaining of "young gentlemen in this city who call themselves students of the physic," and who "under cover of the night, in the most wanton sallies of excess … dig up bodies of our deceased friends and relatives of your petitioners, carrying them away without respect for age or sex."

The petitioners didn't ask for a stop to the grave-robbing, only that it be "conducted with the decency and propriety which the solemnity of such occasion requires." But the petition was ignored; many in the city were willing to turn a blind eye to grave-robbing as long as those bodies were poor and black.

However, on February 21, 1788, the Advertiser printed an announcement saying that a body of a white woman had been stolen from Trinity Churchyard. With that, popular resentment began to boil over.

There are conflicting accounts of how the riot began, but most place the start outside New York Hospital, where a group of boys playing in the grass saw something that upset them - and then incensed the city. In some tellings, the boys saw a severed arm hanging out of one of the hospital windows to dry. In other versions, one of the boys climbed a ladder and peered into the dissecting room, where a surgeon waved the severed arm at him. In yet other versions, the boy's mother had recently died, and the surgeon told the boy the arm had belonged to his mother. In this version of the tale, recounted in Joel Tyler Headley's 1873 The Great Riots of New York, the boy ran off to tell the news to his father, a mason, who went to the cemetery and exhumed his wife's coffin. After finding it empty, he marched on the hospital with a group of angry worker friends still carrying their picks and shovels.

Colonel William Heth, writing in a letter to Governor of Virginia Edmund Randolph, described what happened when the men got to the hospital:

"The cry of barbarity and etc. was soon spread - the young sons of Galen [a poetic allusion to a physician in Ancient Greece] fled in every direction - one took refuge in a chimney - the mob raised - and the Hospital apartments were ransacked. In the Anatomy room, were found three fresh bodies - one, boiling in a kettle, and two others cutting up - with certain parts of the two sex's hanging up in a most brutal position.

The circumstances, together with the wanton and apparent inhuman complexion of the room, exasperated the Mob beyond all bounds, to the total destruction of every anatomy in the hospital."

Although most of the doctors and medical students fled when the workmen appeared, a handful remained to try and guard the valuable collection of anatomical and pathological specimens, many imported. Their efforts were in vain, and the specimens were dragged out in the street and set ablaze. Bayley and his protégé, Wright Post, might have been added to the fire too if it hadn't been for the arrival of Mayor James Duane and the sheriff, who ordered the doctors and medical students escorted to jail for their own protection.

Things quieted down after that, but the next morning, a mob ran around the city searching for doctors, medical students, and bodies. Hundreds descended on Columbia, despite the efforts of alumnus Alexander Hamilton, who pleaded with the crowd from the school's front steps.

He was shouted down and pushed past, and the crowed ran into the school, where they searched the anatomical theatre, museum, chapel, library, and even student's bedrooms for signs of dissection. Finding no bodies (students had removed them all the previous night), the men searched several other doctors' homes - including Bayley's - in vain, then marched down Broadway to the jail. Governor George Clinton, Mayor Duane, and other prominent politicians urged them to disperse, but the crowd refused and swelled into an estimated 5,000.

Armed with rocks, bricks, and timber torn from the nearby gallows, they finally attacked the jail, yelling "bring out your doctors!"

Inside, the medical students clambered over the broken glass and used the rocks and bricks thrown at them to fend off their attackers. One of the rioters climbed inside the jail through a ground floor window, only to be killed by a guard, which further incensed the rioters outside.

Governor Clinton called out several rounds of militiamen, who attempted to calm the scene, although they had strict orders not to fire their muskets. That is, until Secretary of Foreign Affairs John Jay (who would become the first Chief Justice of the Supreme Court the following year) "got his scull almost crackd" with a rock, and the Revolutionary War hero General Baron von Steuben was hit with a brick. The militiamen could no longer be restrained, and they opened fire. In the tumult, at least three rioters and three members of the militia were killed, with the final death toll estimated as high as 20.

In the days that followed, local newspapers stopped running their ads for doctors and medical classes. People regularly went to the cemeteries to inspect the graves of their loved ones, and formed armed groups known as "Dead Guard Men" to protect the cemeteries. Several of the city's most prominent physicians, including Bayley, published notices saying they had never robbed any cemetery in the city, nor asked anyone else to do so. The key there was "in the city" - the Negroes Burial Ground and potter's field had been established outside the city. A grand jury investigated the riot, but there is no record of anyone being convicted. Nevertheless, the reputation of the medical profession in New York was tainted for years.

The New York Doctors Riot was just one in a stream of so-called "anatomy riots" that plagued the United States in the 18th and 19th centuries. Medical historian Michael Sappol has counted at least 17 such incidents between 1765 and 1854, in New Haven, Baltimore, Cleveland and Philadelphia. These riots were sparked by anger over dissections and grave-robbing, which was how most schools got their bodies, since there was no legal supply.

People saw grave-robbing as an affront to the honor of the dead and the sacred nature of graveyards, and dissection frightened many Christians who believed that only complete bodies could be resurrected. Dissection also had a veneer of criminality: in England, the only legal source of bodies was executed criminals, and many saw anatomical dissection as an extra layer of punishment suitable only for the wicked.

In response to these riots, anatomy acts - also knows an "bone bills" - were passed to legislate the supply of cadavers. The year after the Doctors Riot, the New York legislature passed "An Act to Prevent the Odious Practice of Digging Up and Removing for the Purpose of Dissection, Dead Bodies Interred in Cemeteries or Burial Places." The act outlawed grave-robbing, and provided that criminals executed for murder, arson, or burglary could be sentenced to dissection after death. But it wasn't effective: there weren't nearly enough bodies of executed criminals to satisfy demand, and so medical students continued to rob graves, albeit more discreetly than before.

Rumors of grave-robbing and dissection-related scandals continued into the twentieth century before finally disappearing from the newspapers' front pages. (Today, the illegal harvesting of organs and tissues fuels our medical science horror stories.)

But the riot did have other, longer-lasting effects. It led to one of the earliest medical licensing systems in the colonies, in which would-be doctors had to apprentice with a respected physician or attend two years of medical school in addition to passing a rigorous government exam. No longer could medical students simply attend a couple of classes and hang out their shingle in a small town upstate. Nevertheless, memories of the opportunistic "students of the physic" persisted for years, and it took a long time before being a doctor was considered an entirely respectable profession in the city.

http://bit.ly/1kUY4zG

Earth's Most Abundant, Yet Elusive, Mineral Named after Nobel Prize Winner

Scientists get the first-ever glimpse of the magnesium silicate mineral, now named after physicist Percy Bridgman

Jun 17, 2014 |By Jeanna Bryner and LiveScience

Earth's most abundant mineral lies deep in the planet's interior, sealed off from human eyes. Now, scientists for the first time have gotten a glimpse of the material in nature, enclosed inside a 4.5-billion-year-old meteorite. The result: They have characterized and named the elusive mineral.

The new official name, bridgmanite, was approved for the mineral formerly known by its chemical components and crystal structure - silicate-perovskite. The magnesium-silicate mineral was named after Percy Bridgman, a 1946 Nobel Prize-winning physicist, according to the American Geophysical Union blog.

"It is a very exciting discovery," Chi Ma of Caltech and Oliver Tschauner, of the University of Nevada, Las Vegas, told Live Science in an email. "We finally tracked down natural silicate-perovskite (now bridgmanite) in a meteorite after a five-year investigation, and got to name the most abundant mineral on Earth. How cool is that?" [Shine On: Photos of Dazzling Mineral Specimens]

The mineral likely resides beneath Earth's surface in an area called the lower mantle, between the transition zone in the mantle and the core-mantle boundary, or between the depths of416 and 1,802 miles (670 and 2,900 kilometers), scientists said.

Scientists have been searching for the mineral for a long time, because in order to identify a mineral one must know its chemical composition and crystal structure, Ma said.

Researchers found the bridgmanite in a meteorite that had fallen to Earth near the Tenham station in western Queensland, Australia, in 1879. The meteorite, Ma said, is highly shocked, meaning it endured high temperatures and pressures as it slammed into other rocks in space. Those impacts can create shock veins of minerals within the meteorites.

"Scientists have identified high-pressure minerals in its shock-melt veins since 1960s. Now we have identified bridgmanite," Tschauner said, referring to the Tenham meteorite. The meteorite is considered a chondrite, the most common type of meteorite found on Earth; scientists think these meteorites are remnants shed from the original building blocks of planets.

Most meteors (which are called meteorites once they strike Earth) are fragments of asteroids, while others are the cosmic dust discarded by comets. Rarely, meteorites represent impact debris from the moon and from Mars.

Ma and Tschauner used various methods to characterize the extracted mineral, including so-called synchrotron X-ray diffraction mapping and high-resolution scanning electron microscopy.

After five years of work, including multiple experiments, Ma and Tschauner sent their data for review to the International Mineralogical Association's Commission on New Minerals, Nomenclature and Classification (CNMNC), according to the AGU blog. The commission approved the mineral and new name on June 2.

http://www.eurekalert.org/pub_releases/2014-06/jhm-vp061614.php

Vaccine 'reprograms' pancreatic cancers to respond to immunotherapy

Vaccine triggers growth of immune cell nodules within pancreatic tumors essentially reprogramming them and potentially making them vulnerable to immune-based therapies

Researchers at the Johns Hopkins Kimmel Cancer Center have developed and tested a vaccine that triggered the growth of immune cell nodules within pancreatic tumors, essentially reprogramming these intractable cancers and potentially making them vulnerable to immune-based therapies.

In their study described in the June 18 issue of Cancer Immunology Research, the Johns Hopkins team tested the vaccine in 39 people with pancreatic ductal adenocarcinomas (PDAC), the most common form of pancreatic cancer. The disease becomes resistant to standard chemotherapies and is particularly lethal, with fewer than 5 percent of patients surviving five years after their diagnosis.

PDACs do not typically trigger an immune response against the cancer cells that comprise, but with the help of a vaccine developed by Johns Hopkins researcher Elizabeth Jaffee, M.D., the scientists were able to "reprogram" tumors to include cancer-fighting immune system T cells.

The vaccine, known as GVAX, consists of irradiated tumor cells that have been modified to recruit immune cells to a patient's tumor. The researchers tested GVAX in combination with an immune modulator drug called cyclophosphamide, which targets a type of immune cell, called Tregs, that typically suppresses the immune response of certain T cells that destroy cancer.

The reprogramming is designed to make the tumors more vulnerable to other immune-modulating drugs that have been useful in fighting other cancers, said Jaffee, The Dana and Albert "Cubby" Broccoli Professor of Oncology at the Johns Hopkins University School of Medicine. Jaffee and colleague, Lei Zheng, M.D., say the vaccine could potentially convert many types of tumors to a state where immunotherapies can have a much larger impact.

For example, Jaffee says, in certain melanomas, "we've tested immunotherapies that target T cells and have found a 10-30 percent response in cancers that naturally have the ability to trigger immune system responses, but there are few options for the other 70 percent of patients who barely or never respond to immunotherapies."
The researchers found that the vaccine created structures called tertiary lymphoid aggregates within the patients' tumor, structures that help regulate immune cell activation and movement. The aggregates, which appeared in 33 of the 39 patients treated with the vaccine, had surprisingly well-organized structures that do not typically appear in these types of tumors naturally, said Zheng, an assistant professor of oncology and surgery at the Johns Hopkins University School of Medicine. "This suggests that there has been significant reprogramming of lymphocyte structures within the tumor."
The aggregates could "really shift the immunologic balance within a tumor, setting up an environment to activate good T cells to fight the cancer, by tamping down Tregs," Jaffee said, "and such T cells would be educated to recognize the cancer proteins in that specific tumor environment."
The vaccine and the resulting lymphoid aggregates boosted the activity of several molecular mechanisms that, like Tregs, inhibit cancer-fighting immune cells. That may sound like a bad thing, but it actually provides many new potential targets within the tumor for immune-modulating drugs, Zheng explained.
The researchers' next study in PDAC patients will test a combination of GVAX and an antibody to PD-1, one of the immune-suppressing molecules that became more active after vaccination. "We think combinations of immune therapies will have the biggest impact," he says.

Other Hopkins researchers involved in the study include Eric R. Lutz, Annie A. Wu, Elaine Bigelow, Rajni Sharma, Guanglan Mo, Kevin Soares, Sara Solt, Alvin Dorman, Anthony Wamwea, Allison Yager, Daniel Laheru, Christopher L. Wolfgang, Ralph H. Hruban, and Robert Anders. Jiang Wang at the University of Cincinnati College of Medicine was also a co-author on the study. Jaffee is co-director of the Skip Viragh Pancreas Cancer Clinical Care and Research Center at Johns Hopkins.

The study was supported by the National Institutes of Health's National Cancer Institute (K23 CA148964-01, P50 CA062924), the Johns Hopkins School of Medicine Clinical Science Award, the American Society of Clinical Oncology Young Investigator Award, the Viragh Foundation and the Skip Viragh Pancreatic Cancer Center at Johns Hopkins, The National Pancreas Foundation, the Lefkofsky Family Foundation, the Lustgarten Foundation, and the Sol Goldman Pancreatic Cancer Center.

Under a licensing agreement between Aduro BioTech Inc. and the Johns Hopkins University and Jaffee, the University is entitled to milestone payments and royalty on sales of the vaccine product.

http://bit.ly/1uK5ATG

Our humming brains help us learn rapidly

Our brain's ability to rapidly interpret and analyse new information may lie in the musical hum of our brainwaves.

11:40 18 June 2014 by Lauren Hitchings

We continuously take in information about the world but establishing new neural connections and pathways – the process thought to underlie memory formation – is too slow to account for our ability to learn rapidly.
Evan Antzoulatos and Earl Miller at the Massachusetts Institute of Technology decided to see if brainwaves – the surges of electricity produced by individual neurons firing en masse – play a role.
They used EEG to observe patterns of electrical activity in the brains of monkeys as they taught the animals to categorise patterns of dots into two distinct groups.
At first, they memorised which dots went where, but as the task became harder, they shifted to learning the rules that defined the categories.

