

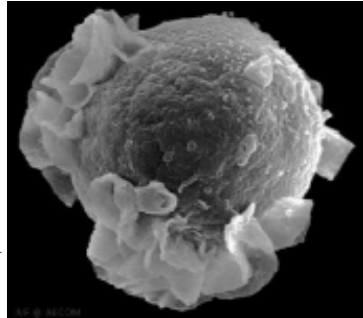
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Study reveals how a cancer-causing virus blocks human immune response

Cancer-causing virus outwits the human body's immune response

Scientists at The University of Texas at Austin and the University of California at San Francisco have revealed how a type of cancer-causing virus outwits the human body's immune response. The discovery might help explain why some cancer therapies fail to treat certain cancers and might lead to more effective treatments.

Epstein Barr Virus (EBV), a virus of the herpes family, causes an estimated 200,000 cancers every year, including lymphomas, nasopharyngeal cancers and some stomach cancers. Better anti-viral drugs could help thousands of people suffering from these cancers.



An Epstein-Barr virus erupts from an infected immune cell, called a B lymphocyte.

Analytical Imaging Facility at the Albert Einstein College of Medicine

Many viruses, including EBV, carry small molecules called microRNAs that they use to hijack natural processes in a host's cells during an infection. Viral microRNAs are known to prevent host cell death, promote host cell growth and dampen the host cell's viral defenses. However, scientists don't yet know which viral microRNAs perform which functions.

Jennifer Cox, a graduate student working with Associate Professor Chris Sullivan at UT Austin, identified microRNAs made by several herpes viruses that block a component of a human's innate immune system called the interferon response. Immune cells within the body release interferon to prevent viral replication, and this often results in slower growth or death of infected host cells. The researchers found that several herpes viruses have independently evolved similar mechanisms to block the host's interferon response.

"I was actually surprised that all these different viruses had converged on the same mechanism for blocking the body's defenses," said Sullivan. "As a biologist, this is evolutionary gold."

Interferon is sometimes used in combination with chemotherapy to treat lymphomas. While an effective treatment for some cancers, it does not significantly affect others. This latest research has demonstrated that EBV lymphoma cells are less susceptible to interferon therapy.

"This could explain the variability seen in the success of previous interferon-based cancer treatments," said Cox. "While this work does not immediately identify new

drugs, the fact that such different tumor viruses have converged on the same strategy makes this an exciting pursuit for future therapies against viral cancers." This work appears online in the January 26 edition of the *Proceedings of the National Academy of Sciences*. In addition to Cox and Sullivan, co-authors include Lydia McClure at The University of Texas at Austin and Andrei Goga from the University of California at San Francisco.

This research was funded by the National Institutes of Health and the Cancer Prevention and Research Institute of Texas.

http://www.eurekalert.org/pub_releases/2015-01/ghri-hdr012215.php

Higher dementia risk linked to more use of common drugs Link persists in University of Washington/Group Health study in JAMA Internal Medicine

SEATTLE - A large study links a significantly increased risk for developing dementia, including Alzheimer's disease, to taking commonly used medications with anticholinergic effects at higher doses or for a longer time. Many older people take these medications, which include nonprescription diphenhydramine (Benadryl). JAMA Internal Medicine published the report, called "Cumulative Use of Strong Anticholinergic Medications and Incident Dementia." The study used more rigorous methods, longer follow-up (more than seven years), and better assessment of medication use via pharmacy records (including substantial nonprescription use) to confirm this previously reported link. It is the first study to show a dose response: linking more risk for developing dementia to higher use of anticholinergic medications. And it is also the first to suggest that dementia risk linked to anticholinergic medications may persist - and may not be reversible even years after people stop taking these drugs.

"Older adults should be aware that many medications - including some available without a prescription, such as over-the-counter sleep aids - have strong anticholinergic effects," said Shelly Gray, PharmD, MS, the first author of the report, which tracks nearly 3,500 Group Health seniors participating in the long-running Adult Changes in Thought (ACT), a joint Group Health-University of Washington (UW) study funded by the National Institute on Aging. "And they should tell their health care providers about all their over-the-counter use," she added.

"But of course, no one should stop taking any therapy without consulting their health care provider," said Dr. Gray, who is a professor, the vice chair of curriculum and instruction, and director of the geriatric pharmacy program at the UW School of Pharmacy. "Health care providers should regularly review their older patients' drug regimens - including over-the-counter medications - to look for chances to use fewer anticholinergic medications at lower doses."

For instance, the most commonly used medications in the study were tricyclic antidepressants like doxepin (Sinequan), first-generation antihistamines like chlorpheniramine (Chlor-Trimeton), and antimuscarinics for bladder control like oxybutynin (Ditropan). The study estimated that people taking at least 10 mg/day of doxepin, 4 mg/day of chlorpheniramine, or 5 mg/day of oxybutynin for more than three years would be at greater risk for developing dementia. Dr. Gray said substitutes are available for the first two: a selective serotonin re-uptake inhibitor (SSRI) like citalopram (Celexa) or fluoxetine (Prozac) for depression and a second-generation antihistamine like loratadine (Claritin) for allergies. It's harder to find alternative medications for urinary incontinence, but some behavioral changes can reduce this problem.

"If providers need to prescribe a medication with anticholinergic effects because it is the best therapy for their patient," Dr. Gray said, "they should use the lowest effective dose, monitor the therapy regularly to ensure it's working, and stop the therapy if it's ineffective." Anticholinergic effects happen because some medications block the neurotransmitter called acetylcholine in the brain and body, she explained. That can cause many side effects, including drowsiness, sore throat, retaining urine, and dry mouth and eyes.

"With detailed information on thousands of patients for many years, the ACT study is a living laboratory for exploring risk factors for conditions like dementia," said Dr. Gray's coauthor Eric B. Larson, MD, MPH. "This latest study is a prime example of that work and has important implications for people taking medications - and for those prescribing medications for older patients." Dr. Larson is the ACT principal investigator, vice president for research at Group Health, and executive director of Group Health Research Institute (GHRI). He is also a clinical professor of medicine at the UW School of Medicine and of health services at the UW School of Public Health.

Some ACT participants agree to have their brains autopsied after they die. That will make it possible to follow up this research by examining whether participants who took anticholinergic medications have more Alzheimer's-related pathology in their brains compared to nonusers.

Drs. Gray and Larson's coauthors are Paul Crane, MD, MPH, an associate professor of medicine at the UW School of Medicine, adjunct associate professor of health services at the UW School of Public Health, and affiliate investigator at GHRI; Sascha Dublin, MD, PhD, a Group Health physician, GHRI associate investigator, and affiliate associate professor of epidemiology at the UW School of Public Health; Melissa L. Anderson, MS, and Onchee Yu, MS, senior biostatisticians, and Rod Walker, MS, biostatistician, at GHRI; Joseph T. Hanlon, PharmD, MS, a professor of medicine at the University of Pittsburgh; and Rebecca Hubbard, PhD, an associate Professor of Biostatistics at the Hospital of the University of Pennsylvania, who did this work while on staff at GHRI.