Humming brainwaves

The researchers found that, initially, brainwaves of different frequencies were being produced independently by the prefrontal cortex and the striatum – two brain regions involved in learning.
But as the monkeys made sense of the game, the waves began to synchronise and "hum" at the same frequency – with each category of dots having its own frequency.
Miller says the synchronised brainwaves indicate the formation of a communication circuit between the two brain regions.
He believes this happens before anatomical changes in brain connections take place, giving our minds time to think through various options when presented with new information before the right one gets laid down as a memory.
Otherwise, the process is too time-consuming to account for the flexibility and speed of the human mind, says Miller.
Previous studies have shown increased synchrony between the two brain regions during learning, but this is the first study to show specific patterns of synchrony linked to specific thoughts.
"We used to think of electrical activity fluctuations just as proof that the brain is working – like the humming of a car's engine. But the brain is producing all different sounds and different frequencies," says Miller.
The research demonstrates that the music the brain produces may actually be central to how it encodes specific thoughts, he adds.

Journal reference: Neuron, DOI: 10.1016/j.neuron.2014.05.005

http://www.eurekalert.org/pub_releases/2014-06/sumc-bb061214.php

Blocking brain's 'internal marijuana' may trigger early Alzheimer's deficits, study shows

Blocking of endocannabinoids implicated in the early pathology of Alzheimer's disease

A new study led by investigators at the Stanford University School of Medicine has implicated the blocking of endocannabinoids - signaling substances that are the brain's internal versions of the psychoactive chemicals in marijuana and hashish - in the early pathology of Alzheimer's disease.

A substance called A-beta - strongly suspected to play a key role in Alzheimer's because it's the chief constituent of the hallmark clumps dotting the brains of people with Alzheimer's - may, in the disease's earliest stages, impair learning and memory by blocking the natural, beneficial action of endocannabinoids in the brain, the study demonstrates.

The Stanford group is now trying to figure out the molecular details of how and where this interference occurs. Pinning down those details could pave the path to new drugs to stave off the defects in learning ability and memory that characterize Alzheimer's.

In the study, to be published June 18 in Neuron, researchers analyzed A-beta's effects on a brain structure known as the hippocampus. In all mammals, this midbrain structure serves as a combination GPS system and memory-filing assistant, along with other duties.

"The hippocampus tells us where we are in space at any given time," said Daniel Madison, PhD, associate professor of molecular and cellular physiology and the study's senior author. "It also processes new experiences so that our memories of them can be stored in other parts of the brain. It's the filing secretary, not the filing cabinet."

Applying electrophysiological techniques to brain slices from rats, Madison and his associates examined a key hippocampal circuit, one of whose chief elements is a class of nerve cells called pyramidal cells. They wanted to see how the circuit's different elements reacted to small amounts of A-beta, which is produced throughout the body but whose normal physiological functions have until now been ill defined.

A surprise finding by Madison's group suggests that in small, physiologically normal concentrations, A-beta tamps down a signal-boosting process that under certain conditions increases the odds that pyramidal nerve cells will transmit information they've received to other nerve cells down the line.

When incoming signals to the pyramidal tract build to high intensity, pyramidal cells adapt by becoming more inclined to fire than they normally are. This phenomenon, which neuroscientists call plasticity, is thought to underpin learning and memory. It ensures that volleys of high-intensity input - such as might accompany falling into a hole, burning one's finger with a match, suddenly remembering where you buried the treasure or learning for the first time how to spell "cat" - are firmly stored in the brain's memory vaults and more accessible to retrieval.

These intense bursts of incoming signals are the exception, not the rule. Pyramidal nerve cells constantly receive random beeps and burps from upstream nerve cells - effectively, noise in a highly complex, electrochemical signaling system. This calls for some quality control. Pyramidal cells are encouraged to ignore mere noise by another set of "wet blanket" nerve cells called interneurons.

Like the proverbial spouse reading a newspaper at the kitchen table, interneurons continuously discourage pyramidal cells' transmission of impulses to downstream nerve cells by steadily secreting an inhibitory substance - the molecular equivalent of yawning, eye-rolling and oft-muttered suggestions that this or that chatter is really not worth repeating to the world at large, so why not just shut up.

But when the news is particularly significant, pyramidal cells squirt out their own "no, this is important, you shut up!" chemical - endocannabinoids - which bind to specialized receptors on the hippocampal interneurons, temporarily suppressing them and allowing impulses to continue coursing along the pyramidal cells to their follow-on peers.

A-beta is known to impair pyramidal-cell plasticity. But Madison's research team showed for the first time how it does so. Small clusters consisting of just a few A-beta molecules render the interneuron's endocannabinoid receptors powerless, leaving inhibition intact even in the face of important news and thus squashing plasticity.

While small A-beta clusters have been known for a decade to be toxic to nerve cells, this toxicity requires relatively long-term exposure, said Madison. The endocannabinoid-nullifying effect the new study revealed is much more transient.

A possible physiological role for A-beta in the normal, healthy brain, he said, is that of supplying that organ's sophisticated circuits with yet another, beneficial layer of discretion in processing information.

Madison thinks this normal, everyday A-beta mechanism run wild may represent an entry point to the progressive and destructive stages of Alzheimer's disease.

Exactly how A-beta blocks endocannabinoids' action is not yet known. But, Madison's group demonstrated, A-beta doesn't stop them from reaching and binding to their receptors on interneurons. Rather, it interferes with something that binding

ordinarily generates. (By analogy, turning the key in your car's ignition switch won't do much good if your battery is dead.)

Madison said it would be wildly off the mark to assume that, just because A-beta interferes with a valuable neurophysiological process mediated by endocannabinoids, smoking pot would be a great way to counter or prevent A-beta's nefarious effects on memory and learning ability. Smoking or ingesting marijuana results in long-acting inhibition of interneurons by the herb's active chemical, tetrahydrocannabinol. That is vastly different from short-acting endocannabinoid bursts precisely timed to occur only when a signal is truly worthy of attention.

"Endocannabinoids in the brain are very transient and act only when important inputs come in," said Madison, who is also a member of the interdisciplinary Stanford Bio-X institute. "Exposure to marijuana over minutes or hours is different: more like enhancing everything indiscriminately, so you lose the filtering effect. It's like listening to five radio stations at once."

Besides, flooding the brain with external cannabinoids induces tolerance - it may reduce the number of endocannabinoid receptors on interneurons, impeding endocannabinoids' ability to do their crucial job of opening the gates of learning and memory.

The study's lead author was postdoctoral scholar Adrienne Orr, PhD. Other co-authors were postdoctoral scholars Jesse Hanson, PhD (now at Genentech) and Dong Li, PhD; and former undergraduate Adam Klotz, now a student at Stanford's Graduate School of Business. The study was funded by the National Institute for Mental Health (grant MH065541), the Harold and Leila Y. Mathers Charitable Foundation and Elan Pharmaceuticals.

http://www.bbc.com/news/world-africa-27902139

Ebola deaths pass 300 in West Africa - WHO

The number of people killed by the deadly Ebola virus in West Africa has risen to 337, the World Health Organization (WHO) has said.

Fourteen deaths and 47 new cases were reported across the region over the last week, it added. Guinea is worst-affected with 264 Ebola-related deaths.

In Sierra Leone, there have been 49 deaths and in Liberia 24, the WHO said.

The three countries have been battling to contain the outbreak since February.

The outbreak began in southern Guinea's Guekedou region, but then spread to its neighbours.

'Unmarked borders'

More than 500 suspected or confirmed cases of the virus have been recorded, the WHO said. There is no cure or vaccine for Ebola - one of the world's deadliest viruses. It is spread by close contact and kills between 25% and 90% of those infected, depending on the strain of the virus, according to the WHO. Symptoms include internal and external bleeding, diarrhoea and vomiting.

On Tuesday, Liberia reported the first Ebola-related deaths in its capital city, Monrovia. Seven people have died there, including a baby and a woman who had come from Sierra Leone, health officials said.

This is the first time an Ebola outbreak has hit multiple locations in three countries, reports BBC International Development correspondent Mark Doyle.

The people who inhabit the region where Guinea, Liberia and Sierra Leone meet are from the Kissy ethnic group and they cross the often unmarked borders freely, to farm and trade. So maintaining medical controls is a real challenge, our correspondent says.

The WHO said it was working with the three countries to strengthen cross-border collaboration aimed at tackling the outbreak. It does not recommend any travel or trade restrictions on the three countries, the WHO added.

http://bit.ly/1jFfjp0

Just Let Detroit's Vacant Lots Run Wild

Neglected and overgrown lots are, it turns out, a boon to Detroit's allergy sufferers

By Rachel Nuwer

Hay fever sufferers in Detroit have it bad. The city's 84,600 to 114,000 vacant parcels of land are prime real estate for ragweed, a potent allergen. The knee-jerk reaction would be to undertake an uphill battle to keep all of these lots mowed, although the city's sparse resources mean that mowing efforts would be sporadic at best.

Now, however, one research team has discovered that the most neglected and overgrown of those lots are, in fact, a boon to allergy sufferers around the city. Rather than harbor more ragweed, those jungle-like enclosures turn out to be a dog-eat-dog mini ecosystem - one in which the weakling ragweed is quickly choked out.

The researchers performed pollen counts at 62 lots across the city, some of which were mowed once every year or two, and others that had been left completely to their own devices.

The team found that just 28 percent of the wildest lots contained ragweed, compared to 63 percent that were mowed once per year and 70 percent that were mowed every other year.

These findings beg the question: should Detroit just give up and let nature take over?

As the Atlantic's City Labs reports, "It's uncertain whether letting nature take its course on Detroit's vacant land will help the city's recovery prospects, but the idea is likely to appeal to anyone who dreads the thought of more pollen."

http://www.medscape.com/viewarticle/826988?

Islet-Cell Transplants Successful in Type 1 Diabetes

Pancreatic islet-cell transplants eliminated the need for insulin treatment in more than half of a specific subgroup of type 1 diabetes patients - those who experience frequent severe hypoglycemia - in a federal -funded phase 3 study.

Miriam E. Tucker

SAN FRANCISCO - Preliminary data from the National Institutes of Health's Clinical Islet Transplant (CIT) Consortium prospective single-arm, multicenter trial were presented here at the American Diabetes Association (ADA) 2014 Scientific Sessions by Bernhard J. Hering, MD, professor of surgery and medicine and director of the islet-cell-transplantation program at the University of Minnesota, Minneapolis.

The primary end point, developed with the US Food and Drug Administration (FDA), was the proportion of subjects with HbA1c less than 7% at day 365 and free of severe hypoglycemic events from day 28 to day 365 inclusive following the first islet transplant. Dr. Hering didn't report that, because the manuscript has been submitted for publication. However, he did report other efficacy and safety parameters that suggest a high degree of success at the Presidents' Oral Session that closed the meeting here yesterday.

He told Medscape Medical News, "I think it's therapy that should be considered in patients in whom type 1 diabetes remains complicated by severe hypoglycemia. We have other treatment options available to those patients, but not all benefit. Each technology has a success and a failure rate. I think it would be reasonable to discuss transplant options with patients if every single intervention has failed to improve the situation. For a small subgroup of patients, I think islet transplantation can provide very fine results."

Session moderator Elizabeth R. Seaquist, MD, president, medicine and science, of the American Diabetes Association, told Medscape Medical News that although a definitive assessment can't be made without the primary end point, the data thus far suggest that the procedure "does offer promise for people who have hypoglycemic unawareness to the point where they are unable to function in their lives that there may be another therapy that can help them."

Focus on Severe Hypoglycemia Patients Is Key

She noted that restriction of the study to patients with severe hypoglycemia is important in shifting the benefit/risk equation more favorably than has been seen in other islet-transplantation trials.

"What's different is the indication. It identifies a group that may have a particular risk with standard therapy [and] in whom it may be justified to expose them to the potential side effects of the transplant," she told Medscape Medical News.

Dr. Seaquist added, "I think the other really exciting piece from this particular study is that it was a multisite project, and they had to do islet isolation at each site under standardized conditions, which is new….If we're ever going to bring islet-cell transplants forward as a therapy and have it approved by FDA, there needs to be tremendous standardization in how they're harvested and prepared for use, and the fact that they were able to do that is very exciting."

Despite the availability of interventions such as structured education and real-time continuous glucose monitoring, studies have found that a significant proportion of type 1 diabetes patients still experience at least 1 episode of severe hypoglycemia requiring assistance - 35% in a recent survey, Dr. Hering said in his presentation. "The results obtained in registry and observational studies suggest that the real-world frequency of severe hypoglycemia is much higher than suggested by clinical-trial data and remains alarmingly high," he added.

Procedure Results in Functioning Islets

The NIH CIT's phase 3 clinical trial enrolled a total 48 adult subjects at 8 North American centers. The protocol involved transplantation of up to 3 intraportal infusions of a standardized purified allogeneic human pancreatic islet product (PHPI) within 8 months. Induction immunotherapy included antithymocyte globulin and etanercept (Enbrel, Amgen), and maintenance immunosuppression comprised sirolimus and tacrolimus.

The patients were aged 18 to 65 with a duration of type 1 diabetes of at least 5 years, documented reduced awareness of hypoglycemia and/or severe glycemic lability, and at least 1 episode of severe hypoglycemia in the prior year.

All of the subjects have reached the 1-year primary-end-point evaluation. Among the 48 patients, 26 received a second transplant and 1 received 3 transplants. At day 365, 94% had evidence of islet graft function, as determined by a basal or stimulated C-peptide level greater than 0.3 ng/mL ($P < .0016$).

Over 50% were insulin-independent at 1 year following the first infusion.

Exogenous insulin use dropped from a median at baseline of 0.49 U/kg/day to a median of 0.00 U/kg/day (range, 0.00–0.43) at day 365.