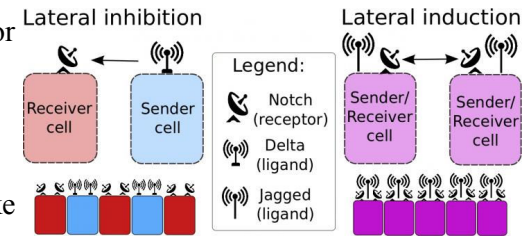
This work was supported by National Institute on Aging NIH Grants U01AG00678 (Dr. Larson), R01AG 027017, R01AG037451, P30AG024827, T32 AG021885, K07AG033174 (Dr. Hanlon), and R03AG042930 (Dr. Dublin) and by the Branta Foundation (Dr. Dublin).

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How cancer turns good cells to the dark side Rice researchers find 'jagged' proteins key as tumors hijack cell-signaling process

Cancer uses a little-understood element of cell signaling to hijack the communication process and spread, according to Rice University researchers.

A new computational study by researchers at the Rice-based Center for Theoretical Biological Physics shows how cancer cells take advantage of the system by which cells communicate with their neighbors as they pass messages to "be like me" or "be not like me."



Cancer cells have the ability to hijack the notch-signaling mechanism that cells use to communicate with each other, especially when "jagged" ligands allow for two-way signaling Marcelo Boareto/Rice University

Led by Rice biophysicists Eshel Ben-Jacob and José Onuchic, the researchers decode how cancer uses a cell-cell interaction mechanism known as notch signaling to promote metastasis. This mechanism plays a crucial role in embryonic development and wound healing and is activated when a delta or jagged ligand of one cell interacts with the notch receptor on an adjacent one. Their open-access study appears this month in the Proceedings of the National Academy of Sciences. It follows a 2014 study in which the researchers mapped the flow of information through genetic circuits involved in cancer metastasis. "At the heart of our new understanding is that the primary agents of metastasis are clusters of hybrid epithelial (nonmobile) and mesenchymal (migrating) cells," Ben-Jacob said. "These, and not the fully mesenchymal cells, are the 'bad actors' of cancer progression that pose the highest risk. By acting together, these hybrid cancer cells have a better chance to evade the immune system during migration and can better survive while circulating in blood vessels." The multifaceted mechanism by which notch-delta-jagged signaling promotes cancer progression has been a mystery until now, Ben-Jacob said, but recent experimental studies have revealed the jagged ligand plays a critical role in tumor progression. The new study provides a fresh theoretical framework for scientists who study the fates of cells. It shows the presence of jagged ligands can give rise to

sender/receiver hybrid cells that send a signal - "be like me" - that is useful for embryonic development and healing, but can also be hijacked by cancer cells. "We realized that hybrid cancer cells can take advantage of that characteristic to establish stable interactions and turn them into 'assault teams' that migrate together during metastasis," Onuchic said.

The focus of research on notch signaling to date has been on notch-delta signaling alone, Ben-Jacob said. In that case, one cell (the sender) expresses high notch receptor and low delta ligand. The other (the receiver) expresses low notch and high delta. This situation leads the two cells to adopt opposite fates: to "be not like me."

The first clues biologists had to notch-delta signaling came a century ago in studies of the wing formation of fruit flies. A visual manifestation of cell messaging is in the checkerboard or salt-and-pepper patterns seen in some organisms when cells tell their neighbors to be "not me" and adopt the opposite color. "Since jagged seemed to play a similar role to delta, the focus has been on notch-delta," Ben-Jacob said. "We were motivated to look closer and focus on the effect of the differences between these ligands."

"Cancer takes advantage of jagged proteins' influence to form what are essentially migrating units of hybrid cancer stem cells," Ben-Jacob said. Notch-jagged signaling also helps cells develop resistance to chemotherapy and radiotherapy and facilitates metastasis formation by promoting communications between cancer and stromal (connective tissue) cells at the new locations, he said. Recent findings showed stromal cells in the tumor environment secrete jagged ligands. The Rice researchers found cancer cells hijack nearby stromal cells and prompt them to boost their production of the ligand, reinforcing the cancer's chances of survival.

The researchers suggested cells' internal expression of jagged may also increase the production and maintenance of therapy-resistant cancer stem cells.

"Because they have a high likelihood to acquire stem-like properties, when arriving at distant organs they utilize this cellular plasticity to differentiate and adapt to new conditions at the metastasis location," said lead author Marcelo Boareto, a former visiting scholar at Rice and now a doctoral student at the University of Sao Paulo, Brazil.

The researchers said their model is a step toward deeper understanding of the signaling mechanisms cancer cells use to evade the immune system and treatment. "Studying single cells cannot give us all the answers," Onuchic said. "We need to understand the decisions made by the cells that are talking to each other."

The paper's co-authors include postdoctoral researcher Mingyang Lu, graduate student Mohit Kumar Jolly and Cecilia Clementi, a professor of chemistry and of chemical and

biomolecular engineering, all at Rice. Onuchic is the Harry C. and Olga K. Wiess Chair of Physics and professor of physics and astronomy, of chemistry and biosciences and co-director of the Center for Theoretical Biological Physics. Ben-Jacob is an adjunct professor of biosciences at Rice, a senior investigator at the Center for Theoretical Biological Physics and a professor of physics and the Maguy-Glass Chair in Physics of Complex Systems at Tel Aviv University. <http://www.pnas.org/content/early/2015/01/15/1416287112.full.pdf+html>

http://www.eurekalert.org/pub_releases/2015-01/uoc-ufc012315.php

UCI, fellow chemists find a way to unboil eggs

Ability to quickly restore molecular proteins could slash biotechnology costs

Irvine, Calif. - UC Irvine and Australian chemists have figured out how to unboil egg whites - an innovation that could dramatically reduce costs for cancer treatments, food production and other segments of the \$160 billion global biotechnology industry, according to findings published today in the journal ChemBioChem.

"Yes, we have invented a way to unboil a hen egg," said Gregory Weiss, UCI professor of chemistry and molecular biology & biochemistry. "In our paper, we describe a device for pulling apart tangled proteins and allowing them to refold. We start with egg whites boiled for 20 minutes at 90 degrees Celsius and return a key protein in the egg to working order."

Like many researchers, he has struggled to efficiently produce or recycle valuable molecular proteins that have a wide range of applications but which frequently "misfold" into structurally incorrect shapes when they are formed, rendering them useless. "It's not so much that we're interested in processing the eggs; that's just demonstrating how powerful this process is," Weiss said. "The real problem is there are lots of cases of gummy proteins that you spend way too much time scraping off your test tubes, and you want some means of recovering that material."

But older methods are expensive and time-consuming: The equivalent of dialysis at the molecular level must be done for about four days. "The new process takes minutes," Weiss noted. "It speeds things up by a factor of thousands."