Serum glucose response to a mixed-meal tolerance test decreased over time, as did the glycemic lability index and mean amplitude of glycemic excursions.

And the Clarke score - a measure of the patient's awareness of hypoglycemia - and the Ryan hypoglycemia score, which assesses a patient's ability to diagnose and self-treat hypoglycemia, both improved significantly.

Safety: Nothing Required Expedited Reporting to FDA

The introduction of immunosuppression overall produced a small but significant reduction in kidney function, with a drop in glomerular filtration rate of 8.1 mL/min ($P < .0001$) but remained stable at days 75 and 365.

The 48 subjects experienced 19 study-related serious adverse events, including 5 with bleeding following percutaneous cannulation of the portal vein, 13 related to immunosuppression (eg, transient neutropenia, cytokine release, or elevated liver-function tests), and hypoglycemia in 1 patient who still required some insulin.

None of these adverse events resulted in death, disability, or permanent problems, and none required expedited reporting to the FDA, Dr. Hering said.

Other adverse events included reactive antibodies in 6 patients (only 1 with a donor antibody) and 1 with acute kidney injury of unclear etiology .

Next Steps to Making Islet Cell Transplants Available

Dr. Hering told Medscape Medical News that once the final results are published the consortium will submit a licensing application to the FDA. If approved, the procedure will become available at centers nationwide. But that process will work very differently from a drug-approval situation, in which a pharmaceutical company markets a proprietary product.

"This is currently being discussed. In contrast to many other interventions developed by corporate business, this is a group of academic investigators. It's a new challenge for all of us to organize. We have regular meetings and conference calls, and we are working with consultants to develop a strategy for making this therapy available to patients," he said.

He envisions that the process will be collaborative rather than competitive. "It would be unreasonable to compete in this," he said, adding that that researchers should work together to make this therapy available. "I think the more people look at this, the more they recognize that a collaborative approach is the most successful. This is clearly the emerging consensus."

Dr. Hering has consulting agreements with Novartis, Sanofi, Janssen, and Otsuka Pharmaceutical Factory, developing encapsulated islets. Dr. Seaquist has reported no relevant financial relationships.

American Diabetes Association 2014 Scientific Sessions; June 17, 2014. Abstract 388-OR

http://bit.ly/1wdIxmN

Universe's Expansion Measured With Unprecedented Precision

Scientists studying more than 140,000 extremely bright galaxies have calculated the expansion of the universe with unprecedented accuracy.

by Nola Taylor Redd, Space.com

The distant galaxies, known as quasars, serve as a "standard ruler" to map density variations in the universe. Physicists were able to extend their calculations almost twice as far back in time as has been previously accomplished.

Using the Baryon Oscillation Spectroscopic Survey (BOSS), two teams of physicists have improved on scientists' understanding of the mysterious dark energy that drives the accelerating universe. By nearly tripling the number of quasars previously studied, as well as implementing a new technique, the scientists were able to calculate the expansion rate to 42 miles (68 kilometers) per second per 1 million light-years with greater precision, while looking farther back in time. [8 Baffling Astronomy Mysteries]

Andreu Font-Ribera, of the U.S. Department of Energy's Lawrence Berkeley National Laboratory, led one of the two teams, while Timothée Delubac of EPFL, Switzerland, and France's Centre de Saclay headed the other one. Font-Ribera presented the new findings in April at a meeting of the American Physical Society in Savannah, Georgia. The new research "explores a region of the universe that was not explored before," Font-Ribera said.

Stretching the standard ruler

The expanding universe stretches light waves as they travel through it, a process astronomers refer to as redshifting. An object's physical distance from the observer depends on how quickly the universe is expanding.

Baryon acoustic oscillations (BAOs) are sound waves imprinted in large structures of matter in the early universe. Competing forces of inward-pushing gravity and outward, heat-related pressure cause oscillations similar to sound waves in the baryonic, or "normal" matter in the universe.

Dark matter, which interacts with normal matter only gravitationally, stays at the center of the sound wave, while the baryonic matter travels outward, eventually creating a shell at a set radius known as the sound horizon.

Quasars, like other galaxies, are surrounded by dust. Light leaving galaxies streams through that dust, revealing the imprint of the BAOs. Studying this light allows researchers to map the distribution of quasars, as well as the gas in the early universe.

By using BOSS, the largest component of the third Sloan Digital Sky Survey, to map BAOs, scientists can determine how matter is distributed in the early universe. When it comes to measuring the expansion of the universe, BAOs serve as a "standard ruler."

"We think we know its size, and its apparent size depends on how far away it is," Patrick McDonald, of the Canadian Institute for Theoretical Astrophysics, said at the conference.

Previously, astronomers have used BAOs to measure the distances to galaxies in order to determine the distribution of mass in the universe, and thus the universe's expansion rate. But galaxies grow fainter at greater distances, so previous studies were limited to looking back only 6 billion light-years into the universe's 13.8-billion-year lifetime.

Font-Ribera and his team, which included McDonald, pioneered a method of measuring BAOs by using quasars, which are galaxies that are far brighter than

normal due to the activity of a supermassive black hole at their center. As matter falls into the black hole, it grows extremely hot, radiating light at far brighter wavelengths and over farther distances than conventional galaxies. This allowed the scientists to measure the mass distribution of the universe out to 12 billion years. Font-Ribera's research involved approximately 50,000 quasars. The new study published by Delubac's team reviewed nearly three times as many sources, more precisely calculating the expansion rate to an accuracy of 2.2 percent. [See images of dark matter in the universe]

"If we looked back to the universe when it was less than a quarter of its present age, we'd see that a pair of galaxies separated by a million light-years would be drifting apart at a velocity of 68 kilometers a second as the universe expands," Font-Ribera said in an accompanying press release.

"The uncertainty is plus or minus only a kilometer and a half per second."

The expanding universe

In the early twentieth century, astronomer Edwin Hubble determined that the galaxies in the universe are all moving away from the Milky Way because the universe is expanding. Further studies led astronomers to conclude that the rate of expansion is speeding up rather than slowing down.

McDonald compared the process to a ball thrown in the air.

"Acceleration is like you throw the ball up, and it starts going up faster and faster," McDonald said. "No normal attractive gravity will do that."

Astronomers determined that an unseen force, dubbed "dark energy," causes the acceleration. McDonald calls dark energy a "placeholder" because scientists aren't certain what, precisely, it is.

"To me, it seems quite possible that it's related to some fundamental hole in our understanding of physics," he said.

In order to patch that hole, scientists must continue to learn more about dark energy, including its role in accelerating the expansion of the universe.

http://www.eurekalert.org/pub_releases/2014-06/uocm-edo061814.php

Evolution depends on rare chance events, 'molecular time travel' experiments show

Chance events may profoundly shape history.

What if Franz Ferdinand's driver had not taken a wrong turn, bringing the Duke face to face with his assassin? Would World War I still have been fought? Would Hitler have risen to power decades later?

Historians can only speculate on what might have been, but a team of evolutionary biologists studying ancient proteins has turned speculation into experiment. They resurrected an ancient ancestor of an important human protein as it existed hundreds of millions of years ago and then used biochemical methods to generate and characterize a huge number of alternative histories that could have ensued from that ancient starting point.

Tracing these alternative evolutionary paths, the researchers discovered that the protein – the cellular receptor for the stress hormone cortisol – could not have evolved its modern-day function unless two extremely unlikely mutations happened to evolve first.

These "permissive" mutations had no effect on the protein's function, but without them the protein could not tolerate the later mutations that caused it to evolve its sensitivity to cortisol. In screening thousands of alternative histories, the researchers found no alternative permissive mutations that could have allowed the protein's modern-day form to evolve. The researchers describe their findings June 16, online in Nature.

"This very important protein exists only because of a twist of fate," said study senior author Joe Thornton, PhD, professor of ecology & evolution and human genetics at the University of Chicago.

"If our results are general – and we think they probably are – then many of our body's systems work as they do because of very unlikely chance events that happened in our deep evolutionary past," he added.

Thornton specializes in ancestral protein reconstruction, a technique that uses gene sequencing and computational methods to travel backwards through the evolutionary tree and infer the likely sequences of proteins as they existed in the deep past. Through biochemical methods, these ancient proteins can be synthesized and introduced into living organisms to study their function.

Thornton and others have previously shown that the evolution of modern-day proteins required permissive mutations in the past. But no one had ever investigated whether there were many or few other possible permissive mutations that could have happened, so it remained unknown how unlikely it is that evolution discovered a permissive pathway to the modern function.

To answer this question, Thornton and co-author Michael Harms, PhD, of the University of Oregon focused on the glucocorticoid receptor (GR), a key protein in the endocrine system that regulates development and stress responses in response to the hormone cortisol. They resurrected the gene for ancestral GR as it existed around 450 million years ago, before it evolved its capacity to specifically recognize cortisol. They included a handful of mutations that occurred slightly later that allowed the protein to evolve its cortisol recognition, but they left out the permissive mutations, rendering the protein nonfunctional.

Thornton and Harms then created millions of copies of this genetic template, using a method that introduced random mutations into every new copy, thus mimicking

the variation that evolution could have produced in the protein under alternative scenarios. To identify permissive mutations in these "might-have-been" pathways, they engineered yeast cells that could grow only if they contained a functional GR and then introduced their "library" of mutated versions of ancestral GR into them. If any of the mutations were permissive, they would restore the GR's function and allow the yeast to grow when exposed to cortisol.

Thornton and Harms tested many thousands of variants but found none that restored the function of GR other than the historical mutations that occurred in actuality. "Among the huge numbers of alternate possible histories, there were no other permissive mutations that could have opened an evolutionary path to the modern-day GR," Thornton said.

By studying the effects of mutations on the ancient protein's physical architecture, Harms and Thornton also showed why permissive mutations are so rare. To exert a permissive effect, a mutation had to stabilize a specific portion of the protein – the same part destabilized by the function-switching mutations – without stabilizing other regions or otherwise disrupting the structure. Very few mutations, they showed, can satisfy all these narrow constraints.

"These results show that contingency - the influence of chance events on the way evolution unfolds - is built into the atomic structure of molecules," said Irene Eckstrand, Ph.D., of the National Institutes of Health's National Institute of General Medical Sciences, which provided substantial funding for the research. "If the results hold true for other systems, this will be a highly significant contribution to our understanding of exactly how proteins can evolve new functions - a process that accounts for the diversity of life and the origins of genetic variation."

While most prior discussions of historical contingency in evolution have focused on external events such as asteroid impacts, mass extinctions, climate change, Thornton and Harms showed that the intrinsic complexity of proteins as physical objects also makes evolution depend profoundly on low-probability chance events.

"It's very exciting to have been able to directly study alternative ancient histories," Thornton said. "If evolutionary history could be relaunched from ancestral starting points, we would almost certainly end up with a radically different biology from the one we have now.

Unpredictable genetic events are constantly opening paths to some evolutionary outcomes and closing the paths to others, all within the biochemical systems of our cells.

The study, "Historical contingency and its biophysical basis in glucocorticoid receptor evolution," was supported by the National Institute of General Medical Sciences (R01-GM104397. R01-GM081592, and F32-GM090650), the National Science Foundation, and the Howard Hughes Medical Institute.

http://www.bbc.co.uk/news/science-environment-27926614

Computer-designed protein triggers self-destruction of Epstein-Barr-infected cancer cells

Delivered to its intracellular target via a novel carrier, 'BINDI' suppresses tumor growth and extends survival in a lab model of lymphoma

A protein molecule, "BINDI," has been built to trigger self-destruction of cancer cells infected with the Epstein-Barr virus.

Numerous cancers are linked to the Epstein-Barr virus, which can disrupt the body's weeding of old, abnormal, infected and damaged cells.

The Epstein-Barr virus persists for a long time after a bout with mononucleosis or other diseases for which it is responsible. It survives by preventing cells from disintegrating to kill themselves and their invaders. The virus' interference with cell population control may contribute to cancerous overgrowth.

In a June 19 report in the scientific journal Cell, researchers describe how they computer-designed, engineered and tested a protein that overrides the virus' interference. BINDI, they discovered, can prompt Epstein-Barr-infected cancer cell lines to shrivel, disassemble their components and burst into small pieces.

The BINDI protein was created at the UW Institute for Protein Design. (BINDI is an acronym for BHRF1-INhibiting Design acting Intracellularly.)

Lead authors of the paper are Erik Procko of the Department of Biochemistry and Geoffrey Y. Berguig of the Department of Bioengineering, both at the University of Washington. They collaborated with scientists and clinicians at the UW, Fred Hutchinson Cancer Research Center, Seattle Cancer Care Alliance and Scripps Research Institute.

The research team also tested the protein in a laboratory model of Epstein-Barr virus-positive lymphoma. Lymphoma is a type of cancer that can affect the lymph nodes, spleen, bone marrow, blood and other areas of the body. The researchers grafted lymphoma tissue onto mice as a living system to evaluate BINDI's therapeutic properties.

The scientists delivered the protein into cancer cells via an antibody-targeted nanocarrier newly designed to deliver protein cargo to intracellular cancer targets. BINDI behaved as ordered: It suppressed tumor growth and enabled the mice to live longer. "We are especially interested in designing proteins that selectively kill targeted cells," the researchers noted, "because they may provide advantages over current compounds that are toxic to other cells."

The work also demonstrates the potential to develop new classes of intracellular protein drugs, as current protein therapeutics are limited to extracellular targets. BINDI was designed to recognize and attach itself to an Epstein-Barr virus protein

called BHRF1, and to ignore similar proteins. BHRF1 keeps cancer cells alive, but when bound to BINDI, it can no longer fend off cell death.

By examining the crystal structure of BINDI, the scientists saw that it nearly matched their computationally designed architecture for the protein molecule.

"This close agreement between the protein model and the actual structure highlights the success in which designer toxins can be developed," the researchers said.