To re-create a clear protein known as lysozyme once an egg has been boiled, he and his colleagues add a urea substance that chews away at the whites, liquefying the solid material. That's half the process; at the molecular level, protein bits are still balled up into unusable masses. The scientists then employ a vortex fluid device, a high-powered machine designed by Professor Colin Raston's laboratory at South Australia's Flinders University. Shear stress within thin, microfluidic films is applied to those tiny pieces, forcing them back into untangled, proper form. "This method ... could transform industrial and research production of proteins," the researchers write in ChemBioChem.

For example, pharmaceutical companies currently create cancer antibodies in expensive hamster ovary cells that do not often misfold proteins. The ability to quickly and cheaply re-form common proteins from yeast or E. coli bacteria could potentially streamline protein manufacturing and make cancer treatments more affordable. Industrial cheese makers, farmers and others who use recombinant proteins could also achieve more bang for their buck.

UCI has filed for a patent on the work, and its Office of Technology Alliances is working with interested commercial partners.

Besides Weiss and Raston, the paper's authors are Tom Yuan, Joshua Smith, Stephan Kudlacek, Mariam Iftikhar, Tivoli Olsen, William Brown, Kaitlin Pugliese and Sameeran Kunche of UCI, as well as Callum Ormonde of the University of Western Australia. The research was supported by the National Institute of General Medical Sciences (grant R01 GM100700-01) and the Australian Research Council (grants DP1092810 and DP130100066).

<http://bit.ly/1KdZMK3>

Ancient Assyrian Soldiers Were Haunted by War, Too

A new study finds evidence of trauma experienced by soldiers returning home from combat over 3,000 years ago

By Laura Clark smithsonian.com

In his account of battle of Marathon in 490 B.C., the Greek historian Herodotus recorded the story of a man that went inexplicably blind after witnessing the death of one of his comrades. Until recently, this was believed to be earliest-known record of what modern medicine calls post-traumatic stress disorder.

But now, as BBC News reports, a team of researchers says they've found references to PTSD-related symptoms in much earlier writings, dating from the Assyrian Dynasty in Mesopotamia, between 1300 B.C. and 609 B.C. They published their findings in the journal *Early Science and Medicine* with an article poetically titled "Nothing New Under the Sun."



A stone relief carving of soldiers made in Assyria and now in the British Museum. (Gianni Dagli Orti/Corbis)

Soldiers in ancient Assyria (located in present-day Iraq) were tied to a grueling three-year cycle, the BBC notes. They typically spent one year being "toughened up by building roads, bridges and other projects, before spending a year at war and then returning to their families for a year before starting the cycle again."

By studying translations of known texts, the historians were able to see just how familiar symptoms of PTSD might have been to Assyrian soldiers. Co-author of the study and director of the Anglia Ruskin University's Veterans and Families Institute, Professor Jamie Hacker Hughs told BBC News:

"The sorts of symptoms after battle were very clearly what we would call now post-traumatic stress symptoms.

"They described hearing and seeing ghosts talking to them, who would be the ghosts of people they'd killed in battle - and that's exactly the experience of modern-day soldiers who've been involved in close hand-to-hand combat."

As the study's abstract states, the researchers also found instances of soldiers reporting "flashbacks, sleep disturbance and low mood."

PTSD wasn't clinically recognized in the U.S. until 1980, following a surge in classifiable cases from soldiers returning home from the Vietnam War. Before that, terms like "shell shock" were used to describe post-combat psychological struggles, and many soldiers, either because of external pressures or their own feelings of shame, kept quiet about emotional injuries first sustained in war. This new research helps to demonstrate that, despite only recently receiving wide recognition, the correlation between war and post-traumatic stress is likely as old as human civilization.

<http://bit.ly/1ye82Uu>

Tape of life may not always be random

Evolution may have fewer options for adapting to new challenges than you'd think.

16:37 26 January 2015 by Bob Holmes

When terrestrial mammals returned to the ocean to become whales, walrus and manatees, the three lineages sometimes made use of strikingly similar genetic changes.

Evolutionary biologists have long debated whether rewinding the tape of life and replaying it would give similar results, or whether outcomes depend largely on chance events that push the course of evolution onto radically different tracks. The two alternatives yield very different views of the history of life on Earth, with some prominent biologists, such as Simon Conway Morris, arguing that human-like, intelligent beings are inevitable products of evolution. Others, such as palaeontologist Stephen Jay Gould, who popularised the tape of life metaphor, argue that if it were possible to turn back the clock, the history of life would not repeat itself. The world would be unfamiliar, and most likely lack humans. To test the reproducibility of evolution at the genetic level, an international team took advantage of a natural experiment. Three different groups of terrestrial mammals have at some point in their evolution re-colonised the ocean, giving rise

to what we now know as whales, walruses and manatees. Comparing the genetic changes in the three lineages, the researchers reasoned, should reveal whether evolution followed similar or very different paths in each case.

Random idea

They sequenced the genomes of walrus, manatee and two whales – killer whales and bottlenose dolphins. The comparisons showed that many genes changed independently in each lineage, suggesting that randomness did indeed play an important role in their evolution.

But for 15 genes, natural selection led to exactly the same genetic changes occurring in all three lineages. This suggests that for some of the challenges of life in the sea, evolution repeatedly arrived at the same solution – that is, replaying the tape does indeed give much the same result again and again. This is a high-resolution replay of the tape, looking at what would happen to individual lineages, rather than what overall diversity would eventually result, which is what Gould looked at.

The team has not yet shown directly that any of these convergent genetic changes is actually adaptive, though some they found – affecting, for example, the structure of ear bones or metabolism related to deep diving – could plausibly be so. However, this result may say less about the predictable creativity of evolution than about a paucity of viable options. When the team performed a similar analysis of the genomes of dog, elephant and cow – related mammals that remained on land – they also found a comparable amount of convergence in their mutations, even though those animals share few similarities of lifestyle.

Lack of options

This may imply that the vast majority of mutations are lethal, so that evolution stumbles on the same few viable ones over and over again. "We think it's because there's only so much you can change and still be functional," says Kim Worley, a genome biologist at Baylor College of Medicine in Houston, Texas.

"If you replayed the tape, you'd probably see the same changes again amongst the marine mammals, but if you took a walrus and a camel, you'd still see the same changes, because of these constraints," says Andrew Foote, an evolutionary biologist at the University of Copenhagen.

But David Wake, an evolutionary biologist at the University of California at Berkeley, cautions that the study was essentially a genome-wide fishing expedition to look for interesting patterns. Much more detailed follow-up work will be needed to show whether the team's hypothesis holds up.

"I find it intriguing, but I think the evidentiary basis for it is still pretty weak," says Wake. "But we're just starting out."

Journal reference: *Nature Genetics*, DOI: 10.1038/ng.3198

<http://bit.ly/1wI70P0>

Australian Stories Capture 10,000-Year-Old Climate History

Aboriginal groups from coast to coast describe walking to places that are now islands

By Marissa Fessenden smithsonian.com

Three islands lie just off the coast near Perth, Australia. All are popular tourist destinations: [Rottne Island](#) is famous for its population of quokkas, a small marsupial. The tiny [Carnac Island](#) has sea lions and deadly tiger snakes. Slender [Garden Island](#) is home to a naval base.

All three of these islands were originally inhabited by Aboriginal people, though. And, [according to Climate Central](#), an early European settler described some stories told by the Aboriginal people of a time when the islands "once formed part of the mainland, and that the intervening ground was thickly covered with trees." But in one story, those trees caught fire and burned "with such intensity that the ground split asunder with a great noise, and the sea rushed in between, cutting off these islands from the mainland."

It may seem like a just a story, but researchers recently matched this and other Aboriginal stories to real events. The sea did rush in - at the end of the last glacial period - about 7,500 to 8,900 years ago.

Another community tells of a time when northeastern Australia's shoreline reached all the way out to the Great Barrier Reef. They recall a river that flowed into the sea at what is now Fitzroy Island. For Climate Central, John Upton writes, "The great gulf between today's shoreline and the reef suggests that the stories tell of a time when seas were more than 200 feet lower than they are today, placing the story's roots at as many as 12,600 years ago."

"It's quite gobsmacking to think that a story could be told for 10,000 years," [Nicholas Reid](#), a linguist specializing in Aboriginal Australian languages at Australia's University of New England, told Upton. "It's almost unimaginable that people would transmit stories about things like islands that are currently underwater accurately across 400 generations."

The story did last because the telling of it was kept alive by rich tradition. Without a written language, Australian tribes relied on oral storytelling to keep [their identity](#) - it is part of the collection of knowledge, practices and faith referred to as [The Dreaming](#). The stories are [more than oral tellings](#). They include paintings on rock or bark, [drawings in sand](#), ceremonies, song and dance. "There are aspects of storytelling in Australia that involved kin-based responsibilities to tell the stories accurately," Reid said. That rigor provided "cross-generational scaffolding" that "can keep a story true."

Reid worked with a geography professor at the University of the Sunshine Coast, Patrick Nunn, to match the stories with the land and how it has changed. A [preliminary draft of their work](#), presented at an indigenous language conference in Japan, makes the case for 18 Aboriginal stories describing the coastal flooding of the end of the last ice age. The paper also argues that researchers who are building a picture of our world and its changes should look to old stories. "[E]ndangered Indigenous languages can be repositories for factual knowledge across time depths far greater than previously imagined, forcing a rethink of the ways in which such traditions have been dismissed," Nunn writes.

"There's a comparably old tradition among the Klamath of Oregon that must be at least 7,700 years old – it refers to the last eruption of Mount Mazama, which formed Crater Lake," Nunn told Climate Central. "I'm also working on ancient inundation stories and myths from India, and I've been trying to stimulate some interest among Asian scholars."

Marissa Fessenden is a freelance science writer and artist who appreciates small things and wide open spaces.

<http://bit.ly/1wI70P0>

Hold the Drug, Go Straight to the Source

Ground-up [artemisia](#) plants, from which the anti-malaria drug [artemisinin](#) is derived, appear to work much better than the refined drug does by itself, according to research at the University of Massachusetts.

Artemisinin, [discovered by Chinese scientists](#) in a project started by Mao Zedong to help the North Vietnamese, has become the newest malaria miracle cure. But parasites resistant to it [have emerged](#).

Scientists infected mice with two strains of rodent malaria - one that is already artemisinin-resistant and one that is not, but is biologically similar to *Plasmodium falciparum*, the deadliest strain of human malaria. They then fed the mice pure artemisinin or dried *artemisia annua* plants bred for high drug content at [Worcester Polytechnic Institute](#). The [study](#) was published this month by [the Proceedings of the National Academy of Sciences](#).

The whole plant cured mice with artemisinin-resistant malaria. In mice with the dangerous strain, parasites resistant to the plant failed to emerge even after 49 successive infections - three times as many as it took for parasites resistant to artemisinin alone to evolve.

"We don't know what the precise mechanism is," said Stephen M. Rich, a University of Massachusetts microbiologist and the paper's lead author, but the plant contains dozens of toxic chemicals that repel or kill fungi, bacteria, insects and even rival plants. Some may protect the artemisinin from being broken down by the liver. Also, he said, malaria parasites share an ancestor with plants and

contain vestigial versions of the chlorophyll-producing organelles. The natural herbicides some plants use to kill rivals may also work on them, he said.

<http://bit.ly/1tEoGOF>

Ancient planets are almost as old as the universe *The Old Ones were already ancient when the Earth was born.*

01:00 27 January 2015 by Lisa Grossman

Five small planets orbit an 11.2 billion-year-old star, making them about 80 per cent as old as the universe itself. That means our galaxy started building rocky planets earlier than we thought.

"Now that we know that these planets can be twice as old as Earth, this opens the possibility for the existence of ancient life in the galaxy," says Tiago Campante at the University of Birmingham in the UK. NASA's Kepler space telescope spotted the planets around an orange dwarf star called Kepler 444, which is 117 light years away and about 25 per cent smaller than the sun.

Orange dwarfs are considered good candidates for hosting alien life because they can stay stable for up to 30 billion years, compared to the sun's 10 billion years, the time it takes these stars to consume all their hydrogen. For context, the universe is currently 13.8 billion years old.

Metal light

Since, as far as we know, life begins by chance, older planets would have had more time to allow life to get going and evolve. But it was unclear whether planets around such an old star could be rocky – life would have a harder time on gassy planets without a solid surface.

The first stars to form in the universe were made of just hydrogen and helium, and forged heavier elements in their interior before exploding. The next generation of stars emerged from their debris, and incorporated those heavier elements into their cores and whatever planets they formed. This means that in general, older stars have fewer metals.

Until recently, planet-hunters assumed that stars needed metals to form planets, partly because the first planets they discovered all orbited metal-rich stars, and partly because planets themselves are made of heavier stuff than hydrogen and helium. But a 2012 survey of Kepler planets showed that low-metal stars could host relatively small planets.

"We knew beforehand that small planets could exist around stars of any metallicity, but it was not really well known if we could go down to Earth-sized planets," Campante says.

Kepler 444's planets are all smaller than Earth, ranging from 0.4 to 0.74 times Earth's radius. Kepler data suggests that planets tend to be rocky when they're smaller than 1.7 Earth radii, and gaseous when they're bigger, making the Kepler

444 worlds almost certainly rocky. But they orbit scorchingly close to the star: the furthest, Kepler-444f, orbits once every 9.7 days, and the closest, Kepler-444b, every 3.6 days. The length of their orbits are all multiples of each other, meaning they eclipse each other regularly and every so often line up all in a row.