Among the scientists on this project:
William Schief, noted for his new approaches to vaccine development;
Oliver Press, a Hutchinson cancer center oncologist who studies and treats lymphoma and related disorders;
David M. Hockenbery, whose lab at Fred Hutchinson explores the mechanisms of programmed cell death;
Barry L. Stoddard, also at Fred Hutchinson, who is engineering therapeutic enzymes;
Patrick S. Stayton, a UW bioengineering professor and director of the UW Molecular Engineering and Science Institute, who works on new drug delivery systems;
David Baker, a UW biochemistry professor and director of the UW Institute for Protein Design, who has pioneered the computational design of proteins.

Grants from the National Institute of General Medical Studies at the National Institutes of Health (P41 GM103533, R01 GM49857, R21EB014572), Washington Life Sciences Discovery Fund and the U.S. Defense Threat Reduction Agency supported this project. Computational resources came from Berkeley Open Infrastructure for Network Computing, which received National Science Foundation support.

Stayton and Press are co-founders of PhaseRx Pharmaceuticals, which holds licenses for aspects of the new drug delivery carrier tested in this study, but indicated that the work reported is independent of the firm.

http://bit.ly/1iVrWMQ

Neanderthals evolved their teeth before big brains

The Neanderthals knew how to make an entrance: teeth first.

19:00 19 June 2014 by Colin Barras

Our sister species' distinctive teeth were among the first unique aspects of their anatomy to evolve, according to a study of their ancestors. These early Neanderthals may have used their teeth as a third hand, gripping objects that they then cut with tools.

The claim comes from a study of fossils from Sima de los Huesos in northern Spain. This "pit of bones" may be an early burial site, and 28 near-complete skeletons have been pulled from it, along with a large hand-axe that might be a funeral gift. The hominins in the pit look like Neanderthals, but are far too old. That suggests they are forerunners of the Neanderthals, and if that is the case they can tell us how the species evolved.

To find out, Juan Luis Arsuaga Ferreras at the UCM-ISCIII Joint Centre for Research into Human Evolution and Behaviour in Madrid, Spain, and colleagues studied 17 of the skulls. They found that the brain case was still the same shape as in older species. But the skulls' protruding faces and small molar teeth were much more Neanderthal-like.

Skull 17 of those found in the Sima de los Huesos, or "Pit of bones", in northern Spain
(Image: Javier Trueba/Madrid Scientific Films)

And… hold

This suggests the earliest Neanderthals used their jaws in a specialised way. It's not clear how, but it probably wasn't about food, says Ferreras. "There are no indications of any dietary specialisation in the Neanderthals and their ancestors. They were basically carnivores."

Instead, Ferreras suggests the first Neanderthals used their teeth to grip objects, giving them two hands free – one to steady the object and the other to cut it with a tool. "We guess that they were grasping a big piece of meat with the front teeth and cutting it into smaller pieces," he says. Many of the Sima front teeth have been scratched by tools, suggesting they sometimes missed.

The alternative is that their faces changed shape by sheer accident, says Jean-Jacques Hublin at the Max Planck Institute for Evolutionary Anthropology in Leipzig, Germany. "Some of these facial features might essentially have been fixed at random by genetic drift."

Last year, researchers extracted DNA from a 400,000-year-old bone from Sima de los Huesos. Unexpectedly, the DNA was similar to that of Denisovans, a little-known Asian group that lived much more recently. This suggests Neanderthals and Denisovans evolved from a common ancestor much like the Sima people, long after that ancestor split from us.

Journal reference: Science, DOI: 10.1126/science.1253958

http://www.eurekalert.org/pub_releases/2014-06/yu-ihm061914.php

In hairless man, arthritis drug spurs hair growth -- lots

A man with almost no hair on his body has grown a full head of it after a novel treatment by doctors at Yale University.

There is currently no cure or long-term treatment for alopecia universalis, the disease that left the 25-year-old patient bare of hair. This is the first reported case of a successful targeted treatment for the rare, highly visible disease. The patient has also grown eyebrows and eyelashes, as well as facial, armpit, and other hair, which he lacked at the time he sought help.

"The results are exactly what we hoped for," said Brett A. King, M.D., assistant professor of dermatology at Yale University School of Medicine and senior author of a paper reporting the results online June 18 in the Journal of Investigative Dermatology. "This is a huge step forward in the treatment of patients with this condition. While it's one case, we anticipated the successful treatment of this man based on our current understanding of the disease and the drug. We believe the same results will be duplicated in other patients, and we plan to try."

The patient had previously been diagnosed with both alopecia universalis, a disease that results in loss of all body hair, and plaque psoriasis, a condition characterized by scaly red areas of skin. The only hair on his body was within the psoriasis plaques on his head. He was referred to Yale Dermatology for treatment of the psoriasis. The alopecia universalis had never been treated.

King believed it might be possible to address both diseases simultaneously using an existing FDA-approved drug for rheumatoid arthritis called tofacitinib citrate. The drug had been used successfully for treating psoriasis in humans. It had also reversed alopecia areata, a less extreme form of alopecia, in mice.

"There are no good options for long-term treatment of alopecia universalis," said King, a clinician interested in the treatment of rare but devastating skin diseases. "The best available science suggested this might work, and it has."

After two months on tofacitinib at 10 mg daily, the patient's psoriasis showed some improvement, and the man had grown scalp and facial hair - the first hair he'd grown there in seven years. After three more months of therapy at 15 mg daily, the patient had completely regrown scalp hair and also had clearly visible eyebrows, eyelashes, and facial hair, as well as armpit and other hair, the doctors said.

"By eight months there was full regrowth of hair," said co-author Brittany G. Craiglow, M.D. "The patient has reported feeling no side effects, and we've seen no lab test abnormalities, either."

Tofacitinib appears to spur hair regrowth in a patient with alopecia universalis by turning off the immune system attack on hair follicles that is prompted by the disease, King said. The drug helps in some, but not all, cases of psoriasis, and was mildly effective in this patient's case, the authors said.

King has submitted a proposal for a clinical trial involving a cream form of tofacitinib as a treatment for alopecia areata.

He cited work by Columbia University scientist Angela Christiano as the reason he decided to try tofacitinib as a therapy in this patient with both alopecia universalis and psoriasis. She has shown that tofacitinib and a related medicine reverse alopecia areata in mice. King called her work exemplary and a clear example of how society's investment in science research leads to improvement in human life.

"This case highlights the interplay between advances in science and the treatment of disease," he said, "and it provides a compelling example of the ways in which an increasingly complex understanding of medicine, combined with ingenuity in treatment, benefits patients."

The paper is titled "Killing Two Birds with One Stone: Oral Tofacitinib Reverses Alopecia Universalis in a Patient with Plaque Psoriasis."

http://www.eurekalert.org/pub_releases/2014-06/cioe-stb061914.php

Single tick bite can pack double pathogen punch

Many blacklegged ticks infected with Lyme disease and babesiosis

Millbrook, NY - People who get bitten by a blacklegged tick have a higher-than-expected chance of being exposed to more than one pathogen at the same time. The new research, published online today in the journal PLOS ONE, was conducted by scientists at Bard College, Sarah Lawrence College, and the Cary Institute of Ecosystem Studies.

"We found that ticks are almost twice as likely to be infected with two pathogens—the bacterium that causes Lyme disease and the protozoan that causes babesiosis—than we would have expected," said Felicia Keesing, a professor of biology at Bard College, Adjunct Scientist at the Cary Institute, and co-author of the paper. "That means health care providers and the public need to be particularly alert to the possibility of multiple infections coming from the same tick bite."

Almost 30 percent of the ticks were infected with the agent of Lyme disease. One-third of these were also infected with at least one other pathogen. The agents of Lyme disease and babesiosis were found together in 7 percent of ticks.

The researchers collected thousands of blacklegged ticks from over 150 sites in Dutchess County, New York, an area with high incidence of tick-borne illnesses. They also collected ticks that had fed on different kinds of wildlife, including birds, rodents, opossums, and raccoons. Ticks acquire pathogens from feeding on infected hosts. DNA from each tick was extracted and tested for the presence of several pathogens.

"Mice and chipmunks are critical reservoirs for these two pathogens, so ticks that have fed on these animals are much more likely to be co-infected," said Michelle Hersh, an assistant professor of biology at Sarah Lawrence College, past postdoctoral researcher at the Cary Institute, and lead author of the study.

"Mice and other small mammals are often particularly abundant in habitats that have been fragmented or degraded by human activity," said Richard Ostfeld of the Cary Institute of Ecosystem Studies. "That means these patterns of co-infection might get worse through time as humans continue to impact forest ecosystems."

The researchers also considered another emerging pathogen, Anaplasma phagocytophilum, the bacterium that causes anaplasmosis in humans. Fortunately,

ticks were not more likely than expected to be co-infected with Anaplasma and the Lyme disease bacterium.

The researchers determined the proportion of ticks infected with each pathogen individually, then calculated the rates of co-infection expected by chance alone. Not only was co-infection with the agents of Lyme disease and babesiosis greater than expected, but rates of triple infection with the agents of Lyme, babesiosis, and anaplasmosis were about twice as likely as expected. "People in tick-infested parts of the United States such as the Northeast, Mid-Atlantic, and Upper Midwest, are vulnerable to being exposed to two or three diseases from a single tick bite," said Keesing. "And, of course, that risk increases when they're bitten by more than one tick."

For more information on this study, visit: http://dx.plos.org/10.1371/journal.pone.0099348.

http://bit.ly/1nWcBQl

Earth's Breathable Atmosphere Tied to Plate Tectonics?

A new study links continents and plate tectonics to the rise of oxygen on Earth.

By Johnny Bontemps - Jun 20, 2014

The rise of oxygen is one of the biggest puzzles in Earth's history. Our planet's atmosphere started out oxygen-free. Then, around 3.5 billion years ago, tiny microbes called cyanobacteria (or blue-green algae) learned out to carry out photosynthesis. They began using energy from sunlight to make their food from carbon dioxide and water, giving off oxygen as waste.

But it took another 3 billion years for oxygen levels to climb from trace amounts to at least 20 percent of the atmosphere, or high enough to support the emergence of complex life. And so far the mechanism behind that rise has remained unclear.

Now a new study by University of Exeter biogeochemist Benjamin Mills and his colleagues offers a new potential clue.

Using a computer model, they showed that plate tectonics may have fueled an increase in oxygen between 1.5 billion and half a billion years ago. In particular, a process tied to the way continents remove carbon dioxide from the atmosphere may have increased the supply of phosphorus, a key nutrient for photosynthetic microbes in the ocean. The paper was published this month in the Proceedings of the National Academy of Science. "This is a novel perspective for the late Proterozoic - a critical time of dramatic climate change, rising oxygen in the ocean and atmosphere, and origins and diversification of complex life," says Timothy Lyons, a biogeochemist not involved in the study.

From Seafloor to Terrestrial "Weathering'

Continents play a crucial role in the carbon cycle by removing carbon dioxide from the atmosphere. Carbon dioxide mixes with rain water, forming a weak acid (carbonic acid) which slowly wears down or "weathers" rocks on land. The process releases minerals such as calcium and magnesium from the rocks. These minerals then combine with carbonate and settle at the bottom of the ocean forming layers of calcium carbonate, or limestone.

In other words, the weathering process simply pulls carbon from the atmosphere and turns it into a layer of sediment on the seafloor.

However, continental rocks aren't the only route by which carbon is removed from the atmosphere. Ocean ridges, the places where fresh crust is made on the seafloor, can undergo a similar "weathering" process. In fact, seafloor weathering was the main route of carbon removal in the early chapter of Earth's history, before the formation of continents.

According to the new study, the rise of oxygen may have been due to a shift in balance between the two processes—between seafloor and continental weathering.

Potential Culprits

What caused that shift? The model looked at two factors: a brighter sun, and a slowdown in fresh crust production.

Our sun has slowly been getting brighter. It's now 20 to 30 percent brighter than when our Earth first formed. Because the terrestrial weathering depends on temperature, a brighter Sun may have sped up the process on land. What's more, the amount of fresh ocean floor formed has slowed down over time, limiting the amount of seafloor weathering (which occurs in newer ocean crust). Taken together, these two factors may have shifted the balance between the seafloor and the terrestrial processes.

The Phosphorus Boost

But why does that shift matter? "Rocks on continents contain phosphorus, which is a key limiting nutrient for photosynthetic microbes," Mills says.

The terrestrial weathering increases the amount of phosphorus in streams and rivers, and ultimately in the ocean. The amount of phosphorus then dictates how much photosynthesis occurs, hence how much oxygen is produced.

"The oceanic crust contains phosphorus too, but seafloor weathering is not able to liberate it, unlike weathering on the continents," Mills adds.

"The paper a great step forward," says Lyons. "The fundamental mechanistic perspective, particularly the co-consideration of seafloor and continental processes, is broadly relevant and clever."

One drawback, though, Lyons says, is that the model doesn't account for the shorter-term variations in oxygen. "The model, as proposed, isn't able to explain the details of the transition," Lyons adds. "But overall it still support the long term increase in oxygen."

Implications for Astrobiology

The study provides an indirect link between plate tectonic and continents on one hand and the evolution of complex life on the other, an idea worth keeping in mind in the search for life beyond our world.

“This is not the only reason oxygen rose to high levels, but it seems to be an important piece of the puzzle. Whilst the carbon cycle can function without large continents, it seems that their emergence was critical to our own evolution,” Mills says in a news release.

A shift from seafloor to continent weathering may have indirectly caused the rise of oxygen, which then led to the emergence of complex life on Earth.

A shift from seafloor to continent weathering may have indirectly caused the rise of oxygen, which then led to the emergence of complex life on Earth.

Mills later adds in a phone interview:

“A large number of nutrients, and not just phosphate, come from the continents. It seems that to develop a biosphere like we have on this planet, you’re going to need significant continental area.”

In fact, the recycling of continents via plate tectonics has become of major interest for many astrobiologists. Several have argued that, along with water, plate tectonics could be an essential requirement for life.

“What I like in particular is the rigorous links between tectonic drivers and oxygen (and life by association), which must be a considered in any view of extrasolar planets and their ability to sustain life through nutrient balances—with oxygenation as a possible consequence,” Lyons adds. “Plate tectonics and relationships to nutrient cycling, phosphorus in particular, should be an essential part in any exploration for life—on the early Earth and farther from home.”

http://bit.ly/1whT54w

Egyptologist unravels ancient mystery

It is one of the greatest archaeological mysteries of all times: the disappearance of a Persian army of 50,000 men in the Egyptian desert around 524 BC.

The Leiden Professor Olaf Kaper unearthed a cover-up affair and solved the riddle. It must have been a sand storm, writes the Greek historian Herodotus. He tells the story of the Persian King Cambyses, who entered the Egyptian desert near Luxor (then Thebes) with 50,000 men. The troops supposedly never returned; they were swallowed by a sand dune. A fantastic tale that was long the subject of many debates.

Egyptologist Olaf Kaper never believed it: 'Since the 19th century, people have been looking for this army: amateurs, but also professional archaeologists. Some expect to find somewhere under the ground an entire army, fully equipped. However, experience has long shown that you cannot die from a sand storm, let alone have an entire army disappear.'

Petubastis III

Kaper is now putting forward an entirely different explanation. He argues that the army did not disappear, but was defeated. 'My research shows that the army was not simply passing through the desert; its final destination was the Dachla Oasis. This was the location of the troops of the Egyptian rebel leader Petubastis III. He ultimately ambushed the army of Cambyses, and in this way managed from his base in the oasis to reconquer a large part of Egypt, after which he let himself be crowned Pharaoh in the capital, Memphis.'

The fact that the fate of the army of Cambyses remained unclear for such a long time is probably due to the Persian King Darius I, who ended the Egyptian revolt with much bloodshed two years after Cambyses' defeat. Like a true spin doctor, he attributed the shameful defeat of his predecessor to natural elements. Thanks to this effective manipulation, 75 years after the events, all Herodotus could do was take note of the sand storm story.

Kaper made this discovery accidentally; he was not looking for it actively. In collaboration with New York University and the University of Lecce, he was involved for the last ten years in excavations in Amheida, in the Dachla Oasis. Earlier this year, he deciphered the full list of titles of Petubastis III on ancient temple blocks. 'That's when the puzzle pieces fell into place', says the Egyptologist. 'The temple blocks indicate that this must have been a stronghold at the start of the Persian period. Once we combined this with the limited information we had about Petubastis III, the excavation site and the story of Herodotus, we were able to reconstruct what happened.'

The discovery will be announced on Thursday at an international conference. Kaper: 'I expect there to be a great deal of interest in the subject. I look forward to the discussions that will follow.'

http://bit.ly/1nueGQp

Researcher calls attention to vast, overlooked zone called 'aquaterra'

Think how people everywhere would marvel at the discovery of a continent lost beneath the sea, one that just a few thousand years ago played home to human civilizations that history has entirely missed.

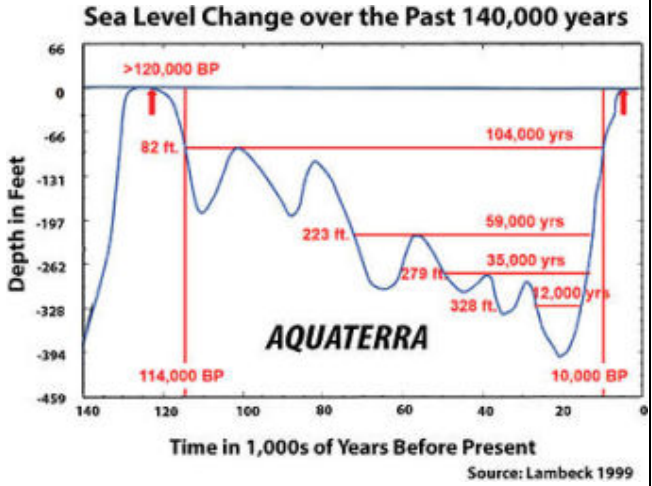
Phys.org -"The public would clamor for every shred of new insight on who those people were, how they lived and how they might be related to us," said Jerome Dobson, professor of geography at the University of Kansas. "Government programs would sponsor massive expeditions for exploration and scientific investigation. Geographers would rush to describe the land and people,

cartographers would map it, and they, together with all earth sciences, would strive to understand every aspect of it."

Dobson wants the same kind of scholarly resources to be focused on a little-studied geographical feature that he said holds insights about the natural and human world that would rival those of a true-life Atlantis.

Dobson calls the area "aquaterra," a new name for the previously undefined lands that were repeatedly exposed and inundated as ice sheets advanced and retreated over the past 120,000 years. "It's like a vast millennial tide," he said, "as glaciers hold and release waters to the oceans, and it's the same timeframe as the rise of modern humans."

Although it's scattered around the globe, in total aquaterra occupies as much space as North America, according to Dobson. He describes aquaterra and its potential for study in the latest issue of Geographical Review, published by the American Geographical Society.



Graph showing sea level change over the past 140,000 years.

"For some reason, no one seems to care much about the same amount of land scattered around the globe in intriguing, often strategic, places," Dobson said. "When scientists do mention aquaterra, they often call it a 'land bridge' as if ancient people only used it to get from one place we know today to another place we know today. This was not just a bridge. When sea level was low, aquaterra was a vast coastal plain with population densities at least as great as those in the lands above. There were houses, roads, villages and possibly cities. It was all coastal, all flat, and mostly tropical—clearly the best place to live during the ice ages."

Dobson underlines the importance of investigating aquaterra, which he describes as having a vertical relief of 400 feet and possessing the gamut of submerged landforms from tidal to wetland to upland—similar to coastal lands today. However, he said aquaterra should hold even more scientific and archeological treasures than modern coasts.

"First, sediments from glacial runoff during the ice ages would have deposited there, and some of those deposits may still be intact," Dobson said. "That's where scientists should look for evidence of biophysical processes occurring during the height of the ice ages. Second and most important, aquaterra is where we should look for evidence of the most advanced human cultures extant during the ice ages. Throughout history humans have settled the coasts in larger numbers than inland, and often our most advanced settlements have been seaside. Why would the pattern have been any different in pre-history?"

To better understand this untapped archeological and scientific resource, Dobson called on researchers to write grants and direct resources to systematically explore aquaterra—but he understands the general public will have to play a role.

"I'd like to see well-funded expeditions for exploration and research, but substantial support is unlikely in today's funding climate," he said. "Fortunately, however, new technologies are available to map and survey easier and cheaper than ever. I advocate a voluntary effort in which boaters and divers report observations to a central clearinghouse. It's called crowdsourcing and is part of the modern movement called popular geographics."

The effort would be huge, as aquaterra is composed of "the upper majority of the continental shelf and lower fringe of the current coastal plain of every continent and island"—parts of the globe that aren't readily accessible.

"Sonar sounding of ocean depths is easier in deep water than shallow," Dobson said. "Satellite gravimetry also encounters more vexing problems in continental shelf crust than deep oceanic crust. Plus, there's actually been more public interest in the mysteries of the deep than in the unknown shallows. Aquaterra may spark more interest in the shallow part."

Dobson has promoted aquaterra as fertile ground for research for almost 15 years, and, with his brother, has written two novels using aquaterra as a setting. He sees boosting aquaterra as the same thing as boosting human understanding.

"What's a university good for if we can't discover new ideas and excite people about them?" Dobson said. "Aquaterra holds the key to the greatest mystery of all: How did we humans evolve from the ice ages to today? In short, how did we get to be so smart?"

http://bit.ly/1prb0BN

Researcher invents continuous, zero-toxic-emission system that converts nonrecycled plastics into crude oil

Catalytic depolymerization system converts up to 10 tons of plastic per day into 60 barrels of oil, with zero toxic emissions

Plastic is becoming a major problem worldwide: In 2012, the United States alone produced roughly 32 million tons of plastic waste, while only recycling about 9 percent of its plastic, according to the Environmental Protection Agency. This is because of the growing use of "nonrecycled" plastics, primarily made of

polystyrene and polypropylene. Seeing little return value, recyclers toss these plastics into landfills, where they pile up and never decompose. As a result, landfill space is becoming a concern.

But now MIT spinout PK Clean, founded by Priyanka Bakaya MBA '11, aims to end the landfilling of plastic with a cost-effective system that breaks down nonrecycled plastics into oil, while reusing some of the gas it produces to operate.

"Plastic comes from oil to begin with, so it makes sense, instead of landfilling plastic, to convert it back to usable fuel," Bakaya says. "The goal is to end landfilled plastic waste forever—not just domestically, but also globally."

PK Clean's so-called "continuous" system—the first of its kind in the United States, according to Bakaya—runs on a process called catalytic depolymerization, where heat and a catalyst break down plastics into crude oil to sell to refineries.

About 70 to 80 percent of the product comes out as oil. Roughly 10 to 20 percent becomes hydrocarbon gas that heats the system, while the remainder is char residue.

Following a trial in Pune, India, PK Clean last year built and installed its first full-scale commercial plant in Salt Lake City, partnering with Rocky Mountain Recycling, Utah's largest recycler.

Operating continuously, the plant can convert up to 10 tons of plastic per day into 60 barrels of oil, with zero toxic emissions. Produced at around $35 per barrel, the oil is sold to a nearby refinery for around $100 per barrel.

After nearly a year of operations in Utah, PK Clean plans to partner with other recyclers across the nation. Eventually, Bakaya says the plan is to move to developing countries, "where plastic waste is even more of an issue."

Pushing the envelope in design

Plastics come in seven categories: Type 1 (such as water bottles and soda bottles) and type 2 (foggy plastics, such as milk cartons) are easily recycled. But types 3 through 7—including plastic foam, disposable utensils, plastic pipes, food-storage containers, and shampoo bottles—are either not easily recycled or unrecyclable.

To convert these plastics into oil, PK Clean first shreds them. The shreds are then entered into a reactor—which runs at about 400 degrees Celsius—where a catalyst helps degrade the plastics' long carbon chains. This produces a vapor that runs through a condenser, where it's made into oil.

Systems using similar processes have been around for years. But these have been too energy-inefficient and costly for recyclers to adopt. On the other hand, PK Clean's system, Bakaya says, costs a quarter the price of other systems to run, while producing greater yields.

"We had to push the envelope with the design and operating costs to make something that can be adopted and easily used," Bakaya says.

Much of the system's innovation is in its continuous operation. Other systems operate through "batch processing," where reactors heat up and then cool down again before the next batch is ready—wasting significant energy and money. But the hydrocarbon gas produced by PK Clean's system maintains the reactor's heat, avoiding constant rebooting and energy loss.

Additionally, PK Clean adds a catalyst that helps produce greater yields in the conversion process. Automated controls also make the system much easier to use.

Within two years, Bakaya says, PK Clean aims to produce more refined fuel that recyclers can immediately pump back into their recycling trucks, without the need for oil refineries.

"The system is pretty close, but we have to be on the exact specs, so we'd rather let a refinery handle that now," Bakaya says.

Throughout 2011, PK Clean won awards and funding from tech entrepreneurship competitions, including the MIT Clean Energy Prize (track winner), the MassChallenge (winner), the Rice Business Plan Competition (best energy business plan), and the Cleantech Open (runner-up for the national grand prize, and track winner).

Since then, Fortune, Forbes, Inc., and other publications have praised the company for its innovation, and lauded Bakaya as a top entrepreneur in clean energy.

A clean-tech journey

Growing up in Australia, Bakaya was introduced to clean technologies through a close family friend, inventor Percy Kean—the creator of PK Clean's catalytic depolymerization technology, and the "PK" in the company's name.

Kean had spent decades researching and inventing clean technologies in his home, even turning his kitchen into a lab. Bakaya and her family would visit him often. "He'd show me oil samples, light it with a match, and say it came from waste. That sparked my imagination," she says.

Those fond memories lingered during Bakaya's undergraduate years at Stanford University, and during her career forecasting oil prices on Wall Street in the mid-2000s. Oil had then crept up to about $140 per barrel, ushering in a new demand for clean energy. So when Kean died in 2007, Bakaya set out to commercialize his work.

Visiting a friend who was studying chemical engineering at MIT, Bakaya sat in on an MIT Sloan School of Management class, 15.366 (Energy Ventures)—taught by Bill Aulet, managing director of the Martin Trust Center for MIT Entrepreneurship—where she saw a path forward.

"I thought, 'Wow, this is exactly what I need to get started on setting up this company,'" she says.

Enrolling in MIT Sloan, she spent two years building her company. Classes such as 15.366, where students incrementally design business plans, "really push you to think about all aspects of the business," Bakaya says.

Through that class, she also met graduate student Arjun Gupta SM '11, who helped with early designs of the system.

Outside the classroom, she frequented MIT's Entrepreneurship Club and Energy Club, where she found support among dozens of other hopeful entrepreneurs.

"Having other people going through the same thing is something I wouldn't have had if I hadn't gone to MIT," Bakaya says. Today, this close group of 30 to 40 budding entrepreneurs still help one another promote their businesses via social media.

After graduation, she says, PK Clean benefitted—and continues to benefit—from the "MIT brand name." Apart from lending credibility to the technology, Bakaya believes coming from MIT helped the company find early investors, and get chosen for a Bay Area incubator.

"Looking back, those three things at MIT—the classes, the network, and the brand name—have been an enormous help in launching the company and getting started," Bakaya says. "PK Clean wouldn't be possible without MIT."

More information: www.pkclean.com/

http://bit.ly/1j0pzZ8

Germany sets record for peak energy use – 50 percent comes from solar (Update)

Germany set a record high for solar use on June 9

The Fraunhofer ISE research institute has announced that Germany set a record high for solar use on June 9—on that day the country's solar power output rose to 23.1 GW—50.6 percent of all electricity demand. The record occurred over a holiday, which meant less demand, but it still marks a major step forward for the world's solar power leader.