Planets align

"You can imagine if you are standing on the surface of the outermost planet, at some points during the orbit you could look in the direction of the star and see all the other four planets aligned," Campante says. "It must be amazing."

To find out how old the star is, Campante and his colleagues used a technique called astroseismology to measure the age of the star very precisely. With the help of the Kepler telescope's entire four-year data set, the team watched Kepler 444's brightness change over time. These fluctuations reflect vibrations within the star, which tell you its mean density. Because a star converts hydrogen to helium in its core as it ages, changing its density, knowing a star's density tells you how old it is.

This technique gave Kepler 444 an age of 11.2 billion years, plus or minus 1 billion years. That makes it the oldest known system of terrestrial planets in the galaxy – when Earth formed, these planets were already older than our planet is today. (The previous record-holder, a red dwarf known as Kapteyn's star, hosts larger planets that are probably mini-Neptunes.)

"These planets mean it only took the universe a couple billion years to figure out how to build rocky planets, and they've been around for a really long time," says Travis Metcalfe at the Space Science Institute in Boulder, Colorado. While Kepler 444's planets are too hot for life, its age suggests there might be cooler, older worlds elsewhere. "If life needs a long time to develop or lots of places to try to develop, having rocky planets this early in the history of the galaxy means planets with advanced civilisations should be everywhere."

"These are all little bits of good news," says Andrew Howard at the University of Hawaii at Manoa. "There are still a lot of other hurdles life would have to overcome, but now we're seeing evidence that small planets are common, and here we have one from when the Milky Way was a kid and it was already forming probably rocky planets."

The next step is to figure out exactly what they're made of, he says. His team has been using the Keck telescope in Hawaii to try to get a handle on these planets' masses by measuring their gravitational tugs on the star. Knowing the planet's mass and radius gives its density, a clue to composition – but the masses are proving too small to measure.

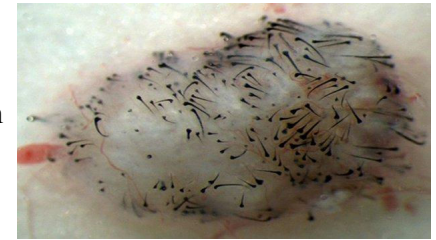
"That's not surprising or concerning, it just confirms that these are really small planets," he says. *Journal reference: arxiv.org/abs/1501.06227*

http://www.eurekalert.org/pub_releases/2015-01/smri-usc012715.php

Using stem cells to grow new hair

Researchers develop method to induce human hair growth using pluripotent stem cells

La Jolla, Calif. - In a new study from Sanford-Burnham Medical Research Institute (Sanford-Burnham), researchers have used human pluripotent stem cells to generate new hair. The study represents the first step toward the development of a cell-based treatment for people with hair loss. In the United States alone, more than 40 million men and 21 million women are affected by hair loss. The research was published online in PLOS One yesterday.



Scientists at Sanford-Burnham used iPSCs to grow new hair. Sanford-Burnham Medical Research Institute

"We have developed a method using human pluripotent stem cells to create new cells capable of initiating human hair growth. The method is a marked improvement over current methods that rely on transplanting existing hair follicles from one part of the head to another," said Alexey Terskikh, Ph.D., associate professor in the Development, Aging, and Regeneration Program at Sanford-Burnham. "Our stem cell method provides an unlimited source of cells from the patient for transplantation and isn't limited by the availability of existing hair follicles."

The research team developed a protocol that coaxed human pluripotent stem cells to become dermal papilla cells. They are a unique population of cells that regulate hair-follicle formation and growth cycle. Human dermal papilla cells on their own are not suitable for hair transplants because they cannot be obtained in necessary amounts and rapidly lose their ability to induce hair-follicle formation in culture. "In adults, dermal papilla cells cannot be readily amplified outside of the body and they quickly lose their hair-inducing properties," said Terskikh. "We developed a protocol to drive human pluripotent stem cells to differentiate into dermal papilla cells and confirmed their ability to induce hair growth when transplanted into mice."

"Our next step is to transplant human dermal papilla cells derived from human pluripotent stem cells back into human subjects," said Terskikh. "We are currently seeking partnerships to implement this final step."

*The study was performed in collaboration with the Laboratory of Cell Proliferation at the Koltsov Institute of Developmental Biology in Moscow, Russia
This study was supported by funds from Sanford-Burnham.*

http://www.eurekalert.org/pub_releases/2015-01/bidm-ar1012615.php

Analysis rejects linkage between testosterone therapy and cardiovascular risk

Article contrasts 4 flawed studies to dozens showing reduced mortality and other cardiovascular benefits with therapy or with high levels of testosterone

BOSTON - Fears of a link between testosterone replacement therapy and cardiovascular risk are misplaced, according to a review published in this month's Mayo Clinic Proceedings. The therapy has come under widespread scrutiny in recent months, including by a federal Food and Drug Administration (FDA) panel convened last fall.

"There's no good evidence that we could find that testosterone therapy increases cardiovascular risk," says lead author Abraham Morgentaler, MD, of Director of Men's Health Boston and a urologist on staff at Beth Israel Deaconess Medical Center. "That's not to say it's perfectly safe. But we cannot find evidence and the headlines that jumped out on recent retrospective studies appear to be too strong." Importantly, and under-recognized among physicians, Morgentaler adds, "review of the literature clearly reveals a strong relationship between higher serum testosterone concentrations ... as being beneficial for reduction in cardiovascular disease and cardiovascular risk factors."

Testosterone is a hormone that, during puberty, helps build a man's muscles, deepens his voice and increases the size of his reproductive organs. As adults, men rely on the hormone to keep muscles and bones strong and to maintain an interest in sex. Testosterone levels generally begin a gradual decline after the age of 30, a drop that may be accompanied by a decrease in sex drive. In recent years, the use of testosterone replacement therapy has increased substantially, aided in part by "patient-friendly formulations" such as topical gels that are widely advertised on television.

Such advertisements, combined with two recent studies raising questions about cardiovascular risk associated with the treatment, were the backdrop to an FDA advisory panel on testosterone therapy convened in September 2014. The panel voted 20-1 in favor of conducting a large-scale study to assess cardiovascular risk associated with testosterone therapy; the panel also voted in favor of a change in labeling requirements restricting the indications for use of testosterone.

"Testosterone has been presented as if there were a debate about whether it is good or evil," says Morgentaler. "Rather, it is a long-accepted medical treatment for a medical condition recognized for centuries. Our intention was to cut through the confusion of loudly expressed opinions on non-scientific issues - such as pharmaceutical advertising, anti-aging claims, and the importance of sexuality in

older men - to provide the most comprehensive review to date of the literature on testosterone and cardiovascular risk."

The article by Morgentaler and colleagues in the fields of urology, endocrinology, family medicine and steroid research identified only four published scientific journal articles since 1940 that suggest increased cardiovascular risks with testosterone prescriptions. Two of those four articles, which generated substantial media coverage over the last 15 months, had "serious methodological limitations; one placebo-controlled trial with few major adverse cardiac events and one meta-analysis that included questionable studies and [cardiovascular] events."

In contrast, Morgentaler and his co-authors cite dozens of studies examining the relationship between testosterone and heart-related issues, including coronary artery disease, ischemic stroke, cholesterol levels, angina and heart failure. They found that many of those studies identify a positive correlation between "low testosterone levels and increased mortality ... as well as atherosclerosis, incident coronary artery disease and the severity of coronary artery disease."

Two observational studies have shown that men with low testosterone levels who did not receive testosterone replacement therapy died at double the rate of similar men who did receive testosterone. A small number of randomized controlled studies have even shown that men with known heart disease (specifically angina or congestive heart failure) were able to function better when they received testosterone compared with a placebo. Numerous studies have shown that risk factors for cardiovascular disease - such as waist circumference, obesity, and fat mass - all improve with testosterone therapy.

Additional studies have shown benefits of testosterone therapy, including increased sex drive, energy and bone mineral density. The authors also describe "promising new data" that suggest testosterone therapy improves insulin sensitivity, reduces blood glucose and hemoglobin A1C levels in men with Type 2 diabetes or obesity. Yet public attention appears to have been focused on the four studies that "have undergone serious criticism in the scientific literature. The FDA itself has provided commentary on these studies, concluding that none provide compelling evidence of increased cardiovascular risk."

The testosterone story "has been turned upside-down," says Morgentaler, "by trumpeting studies providing remarkably weak evidence of risk, and ignoring a substantial literature with reassuring or beneficial results."

Morgentaler and his colleagues write "public health may be harmed not only by inadequate appreciation of an actual risk but also by the failure to offer beneficial treatment for a medical condition because of false claims of risk concerns."

In addition to Morgentaler, an Associate Clinical Professor of Urology at Harvard Medical School, authors include: Martin M. Miner, MD, Department of Family Medicine at Brown

University and on staff at Miriam Hospital Men's Health Center, Providence, RI; Monica Caliber, MSc, Fort Lauderdale, FL; Andre T. Guay, MD (deceased), Department of Endocrinology, Lahey Clinic, Burlington, MA; Mohit Khera, MD, Scott Department of Urology, Baylor College of Medicine, Houston TX; and Abdulmageed M. Traish, PhD, Department of Biochemistry and Department of Urology, Boston University School of Medicine, Boston, MA. Morgentaler has been on the scientific advisory board or worked as a consultant for AbbeVie, Inc., Auxilium Pharmaceuticals, Inc., Clarus Therapeutics, Endo Pharmaceuticals, and TesoRx, and has received research funding from Antares Pharma, Auxilium Pharmaceuticals, Inc., Lipocine Inc. and Eli Lilly and Company. Khera has worked as a consultant for Auxilium Pharmaceuticals, Inc. and Merck & Co. and has received research funding from Auxilium Pharmaceuticals, Inc. Miner has worked as a consultant for AbbeVie Inc. and Lipocine Inc., and has received research funding from Forest Laboratories, Inc. Guay has worked as a consultant for Endo Pharmaceuticals Inc., and Repros Pharmaceuticals, Inc. Beth Israel Deaconess Medical Center is a patient care, teaching and research affiliate of Harvard Medical School and consistently ranks as a national leader among independent hospitals in National Institutes of Health funding. BIDMC is in the community with Beth Israel Deaconess Hospital-Milton, Beth Israel Deaconess Hospital-Needham, Beth Israel Deaconess Hospital-Plymouth, Anna Jaques Hospital, Cambridge Health Alliance, Lawrence General Hospital, Signature Healthcare, Beth Israel Deaconess HealthCare, Community Care Alliance and Atrius Health. BIDMC is also clinically affiliated with the Joslin Diabetes Center and Hebrew Senior Life and is a research partner of Dana-Farber/Harvard Cancer Center and The Jackson Laboratory. BIDMC is the official hospital of the Boston Red Sox. For more information, visit <http://www.bidmc.org>

http://www.eurekalert.org/pub_releases/2015-01/uoc-nrb012615.php

Neuroscience researchers believe in quitting smoking gradually Brain's oxygen uptake and blood flow decreases by up to 17% immediately after people stop smoking

University of Copenhagen - The Faculty of Health and Medical Sciences
Smoking is harmful in almost every respect. Cancer, stroke, and other cardiovascular diseases are just a small part of a well-documented portfolio of serious consequences of smoking. Nicotine is what makes smoking addictive, but new Danish research suggests that smoking initially increases brain activity. However, the brain tissue quickly adapts and the effect will disappear. On the other hand, according to brain scans, the brain's oxygen uptake and blood flow decreases by up to 17% immediately after people stop smoking: Regular smokers experience an almost dementia-like condition in the early hours after quitting, as suggested by brain scans. This can be quite an unpleasant experience, and is probably one of the reasons why it can be very difficult to quit smoking once and for all. Smokers drift back into abuse, perhaps not to obtain a pleasant effect - that ship has sailed - but simply because the withdrawal symptoms are unbearable, says Professor Albert Gjedde, neuroscience researcher at the Department of Neuroscience and Pharmacology, University of Copenhagen.

Together with Associate Professor Manouchehr Seyedi Vafae from the same department and other scientists, Albert Gjedde is behind the new findings published in the Journal of Cerebral Blood Flow & Metabolism.

The researchers compare the nicotine in tobacco smoke with other pharmacologically active substances:

After a period of time, many users of medicine will no longer experience an effect from treatment - for example with antidepressants. However, the consequences of discontinuing treatment could still be overwhelming if the withdrawal symptoms are very unpleasant, says Albert Gjedde.

Habitual smokers seemingly need to continue smoking just to keep their brain functioning normally. With time, they may become less dependent on smoking, but the researchers still do not know how long it takes before the brain of a former smoker has regained its normal energy consumption and blood flow:

We assume that it takes weeks or months, but we do not know for sure. The new findings suggest that it may be a good idea to stop smoking gradually - simply to avoid the worst withdrawal symptoms that make it so difficult to stick to the otherwise very sensible decision to stop smoking, says Albert Gjedde. He emphasises that there are still many blind spots in relation to researching the brains of smokers.

<http://bit.ly/1Hxtyxf>

Without Friends or Family, even Extraordinary Experiences are Disappointing

Happiness is inherently social, two studies find

January 27, 2015 |By Daniel Yudkin

Imagine you are with some friends at a concert, and the bouncer approaches the group and says that, because you are all looking so ravishing tonight, he's been instructed to offer one of you - just one! - a backstage pass to meet the artist. Do you raise your hand? For most people, this would be a no-brainer: who wouldn't leap at the chance to meet a famous singer or secure a long-sought autograph? The results of a recent study, published in Psychological Science by Gus Cooney, Daniel Gilbert, and Timothy Wilson, however, suggest taking a second's pause before snapping up that backstage pass.