Despite not having a generally sunny climate, Germany has been pushing solar energy—but not from the huge solar farms seen in other countries. In Germany, the focus has been on rooftop solar collectors mounted on homes, businesses and buildings of any other kind. Currently, over 90 percent of mounted solar panels in the country are on rooftops. The country broke two other records around the same time, producing 24.24 GW of solar generated power between 1 and 2pm on June 6, and over that entire week, the country produced 1.26 TWh of electricity from solar power. In stark contrast, recent reports indicate that solar power makes up just 0.2 percent of total energy production in the U.S.

The popularity of solar panels on rooftops has been bolstered by generous solar subsidies from the government along with a successful ad campaign. The movement is part of a plan by the German government to reduce greenhouse emissions due to electricity being produced in coal fired power plants and a simultaneous phasing out of nuclear power plants (all such plants are scheduled for closure by 2022). That leaves solar, wind and biomass—the country has been eagerly pursuing all of them, though clearly solar has become the national leader.

The move to solar has not been without its problems, of course. The government plans to lower or remove subsidies as soon as possible and the demand for batteries to store all that home-grown electricity is outstripping demand, causing a rise in prices. Also, it's not clear what sort of role utilities will play going forward—currently, many homeowners are reporting surplus energy production on sunny days which they sell to electric companies, which now find themselves having to store it for use during cloudy stretches.

There's another problem too, though it's not as obvious—the German government noted recently that almost seven million households in the country are living in energy poverty—defined as having to spend more than 10 percent of income on energy bills. The national energy program, Energiewende, has resulted in some transfer of wealth, economists note—even with subsidies, it's generally the wealthy (and sometimes the middleclass) who can afford to put solar panels on top of their house—the poor continue to live off the grid and pay taxes that provide the funds for the subsidies. There's also some evidence that the country's energy program is pushing energy costs higher overall, resulting in more electricity being produced by cheaper fossil fuels.

http://www.bbc.com/news/health-27940932

Stem cell treatment for horses to be trialled on humans

"No-one has ever put a stem cell into a human Achilles tendon before in the UK," says Andy Goldberg, a consultant orthopaedic surgeon at the Royal National Orthopaedic Hospital.

But all of that is about to change.

Dr Goldberg is leading the first trial of a new treatment which has been working wonders in racehorses - and could produce the same results in humans. The stem cell treatment for what's known as "tendinopathy" in racehorses has been so successful that one horse to receive the treatment, Dream Alliance, went on to win the 2009 Welsh Grand National. The technique also saw the re-injury rate of the treated horses fall by 50%.

In humans, the condition is called Achilles tendinopathy and causes severe pain in the heel. About 85,000 people are affected by the problem in the UK each year.

At present there are limited options for treating the condition apart from surgery, but stem cells offer a different solution because of their ability to regenerate.

Exploring the use of stem cell treatments in racehorses provided the perfect test-bed for humans because, Dr Goldberg says, "horses have similar problems to tendon problems found in humans". "Their injuries are akin to human injuries. We've been able to solve the problem in horses so the next step is to translate it into humans," he says.

Horses are a good animal model for this condition because it occurs as naturally in racehorses as it does in male and female athletes. That is why Dr Goldberg has been working closely with the Royal Veterinary College, which pioneered the stem cell treatment in horses.

Dr Jaysh Dudhia, senior lecturer at the Royal Veterinary College, says horses will inevitably play a big role in the future as natural disease models, but they are not the only animals that could help increase understanding of human conditions.

Dogs suffer from similar hip problems and other large dogs develop similar cardiac problems to humans. Cats appear to suffer from hypertrophic cardiomyopathy, a disease of the heart muscle, which is also seen in humans. Ageing dogs also display signs of cognitive decline, which mirrors Alzheimer's disease in humans.

"At the small-animal hospital, we are working hard to investigate these connections," Dr Dudhia says.

What are stem cells?

A stem cell is a cell capable of becoming another cell type in the body, such as a skin cell, a muscle cell or a nerve cell

Because of their ability to become different types of cells they offer the potential to treat degenerative conditions and illnesses

Stem cells could be used to treat spinal cord damage, sports injuries, bone, cartilage and tendon damage, blood cancer, diabetes, stroke and heart disease

Source: UK Stem Cell Foundation

Only 10 patients are involved in the first study of the stem cell treatment that proved so successful for horses. It will take place at the Royal National Orthopaedic Hospital and University College London. The patients will each have stem cells taken from their pelvis, which will then be left to grow in the lab for four to five weeks, before being implanted directly into their damaged Achilles tendon.

Over the next six months, the tendons will be measured using a special 3D colour scan, to see if the treatment has been successful in regenerating the tendon. "Before we relied on the body repairing it," says Dr Goldberg. "But a degenerative problem need stem cells."

The process of repair could work in two ways, he says. Either the stem cells will turn into new tendon cells, or the stem cells will encourage other cells around them to form new healthy cells. The UK Stem Cell Foundation is funding the study and hopes this could lead to new treatments within the next three to five years.

http://bit.ly/1lJqt0g

Scientists discover link between climate change and ocean currents over 6 million years

Scientists have discovered a relationship between climate change and ocean currents over the past six million years after analysing an area of the Atlantic near the Strait of Gibraltar, according to research published Friday, 13 June in the journal Science.

An expedition of scientists, jointly led by Dr Javier Hernandez-Molina, from the Department of Earth Sciences at Royal Holloway, University of London, examined core samples from the seabed off the coast of Spain and Portugal which provided proof of shifts of climate change over millions of years.

The team also discovered new evidence of a deep-earth tectonic pulse in the region, as well as thick layers of sand within mountains of mud in a vast sheet, spreading out nearly 100km into the Atlantic from the Gibraltar gateway. The quantity of sand is far more than was expected and has been caused by the strength, speed and long duration of bottom currents flowing through the Strait of Gibraltar from the Mediterranean.

“The sediments we examined show various shifts of climate change over millions of years”, Dr Hernandez-Molina said. “In addition, our findings could herald a significant shift in future targets for oil and gas exploration in deep-water settings. The thickness, extent and properties of these sands make them an ideal target in places where they are buried deep enough to allow for the trapping of hydrocarbons. The sand is especially clean and well sorted and therefore very porous and permeable.”

The expedition, carrying an international team of 35 scientists from 14 countries, recovered 5km of core samples from an area along the Gulf of Cadiz and west of Portugal.

The research found that a powerful cascade of Mediterranean water spilling into the Atlantic was scouring the rocky seafloor, carving deep-sea channels and building up mountains of mud. This is due to Mediterranean water being saltier than the Atlantic and therefore denser, causing it to plunge downwards.

Dr Hernandez-Molina added: “We set out to understand how the Strait of Gibraltar acted first as a barrier and then a gateway over the past six million years. The fascinating results we came back with have hugely increased our understanding of the Mediterranean Outflow Water (MOW) that flows through the Gibraltar gateway and have led to some key discoveries about the relationship between climatic shifts, deep-water circulation and plate tectonic events over a huge timescale.”

http://www.bbc.com/news/health-27814124

'Shoebox IVF' hope for infertile couples

Could an IVF kit that fits in a shoebox and some kitchen cupboard essentials provide hope for people who long for children?

By Cathy Edwards BBC Health Check

Infertility is a source of distress the world over, but in many places the terrible stigma attached to childlessness makes it even harder to bear. The answer could be a pared-down system that can fit inside a shoebox and uses cheap ingredients you might find in a kitchen cupboard.

In the past infertility has been neglected in developing countries, partly because of a focus on controlling overpopulation. But experts argue that true reproductive health has to address both sides: family planning for those who want to avoid pregnancy, and fertility treatment for those who long to have children but can't conceive naturally.

Nosiphiwo, from South Africa, had been trying to conceive for years when her husband's family asked her for their lobola back - the bride price they paid when she married their son. She was ostracised by her in-laws for being childless, and felt cut her off from the rest of her community too. She says women in her situation sometimes turn to suicide. "I thought of doing that. Because you don't have any option."

Sophisticated labs

The prohibitive costs of fertility treatment mean that worldwide, most couples cannot afford it - though their desperation can be such that many become destitute trying to pay for it, selling property or going into debt. One of the biggest obstacles is the cost of the complex, sophisticated labs where "in vitro" egg fertilisation takes place.

Belgian obstetrician Dr Willem Ombelet worked in South Africa in the 1980s and saw many cases like Nosiphiwo's. He carried out IVF treatment for those who could afford it. Those who couldn't came to the hospital on other pretexts - but the real reason was their longing for a child. "They would wait shyly around the corner and ask if there was anything we could do for them." Back then, the heartbreaking answer was no. But he has campaigned ever since to improve global access to infertility care, co-founding The Walking Egg non-profit organisation to raise awareness of fertility in developing countries.

DIY embryo transport

The centrepiece of the Walking Egg's mission is a simplified system for egg fertilisation. The best conditions for a sperm to fertilise an egg outside the body are slightly alkaline, at a temperature of 37C (98F).

Usually this involves a sophisticated laboratory equipped with huge ventilators, complex incubators and a supply of expensive gases. But when Dr Ombelet met the embryologist Jonathan Van Blerkom in 2008 the idea of a cheap, portable lab was born. Van Blerkom revived a technique he used in the 1980s when transporting cow embryos long distances across Nebraska. By mixing baking soda and citric acid he created his own CO2, periodically adding it to the solution holding the embryos to maintain the optimal CO2 concentration and alkalinity levels.

IVF in a shoebox

For humans the technique had to be refined to create a closed system and thus minimise any risk of contamination. Precise quantities of citric acid and sodium bicarbonate are mixed in one test tube. The CO2 bubbles this creates are fed via a tube into a second test tube containing a culture medium for the embryo.

To maintain the perfect temperature for egg fertilisation and embryo development, Van Blerkom tried out various low-tech methods. "I put the test tubes into a thermos at the right temperature - that worked. I put them in an aluminium heating block, and that worked too. The embryos didn't care if they were in an expensive triple walled incubator or a thermos flask." Once the atmosphere has stabilised, the egg and then the sperm are injected into the test tube containing the culture medium. The next day this test tube goes under a microscope to see if it contains an embryo - meaning egg fertilisation has taken place. If a successful embryo is created, it is transferred from the test tube to the woman's womb after about six days.

This simplified system reduces the whole IVF lab to an aluminium heating block containing one pair of test tubes for each embryo, all inside a shoebox-sized container. For additional safety the human trials of the system have so far been conducted inside a sterile laboratory.

The team are developing a self-contained unit to house the system in hospitals or health centres that don't have advanced lab facilities. This would provide heated, sterile air and space to examine the embryo under a microscope.

The researchers believe that - because of the closed nature of their system - this unit is not strictly necessary, but will help convince health authorities of the quality of the system.

"Embryo quality"

Trials began in Genk, Belgium in 2012, and so far 17 healthy babies have been born using the system. Dr Ombelet is thrilled with their preliminary results, saying they indicate fertilization and pregnancy rates are similar to expensive IVF methods. "We have proved that with our system embryo quality is at least as good as with regular IVF."

Geoffrey Trew, a consultant in reproductive medicine and surgery at Hammersmith Hospital in London who is not connected with the research, agrees this is an

exciting technique. "It has been shown to work in a developed country. Now we've got to see how well it is reproduced in the developing world where the conditions are more fickle."

Fertility on a shoestring

The trials are due to be rolled out in South Africa and the UK later this year, and the team hope that by early next year the system can be tested in the kind of low-resource settings it was designed for.

Each IVF cycle costs less than 200 euros (£159) using this system, not including staff and medication costs, which vary from country to country. But Dr Ombelet says they can decrease the normal price for IVF in any given country by at least 70-80%. "With very low dose medication schemes we hope to perform IVF in developing countries for less than 500 euros (£399)"

Prof Thinus Kruger and Dr Matseseng are fertility experts from Tygerberg Hospital in Cape Town. They already have a special fertility programme that cuts costs by economising on medication and staffing - Nosiphiwo was one of the many women who was helped to conceive by this programme.

Now they want to see how the tWE system compares to their normal laboratory procedure. "It's really theoretically amazing," says Professor Kruger.

"But we will have to see how patient and scientist friendly this system is. It is a little lab, so you still need the knowledge to handle those small embryos."

Prof Van Blerkom believes that efforts to bring the cost of fertility treatment down would please the IVF pioneer Robert Edwards, whose work led to the birth of the first test tube baby, Louise Brown.

"People can make fortunes through IVF. But Bob Edwards was a real believer that IVF should be universal, because he knew the suffering that infertility caused."

http://www.rawstory.com/rs/2014/06/22/nasa-our-plan-is-to-colonize-mars/

NASA: 'Our plan is to colonize Mars'

Dr Ellen Stofan says that missions to the red planet are a priority of the US space agency – and that the best way to search for extraterrestrial life is by setting up a permanent presence. Interview by Nicola Davis

By Nicola Davis, The Guardian

Is NASA looking for intelligent life?

NASA, right now, is really taking a step-wise approach: let's look at our own solar system and the most likely places where we might find life. That's why we are so focused on Mars, because we know Mars had liquid water on the surface and we think that is essential to life. What we expect to find, certainly in our own solar system, are probably simple single or multiple-cell forms of life. To get to intelligent life takes stability of conditions over huge long periods of time. [We're] not sure that condition exists anywhere else in our solar system. But certainly when we go out and look for habitable planets around other stars it's something that we can start thinking about.

The Kepler mission has found planets orbiting stars other than our sun. What is the impact?

It's turned around our understanding of how our own solar system formed, because when you only have one solar system to study you make assumptions that are based on that information. [When] you have many solar systems to study, many planets to study, it is really making us rewrite textbooks. We're launching the James Webb space telescope in 2018 that is going to study the atmospheres of those planets around other stars.

Some might argue that it's a luxury to be spending money investigating other planets instead of solving Earth's problems …

I always like to say just think you were a doctor with only one patient. You might understand how that person gets sick, how they get better, but you understand nothing about the progression of disease or how humans in general get ill. Now take an Earth scientist: you only have one planet to study. Our studies of other planets are really what we call comparative planetology. Think of the other planets as being simpler versions of the Earth where you've tweaked the physical conditions, maybe the composition, the density of the atmosphere. It allows us to rip apart the physics of some of these problems and give us a better comparison.