Cooney, Gilbert, and Wilson suspected that extraordinary experiences - like meeting a musical idol - carry hidden costs. They hypothesized that, while such occurrences undoubtedly make us happier in the moment, they also risk separating us from our peers, leading to a sense of isolation so unpleasant as to outweigh whatever enjoyment they initially confer.

To test this idea, the researchers recruited subjects in groups of four and had them watch a video clip. Of the group, three were told that they would watch a clip that

previous viewers had given a 2-star rating; the remaining subject, by contrast, was granted the opportunity to view a special 4-star clip. After watching the videos, all four subjects were given some time to talk amongst themselves, and then each reported on their general happiness.

Normally, we might expect the 4-star subject to feel the happiest. After all, this was the lucky individual who had seen the “extraordinary” video while the others - poor suckers - had had to suffer through a bad one. The reality, though, was just the opposite: those who’d seen the “better” clip actually felt worse than their peers. Why? The data suggested that these people - the “extraordinary experiencers” - had felt so excluded from the post-viewing conversation that any thrill they’d gotten from the video itself was utterly erased. This would be as if, while you went backstage to fawn over your favorite artist, your friends traipsed off to a bar and developed a hilarious inside joke.

This study suggests that the hedonic value we glean from experiences stems not so much from the immediate pleasure they bestow but from the subsequent joy we take in reliving them with others. For many of us, the stories we tell, like those in Springsteen’s “Glory Days,” accrue, through their retelling, added layers of richness unattainable if experienced alone.

At a broader level, the study also demonstrates the deep social contingency of our understanding of the world. Everything we do and see is interpreted through our interactions with others. This social embeddedness is so complete, in fact, that our company shapes not just our experiences after they have taken place, but also while they’re occurring - a point which is demonstrated vividly in a separate study published in the same issue of Psychological Science. This study, conducted by Erica Boothby, Margaret Clark, and John Bargh, examines the power of “shared experience,” showing that the mere feeling of togetherness is sufficient to amplify the perceived intensity of sensations like the flavor of chocolate.

In a cleverly designed experiment, the researchers asked that subjects sit at a table with a partner and rate two chocolate bars. Unbeknownst to them, the “partner” was actually a confederate - someone in cahoots with the scientists. Subjects tasted one of the chocolate bars simultaneously with the partner, the other while the partner was otherwise occupied. (Pains were taken to ensure that people couldn’t see each others’ responses.)

Which of the two chocolate bars was tastier? According to the subjects, one of the bars was significantly more flavorful than the other - and more enjoyable overall. Here’s the rub, though: the bars were identical. The only difference was that subjects had tasted one of the bars - the more “flavorful” one - at the same time as their partner.

This study demonstrates the power of togetherness to change basic qualities of experiences. Note this is not because company makes all experiences better - but rather because it makes them more extreme: in a subsequent experiment, the researchers showed that co-experience renders bitter flavors worse. The sense of being together seems thus to heighten both the pleasure of the positive and the nastiness of the negative.

Like bits of matter floating in space, humans cluster into communities. These communities serve several purposes: they offer protection and security, they provide resources both physical and emotional, and they give a sense of meaning and belonging. They also hold an arguably even greater power: to actively influence the way we interpret the world. The most dazzling firework can seem muted if viewed alone; the most unremarkable vista inspiring with good friends. Being with others adds a Technicolor tinge to the drab mundanity of daily life. It would seem, then, that the best way to go about choosing your next concert should be to focus not on the fame of the headliner but on the quality of the company.

Daniel Yudkin is a doctoral candidate in social psychology at New York University and jazz pianist. He graduated from Williams College, was a Fellow at Harvard University, and currently lives in Brooklyn. You can follow him @dyudkin and learn more at his website.

<http://nyti.ms/1uMbFQf>

Myriad Genetics Ending Patent Dispute on Breast Cancer Risk Testing

Myriad Genetics has essentially given up trying to stop other companies from offering tests for increased risk of breast cancer

By ANDREW POLLACK JAN. 27, 2015

Myriad Genetics has essentially given up trying to stop other companies from offering tests for increased risk of breast cancer, ending a dispute that was the subject of a landmark Supreme Court ruling that human genes cannot be patented. The company has settled or is in the process of settling patent-infringement lawsuits it filed against other companies that now offer such testing, a Myriad spokesman said on Tuesday.

Myriad’s lucrative monopoly on testing for mutations in two genes linked to an increased risk of breast and ovarian cancer ended in 2013, when the Supreme Court ruled that human genes were not eligible for patents because they were products of nature. Numerous laboratories began offering tests, some for much less than the roughly \$4,000 Myriad charged for a complete analysis of the two genes, known as BRCA1 and BRCA2.

Myriad sued many of those companies, saying they were infringing other patent claims that had not been invalidated by the Supreme Court.

But last March, a federal district judge in Utah ruled against Myriad's request for a preliminary injunction against one competitor, Ambry Genetics. Last month, the Court of Appeals for the Federal Circuit upheld the lower court's decision and ruled that those remaining claims were also ineligible for patents.

After that ruling, "we decided it was in the best interest of the company to settle these matters," the Myriad spokesman, Ronald Rogers, said. Settlements have been reached with LabCorp, Invitae and Pathway Genomics. Mr. Rogers said Myriad was in talks with Ambry, Quest Diagnostics, GeneDx and Counsyl. In the settlements announced so far, the companies have agreed to dismiss the claims and counterclaims against one another, and Myriad has promised not to sue the companies on any remaining patents in the litigation.

Myriad is shifting from the BRCA gene test to a more comprehensive test of 25 genes linked to cancer risk. It is also developing new types of tests to reduce its reliance on the BRCA test.

http://www.eurekalert.org/pub_releases/2015-01/acs-bcc012815.php

Beer compound could help fend off Alzheimer's and Parkinson's diseases

The health-promoting perks of wine have attracted the spotlight recently, leaving beer in the shadows.

But scientists are discovering new ways in which the latter could be a more healthful beverage than once thought. They're now reporting in ACS' Journal of Agricultural and Food Chemistry that a compound from hops could protect brain cells from damage - and potentially slow the development of disorders such as Alzheimer's and Parkinson's diseases.

Jianguo Fang and colleagues note that mounting evidence suggests that oxidative damage to neuronal cells contributes to the development of diseases that originate in the brain. If scientists could find a way to guard these cells from this type of damage, they might be able to help prevent or slow down Alzheimer's disease, Parkinson's disease and other neurodegenerative conditions. One compound found in hops, called xanthohumol, has gotten the attention of researchers for its potential benefits, including antioxidation, cardiovascular protection and anticancer properties.

Fang's team decided to test xanthohumol's effects on brain cells.

In lab tests, the researchers found that the compound could protect neuronal cells and potentially help slow the development of brain disorders. The scientists conclude xanthohumol could be a good candidate for fighting such conditions.