Mars doesn't seem to be teeming with life. Aren't we better looking at planets where there might be something alive and kicking?

On Mars we are not totally sure – there could be something still living under the surface. Mars is close by, humans can operate and work on the surface – that's part of why we are so focused on [it]. Over the last few years we have started to formulate the next mission to [Jupiter's moon] Europa – we know there is an ocean under that icy crust. There are plumes of water coming out of the cracks in the south polar region. There's orange gunk all over the surface – what the heck is that stuff? Huge questions about Europa – it is clearly our next step. We have [also] flown the Cassini spacecraft through the geysers erupting off [Saturn's moon] Enceladus – we know there are organics in those water plumes but we don't know how complex those organics are. So here we have three really rich targets.

Is NASA going to send humans to Mars just to show that it can?

Well, I'm biased because I'm a field geologist. Humans can actually read a landscape, go through a lot of rocks – crack them open, throw them, pick up the next one. Rovers are great, they do amazing science, but it is a lot more tedious process – they go much less far than a human can cover in a day. Having humans on the surface is how I think we are going to be able to demonstrate totally conclusively that life did evolve on Mars.

There is a lot of talk about settling Mars. Will NASA be bringing its astronauts back?

We would definitely plan on bringing them back. We like to talk about pioneering Mars rather than just exploring Mars, because once we get to Mars we will set up some sort of permanent presence.

Given NASA's shuttle program ended in 2011, are the astronauts going to have to hitch a ride?

We are working right now to return our capability of launching astronauts from the US through a programme we call "commercial crew". Right now there are three companies that are still interested in developing the capability to launch humans from US soil. We are also, at the same time, developing our own rocket called the "space launch system", which is a new NASA rocket that will be a heavy lift vehicle that will be able to get humans out to the lunar vicinity to do this asteroid work, and then it will eventually have the capability to get humans on the path to Mars.

I heard there are plans to send humans to an asteroid that has been kidnapped and put in orbit around the moon. Seems pretty radical …

We are actually looking at two different mission concepts and trying to decide between them. One would be to find a small asteroid – so less than 10 meters across – basically encase it in a bag and bring it back with us. That enables us to test a type of propulsion system called solar-electric propulsion that we would need to use to take large amounts of cargo to Mars. The other option is to go to a larger asteroid and grab a boulder and bring the boulder back. All of the asteroid mission is actually testing capabilities and technologies that we need for the Mars mission.

Isn't there a risk that we will contaminate Mars?

It's a huge concern. First, of all you wouldn't want to bring any weird microbes from Mars to the Earth that could potentially be harmful to people here. In the future when we have Mars samples come back, they will go through an incredible procedure basically equivalent to an ebola level quarantine facility to make sure they are not going to contaminate Earth. Then there is contamination of Mars. We are looking for life on Mars so we don't want to carry microbes with us, "discover them" and declare victory. All spacecraft that go to Mars go through an incredible level of decontamination, they get swabbed all the time for microbes before they leave – also for spacecraft that go anywhere near Europa, anywhere near Enceladus, because we are really concerned about contaminating these watery worlds. Obviously when we send people, it's going to be a huge concern. We're kind of dirty things so keeping Mars uncontaminated at that point is going to take some work.

You were principal investigator on Titan Mare Explorer (TiME), a "boat" designed to land on the hydrocarbon seas of Saturn's moon, Titan. Has that idea been sunk?

It was like a little floating buoy that would make measurements, not that different from what ocean buoys here on Earth make. We made it through the final round of a competition and then got defeated [in 2012] by a mission to Mars called "InSight" that's going to measure seismic activity on Mars. The [TiME] has now been turned over to one of my colleagues – a guy named Ralph Lorenz at Applied Physics Lab – so what happens to the mission in the future will be up to Ralph.

Is a tight budget compromising NASA's position as a leading space agency?

We have a really vigorous space program with the budget that we have, and we've actually gotten extremely favorable budgets in the last few years. So I feel like we are in great shape.

So having a tight budget focuses the mind?

I think it focuses your mind on priorities and, frankly, I also think it fosters innovation. I'm not out there asking for budget cuts to create innovation, but on the other hand NASA's budget is always going to be limited – it is one of many priorities of the US federal government. In that limited-budget situation we want to do amazing things so what we need are innovations – using technology in creative ways, approaching problems sideways. That's why we need [a] diverse workforce – you need people coming in with all kinds of backgrounds, multiple points of view, to look at problems sideways.

How does NASA view the space race between India and China?

I think it is really exciting when we see how many countries are coming into space programs. When you look at the global exploration roadmap – this path to Mars – we have 12 countries involved in that. These things we are trying to do are hard, going to Mars is hard. It's not a US thing, it's not a US and Europe thing, it's a worldwide effort to try and accomplish these things.

All these missions create space junk – is that a problem?

We move the international space station (ISS) every year, several times a year, to have it avoid debris in space so it's certainly something we keep an eye on.

Is there any doubt about the future of the ISS given tensions between Russia and America over the Ukraine situation?

We have excellent relationships on the working level with the Russians. We have not received any official notification that anything other than what is occurring now is going to occur. Our feeling is Russia has been a great partner on the ISS, and we hope that will continue.

Do you have a NASA space pen?

I don't, actually. We get little badges, that's about it.

http://www.bbc.com/news/science-environment-27957274

Titan: Clue to 'Magic Island' mystery on Saturn moon

Scientists have outlined their best explanations for a mysterious feature dubbed the "magic island", which has been spotted on Saturn's moon Titan.

By Paul Rincon Science editor, BBC News website

The Cassini spacecraft captured the "island" during a flyby, but it had vanished by the time of the next pass. The bright splodge is seen in Ligeia Mare, one of the seas of methane and ethane found at Titan's north pole. Icebergs, waves and gas bubbling up from the sea bed are all possibilities, the scientists say. The study by an international team has been published in the journal Nature Geoscience.

The bright feature was spotted in images from July last year, but a few days later it had vanished

Saturn's largest moon shares much in common with Earth, such as a substantial atmosphere and a seasonal cycle. Wind and rain shape the surface to form river channels, seas, dunes and shorelines. Titan's mountains and dune fields are made of ice, rather than rock or sand, and liquid hydrocarbons take many of the roles played by water on Earth. The seas and lakes peppering the moon's north polar region are filled with methane and ethane. These are gases on Earth, but at typical Titan temperatures of -180C, they exist in a liquid state.

Titan - 'Looking-glass Earth'

Titan is Saturn's largest moon and the second biggest in the Solar System
It is the only moon in the Solar System with clouds and a substantial atmosphere
Wind and rain create similar features to those found on Earth, such as dunes, lakes and rivers
But on Titan it rains liquid methane, filling the rivers, lakes and seas with hydrocarbons

The bright feature was spotted in pictures from a Cassini flyby of Titan on 10 July 2013. The "island" is absent in imagery of Ligeia Mare taken on three previous flybys. By the time of the next pass of Titan, on 26 July, the feature had vanished, and was not visible in two subsequent flybys.

"'Magic island' is a colloquial term that we use within the team to refer to this. But we don't actually think it's an island," co-author Jason Hofgartner told BBC News. The feature appears and disappears too quickly to be a volcanic islet. So the team were left with a handful of potential explanations.

Mr Hofgartner, who is based at Cornell University in New York, explained: "We have four different hypotheses that are all equally preferred. In no particular order they are: waves, rising bubbles, floating solids and suspended solids."

Titan operates on a 30-year seasonal cycle, and the moon's northern region is expected to become a more dynamic place as Titan approaches its summer solstice in May 2017. "Right now, Titan is basically half way between the vernal equinox (August 2009) - at the beginning of spring - and the summer solstice, the start of summer. It's roughly equivalent to what we would consider the beginning of May," said Mr Hofgartner. "As Titan approaches its summer, more of the Sun's energy is being deposited in the northern hemisphere." Winds will get stronger, causing an increase in waves, which are one potential explanation for the "magic island". Researchers have already seen possible evidence for small waves on another Titan sea.

Iceberg ahoy?

Another intriguing possibility is that of floating or suspended solids, including icebergs. However, any 'bergs couldn't be made of water-ice - which, because of its relatively high density, would sink in a liquid hydrocarbon sea. Instead, icebergs on Titan would have to be made from a frozen mixture of methane and ethane.

A previous study by Mr Hofgartner and Prof Jonathan Lunine, also of Cornell, suggested conditions on Titan might cause methane-ethane ice to sink in the winter and float in summer.

But the moon's surface is also thought to be covered in various organic (carbon-based) compounds, including one, polyacetylene, with a density low enough to allow it to float. It could be suspended below the surface of a sea much like silt in a terrestrial river delta.

The final possibility is that Cassini captured gas bubbling up to the surface from a subsea volcanic vent. The abundant methane in Titan's atmosphere must be continually replenished, because the molecule can only exist for a short time before it is destroyed by UV rays.

Although researchers haven't yet found the "smoking gun" of volcanic activity on Titan, it remains one of the best candidates for the source of the moon's methane.

John Zarnecki, an emeritus professor at the Open University in Milton Keynes, co-authored the first paper to predict wave heights on Titan. But he said there was little evidence from more recent observations that winds on the moon could raise waves big enough to detect.

Speaking to the BBC from Rio de Janeiro, where he has been following England at the World Cup, Prof Zarnecki referred to the waves he could see crashing on to Copacabana Beach, and said: "I'd love to think that this paper represents the first positive indication of a similar phenomenon on Titan."

He added: "These are clearly observations that are close to the limit of detectability - and therefore very difficult to interpret. But it looks like something is going on in Ligeia Mare. Titan surprises us at every turn.

"Is this feature showing us floating solids or gases erupting at the surface - or a phenomenon that we haven't thought of? After all, we tend to think in terms of Earth-like phenomena. But based on this so far sparse data, any suggestion is likely to be little more than speculation until we get some more supporting information."

The authors of the latest study hope that future observations by Cassini might yield evidence of similar phenomena. And if they do, there might be a chance of distinguishing between different possible causes. For example, if a similar feature is seen to move its location on successive passes by Cassini, it could be suggestive of an iceberg being moved by currents.

Prof Zarnecki commented: "This is just further evidence, if we need it, that we just must go back to Titan with a dedicated mission, ideally to land in one of Titan's seas - a Titan Sea Probe. And then we can understand what is happening on the seas of this incredible place."

http://bit.ly/T2VeSL

Scholars and scientists explore factors underlying serendipitous discoveries

What do Velcro, Tang, penicillin, the structure of DNA and the World Wide Web have in common?

They all involved serendipitous discoveries—chance discoveries made by alert, curious scientists who were looking for other things when they happened across a fortuitous finding. Rather than ignoring their accidental discoveries, these curious, open-minded scientists harnessed their luck. "Chance favors only the prepared mind," as Louis Pasteur put it.

Since serendipity has played such a big role in science, the Office of the Vice President for Research and for National Laboratories at the University of Chicago selected serendipity as the topic for its ninth Joint Speaker Event. The series is designed to promote dialogue and collaboration among University faculty members and scientists, researchers and engineers from Argonne National Laboratory and Fermi National Accelerator Laboratory.

"Science and Serendipity: Happenstance and other Factors Underlying Accidental Discoveries" was held last month at the new Chicago Innovation Exchange. This hub for multidisciplinary collaboration and support for business start-ups was a perfect setting for the event because "the Exchange is about engineering serendipity: putting people together that need to be together—but would not otherwise be together," said John Flavin, executive director.

Panelists discussed several examples of serendipity in science. Radar was developed to watch airplanes in the sky, and scientists were initially frustrated when other things showed up on their screens. Little by little, however, they appreciated the ability to use radar to track weather patterns; see flocks of birds and swarms of insects; and study the dynamics of animals in flight. "Meteorologists, ecologists and other scientists got interested and, thus, a series of serendipitous events in radar led to the development of entire branches of science," said panelist Rick Stevens, professor of computer science at the University and associate lab director for computing, environment and life sciences at Argonne.

Another good example of serendipity is the work of Charles Darwin, according to panel moderator Robert Richards, professor of philosophy, history and psychology at the University. Had Darwin not failed in medical school, been recommended by a professor to go on the Voyage of the Beagle, consulted with John Gould, the leading ornithologist at the British Museum, and read Thomas Malthus' An Essay on the Principle of Population,—"for amusement," as Darwin put it—he might never have come up with the theory of Natural Selection, Richards said.

In an experiment designed to determine whether protons decay, 3,300 tons of matter were put in a salt mine under Lake Erie—so far underground to protect the matter from cosmic rays that would throw off the readings of "breathtakingly sensitive" detectors, according to panelist Robert Tschirhart, a senior scientist at Fermilab. But in 1987, a supernova went off in the Milky Way, and the experiment detected a neutrino from that star. "By sheer chance, an experiment that was designed to protect against cosmic rays serendipitously detected the oscillation of a neutrino from a distant star, which allowed us to discover that neutrinos have mass," Tschirhart said.

And a few years ago, a cardiologist was using beta-blockers to treat an infant for arrhythmia. The child happened to have a hemangioma, a benign tumor that can cause serious problems when it develops on the larynx or eye. When the physician noticed that the tumor disappeared after beta-blockers were administered, he reported his findings. "As a result, the preferred treatment for hemangioma around the world went from toxic steroids to much safer beta blockers within only about a year," said panelist Jessica Kandel, professor of surgery and chief of pediatric surgery at the Comer Children's Hospital. "Now, that's serendipity!"

Science is also full of stories where serendipity did not happen. The first scientists to detect buckminsterfullerene through mass spectroscopy did not notice the distinctive carbon-60 molecule, said Donald Levy, vice president for Research and for National Laboratories at the University. "Buckyballs (as they are called) popped up, and the researchers missed it." They "really blew it," Levy added, noting that

three Nobel Prizes have been awarded related to buckminsterfullerene, and that buckyballs later proved critical to the development of nanotechnology.

Stevens said the common features of serendipity include working in collaboration with others rather than in isolation, not being at risk of immediately losing your funding, and being confident and fearless of failure.

So how surprising are serendipitous discoveries? It turns out not very, if you are looking for connections made by scientists reaching out to local networks, said panelist James Evans, director of the Knowledge Lab and professor of sociology at the University. Using probability models, Evans found that these types of local connections are predictable as well as much less risky than reaching out across other areas of science.

"If you only have a budget for 10 experiments, but the really field-defining experiments end up being these distant experiments, it's very difficult for an individual scientist to underwrite that risk," said Evans. The greater the risk, he suggested, the more valuable institutions like national labs and industry labs become.

Of course, scientists have to explain and justify their work ahead of time in order to secure funding, but many great discoveries have been made outside of the explicit goals and parameters of the prescribed research. "Being too restrictive is the enemy of new knowledge," Kandel said.

http://phys.org/news/2014-06-electrocatalytic-boron-nitride-thin-oxygen.html

Researchers demonstrate electrocatalytic activity of boron nitride thin films for oxygen reduction reaction

A Japanese research team has successfully demonstrated a new approach toward a non-precious metal oxygen reduction catalyst for fuel cells.

The research was conducted by a group of researchers led by Professor Kohei Uosaki, a NIMS Fellow at the International Center for Materials Nanoarchitectonics and the Global Research Center for Environment and Energy based on Nanomaterials Science (both of which are affiliated with the National Institute for Materials Science) in partnership with another research group led by Professor Tetsuya Taketsugu at the Faculty of Science, Hokkaido University. The team advocated and successfully demonstrated a theory that when boron nitride (BN), which is originally an insulating material, is placed on a gold surface, it can function as an electrocatalyst for the oxygen reduction reaction—an important reaction in fuel cells.

Hydrogen-oxygen fuel cells are the ultimate clean power-generating device, which extract electric power from hydrogen and oxygen with high efficiency and emit only water as waste. However, there are still many challenges to overcome before achieving wide use of this technology. One such challenge is the slow rate of the oxygen-reduction reaction at the oxygen electrode—that is, low reaction efficiency. Platinum has been widely used as a catalyst for promoting this reaction. However, as platinum is expensive, limited in quantity and involves stability problems, efforts have been made worldwide toward the development of a new catalyst that can solve this challenge without using platinum or other precious metals. Yet, none of the catalysts that have been developed thus far has reached a satisfactory level. Accordingly, there has been a call for initiatives to develop a completely new type of catalyst, using materials that have never been thought of as catalyst materials.

The two research groups worked together on the development of a precious metal-free catalyst through a combination of theoretical and experimental approaches from the perspective of elements strategy. In their theoretical research, the researchers discovered that when BN, which is originally an insulating material, is placed on a gold surface, its electronic state changes, it becomes conductive, and oxygen molecules are absorbed on BN stably. They further calculated changes in energy at different stages in the oxygen-reduction reaction process on the surface and found that BN has the potential to function as an oxygen-reduction catalyst. Then, they prepared various types of BN (e.g. nanosheets, nanotubes) placed on a gold surface, and examined the activity for the oxygen-reduction reaction by a rotating disk electrode. They observed a maximum of about 270 mV positive shift for oxygen reduction current to be observed at the gold electrode. On the other hand, no such catalyst activity was observed when a carbon was used as the substrate. Thus, they demonstrated that BN-gold interaction is a key factor for BN to function as an electrocatalyst for the oxygen reduction reaction.

Although the new catalyst is still less reactive than platinum, the researchers succeeded in showing an extremely promising direction in the process of searching for and designing a new catalyst material, through the combination of theoretical calculation and experiments. This approach is expected to lead to the future development of materials for an electrode for fuel cells without using platinum.

This research was conducted as part of the "Elements Strategy Initiative for Industry-Academia-Government Collaboration: Creation of Precious Metal-Free Nano-Hybrid Catalyst" and the "Environmental Technology Development Program with the Use of Nanotechnology," sponsored by the Japanese Ministry of Education, Culture, Sports, Science and Technology. The research results were published in the online version of the Journal of the American Chemical Society (DOI: 10.1021/ja500393g), on April 28, 2014 and detailed results of the experiment that used various types of BN were published in the online version of a UK journal,

Physical Chemistry Chemical Physics (DOI: 10.1039/C4CP00402G), on May 6, 2014.

More information: Ganesan Elumalai, Hidenori Noguchi, Kohei Uosaki. "Electrocatalytic activity of various types of h-BN for the oxygen reduction reaction," (Physical Chemistry Chemical Physics). Tuesday May 6th 2014. DOI: 10.1039/C4CP00402G

http://phys.org/news/2014-06-frogs-vivid-colour-ward-predators.html

Frogs with vivid colour markings to ward off predators can also appear invisible

Frogs that rely on their vivid colour markings to ward off predators can also appear invisible, Deakin University scientists have discovered.

Phys.org - Researchers from Deakin University's Centre for Integrated Ecology found the anomaly among a species of frog in which some individuals use their rainbow hues to warn they are poisonous or toxic while others rely on their colourings to make them difficult to detect.

The vibrant blue and yellow poison dart frog (Dendrobates tinctorius). **Bibiana Rojas**

Centre for Integrated Ecology evolutionary biologist Professor John Endler and his Ph.D. student and co-author studied poison dart frogs in the wild and identified the reason behind the paradoxical observation that the amphibian's colourful markings varied between each animal.

Studying the vibrant blue and yellow poison dart frogs (Dendrobates tinctorius) in their natural habitat in French Guiana, Deakin researcher Bibiana Rojas found that one group of frogs exhibited bold or elongated patterns, the other demonstrated mottled and more variable markings.

The paradox was that the more variable the frog's markings, the less likely were predators to learn to recognise the danger. But if different colour patterns accompanied different behaviour, then pattern variation can be an advantage instead of a disadvantage.

"We found that some frogs move frequently in the same direction while others moved randomly, and that this movement behaviour variation corresponded to the colour patterns," Professor Endler said.

"The frogs with elongated patterns moved continuously in the same direction to create an illusion of static pattern, or a pattern travelling at a slower speed, to thwart predators attempting to track their trajectory.

"But frogs which moved randomly and changed directions frequently, rely on interrupted colour patterns that appear visually disruptive and hard to see at a distance, giving them an advantage in predator detection rather than tracking.

"These findings are quite exciting as we believe they might have application in the human world for defence camouflage," Professor Endler said.

"Alternatively, taxis, police cars, and ambulances could be painted with horizontal stripes to make them more easily seen when they are going past."

Professor Endler has been studying aposematic animals for many years - species that use colour in the wild to either attract a mate or ward off predation.

In Australia, Blue-ringed octopus and redback spiders, and the introduced Monarch Butterfly are among a variety of animal species instantly recognised for their chromatic colours that warn of their danger to would-be predators.

Black, red, yellow and white are among the bold colour schemes employed in nature to warn of danger.

The researcher's report, "Paradox lost: variable colour-pattern geometry is associated with differences in movement of aposematic frogs", has been published in the latest copy of the prestigious Royal Society Journal Biology Letters.

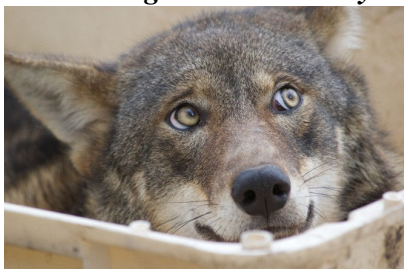
More information: Bibiana Rojas, Jennifer Devillechabrolle, and John A. Endler. "Paradox lost: variable colour-pattern geometry is associated with differences in movement in aposematic frogs." Biol. Lett.. 2014 10 6 20140193; DOI: 10.1098/rsbl.2014.0193 (published 18 June 2014) 1744-957X

http://www.wired.com/2014/06/wolves-might-use-their-eyes-to-talk-to-each-other/

Wolves Might Use Their Eyes to Talk to Each Other

Research hints that canids could be sending each other signals with their eyes

- **By Nick Stockton**

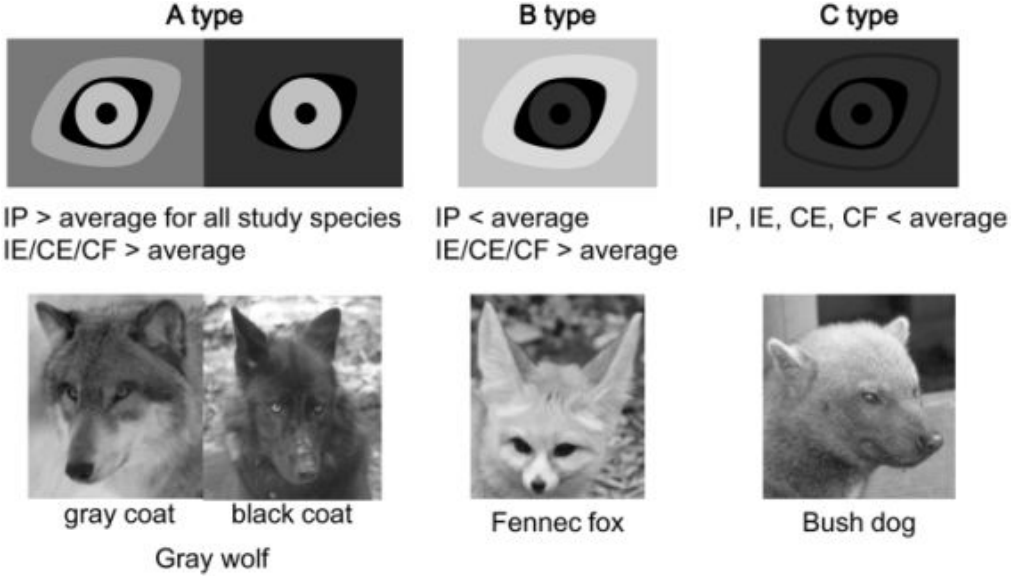


He's trying to say he wubs you. **US Fish and Wildlife Service**

It's no secret that wolves, foxes, and dogs are highly social animals. But beyond all the wagging, pawing and yipping, we like to try to interpret, canids may have yet another way to communicate. New research hints at the possibility that dogs and their ilk could be sending each other signals with their eyes.

A team of Japanese researchers looked at pictures of nearly every canid species and found that those with highly social pack and hunting behaviors were more likely to have easily-visible eyes. They then watched some of those species interact in zoos and concluded that those with eyes that were easier to see were more likely to be social. The results were published in a study in PLoS One on June 11.

"What this study shows is that there's a correlation between facial markings and sociality and the need to communicate," said zoologist Patricia McConnell of the University of Wisconsin-Madison, a dog behavior researcher who was not involved in the study.



PLoS

The scientists organized 25 different wild canid species according to their facial features (using around a dozen photos of individuals from each species) into three groups and then looked to previous research to characterize the social behavior of each group.

Group A contained species that have irises much lighter than their pupils, and faces with markings that make their eyes easy to locate. These animals, which include species like the gray wolf, coyote, and golden jackal, are more likely to live in social groups and hunt as part of a pack.

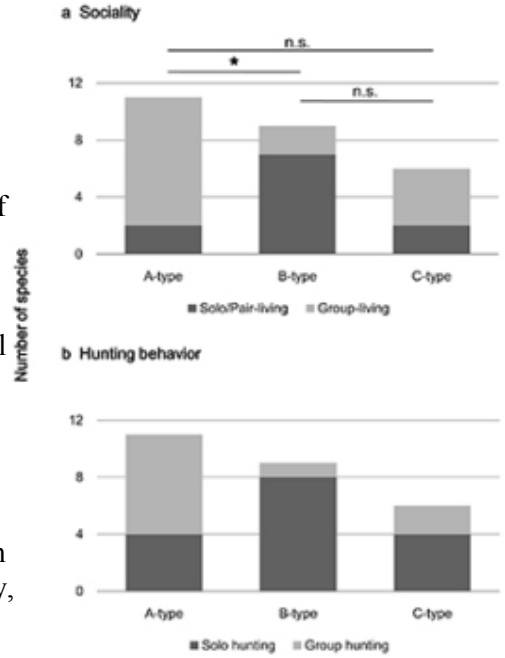
In group B were species where only the facial markings indicated the position of the eyes and the pupils weren't visible, such as the maned wolf, the dingo and the kit fox. These canids tend towards solo life or bonded pairs, and hunt alone.

The canid's eyes in group C were camouflaged, with no markings either within or around the eye to mark it from the rest of the face. Group C were mostly the more primitive canid species, like bush dogs, tanukis, and African wild dogs, which tend to live in social packs, but mostly hunt alone.

To field test their groupings, the researchers went to Japanese zoos and observed gazing behavior in a species from each of the groups: gray wolves (group A), fennec foxes (group B), and bush dogs (group C). All three species gazed at each other about the same number of times, but the wolves held their gazes significantly longer than the foxes or bush dogs. The gray wolves also did twice as many distinct playful postures as the other two species.

Gaze communication isn't unheard of in the animal kingdom, and humans are prime examples. Scientists believe one of the reasons we have white surrounding our irises is so we can pick up on what other people are looking at. If certain canids communicate with gaze, they probably rely mostly on having a high contrast between the iris and the pupil, rather than the sclera (the technical name for the white of the eye).

McConnell says that canids are big communicators, but we still have a long way to go before we understand all the channels they use. "You can say without a question that a canid's entire body is an information source," she said. This study, while it does show a link between gaze and sociability, isn't conclusive.



These graphs show that canids with highly visible eyes tended to be more social, and hunted in groups. **PLoS**

One way to take these tests further would be to camouflage the eyes of heavy-gazing canids and watch how it affects their social interactions. And maybe you could be the one to do it. McConnell says her field is suffering from a lack of researchers.