The authors acknowledge funding from Lanzhou University and the Natural Science Foundation of Gansu Province.

http://www.eurekalert.org/pub_releases/2015-01/lsh-lhn012815.php

LSU Health New Orleans research finds novel compound switches off epilepsy development

Novel compound helps curtail the onset and progression of temporal lobe epilepsy

New Orleans, LA - Researchers at the LSU Health New Orleans Neuroscience Center of Excellence have found that a novel compound they discovered helps curtail the onset and progression of temporal lobe epilepsy. The finding, which may contribute to the development of anti-epileptic therapies, is [published online in the journal PLOS ONE](#).

In temporal lobe epilepsy, seizures arise in the hippocampus and other structures of the limbic system located in the temporal lobe when a cascade of molecular and cellular events results in aberrant brain wiring. (The limbic system is the region of the brain associated with memory and emotions.) Seizures reflect uncontrolled electrical brain activity. The period between a brain injury and the onset of seizures, called epileptogenesis, is a "silent" period because this brain abnormality cannot be detected by current neurological exams or electroencephalography (EEG).

Temporal lobe epilepsy (TLE), or limbic epilepsy, is a common adult epileptic disorder characterized by spontaneous recurrent seizures that may also spread to other brain regions, triggering secondary severe generalized seizures. Aside from neurosurgery, which benefits only a small population of TLE patients, there are no other effective treatments or preventive strategies.

Working in a mouse model, the research team led by Drs. Nicolas Bazan, Boyd Professor and Director of the LSU Health New Orleans Neuroscience Center of Excellence, and Alberto Musto, Assistant Professor of Research, Neurosurgery and Neuroscience, found that brief, small electrical microbursts, or microseizures, occur before the onset of clinical recurrent seizures. When they systemically administered Neuroprotectin D-1 (NPD1), the researchers discovered that NPD1 regulated these bursts of brain electrical activity that not only reduced the aberrant brain cell signaling leading to severe generalized seizures, but also spontaneous recurrent seizures. Neuroprotectin D-1, discovered in the Bazan lab, is derived from docosahexaenoic acid (DHA), an essential omega 3 fatty acid found in fish oil.

"We have searched for years to unravel the significance of the mechanism by which DHA is released in the brain at the onset of seizures," notes Dr. Bazan. Called the "Bazan Effect" in the literature, with the discovery of NDP1, another piece of the puzzle fell into place.

Epilepsy is a chronic neurological disorder characterized by recurrent seizures. It's estimated that 66 million people in the world have epilepsy. In the US, 1 in 26 people will develop epilepsy at some time during their lifetime. The incidence of epilepsy is higher in young children and older adults. Although the cause of epilepsy is unknown, there are some types of epilepsy associated with previous brain injury. Recurrent seizures might cause brain damage.

According to the Epilepsy Foundation, temporal lobe epilepsy is the most common form of partial or localization related epilepsy. It accounts for approximately 60% of all patients with epilepsy. The medial form accounts for almost 80% of all temporal lobe seizures. While medial temporal lobe epilepsy is a very common form of epilepsy, it is also frequently resistant to medications. The overall prognosis for patients with drug-resistant medial temporal lobe epilepsy includes a higher risk for memory and mood difficulties. This in turn leads to impairments in quality of life and an increased risk for death, as observed in patients who have frequent seizures failing to respond to treatment.

"These observations will contribute to our ability to predict epileptic events, define key modulators of brain circuits, especially after a brain injury, and provide potential biomarkers and therapeutic approaches for epileptogenesis," says Dr. Musto.

The research team also included Chelsey P. Walker from the LSU Health New Orleans Neuroscience Center of Excellence and Nicos A. Petasis from Loker Hydrocarbon Research Institute at the University of Southern California, Los Angeles. The research was supported by a grant from the National Institute of General Medical Sciences of the National Institutes of Health.

<http://nyti.ms/1yjoAdN>

Skull Fossil Offers New Clues on Human Journey From Africa ***Anthropologists discovered a 55,000-year-old skull fossil in the Manot Cave in western Galilee in 2008, and it was subjected to years of analysis***

By JOHN NOBLE WILFORD JAN. 28, 2015

Anthropologists exploring a cave in Israel have uncovered a rare 55,000-year-old skull fossil that they say has a story to tell of a reverberating transition in human evolution, at a point when and where some early humans were moving out of Africa and apparently interbreeding with Neanderthals.

The story is of when the Levant was a corridor for anatomically modern humans who were expanding out of Africa and then across Eurasia, replacing all other forms of early human-related species. Given the scarcity of human fossils from that time, scholars say, these ancestors of present-day non-African populations had remained largely enigmatic.

From the new fossil find, which could be closely related to the first modern humans to colonize Stone Age Europe, it appears that these people already had physical traits a bit different from the Africans they were leaving behind and many other human inhabitants along the corridor.

Could this support recent genetic evidence that modern Homo sapiens and their Neanderthal cousins interbred, perhaps in the Middle East and most likely between 65,000 and 47,000 years ago? The discovery team urged caution on the interbreeding issue, but noted anatomical features of the cranium suggesting that some human-Neanderthal mixture had presumably occurred before any encounters in Europe and Asia.

The discovery in Manot Cave in western Galilee, made in 2008 and subjected to years of rigorous analysis, was reported on Wednesday in the journal *Nature* by an international team of researchers led by Israel Hershkovitz of Tel Aviv University. They said this was "the first fossil evidence from the critical period when genetic and archaeological models predict that African modern humans successfully migrated out of Africa and colonized Eurasia."

The researchers further concluded that the Manot specimen "provides important clues about the morphology of modern humans in close chronological proximity to a probable interbreeding event with Neanderthals." They also noted that the shape of the cranium established this as a fully modern human at a time when warmer and wetter conditions were favorable for human migration out of Africa. In other words, Dr. Hershkovitz said in an interview, the Manot cranium "is the missing connection between African and European populations."

Scientists not involved with the research team praised the "fascinating new fossil" and the cautious interpretation of its broader implications in understanding the early migrations into Eurasia. "This fossil fits previous predictions," said Eric Delson, a paleoanthropologist at Lehman College of the City University of New York, "which is a nice rarity in our field."

Dr. Delson, also a researcher at the American Museum of Natural History, added, "As always, we want more fossils to document variations in and details about this presumed fossil population."

In an email, Dr. Delson praised the journal authors "for hitting the mark with their analyses and interpretations, reaching all the possible conclusions one could think of: The partial skull combines a basically modern human form with a few features also found in Neanderthals." In addition, he pointed out, the analysis "supports the similarity of its shapes" to those of modern Africans and early modern humans from Europe, such as the Cro-Magnons.

The partial skull, designated Manot 1, is of a fairly small adult individual, its sex undetermined. The distinctive bunlike shape at the base of the skull resembles

modern African and European skulls but differs from other anatomically modern humans from the Levant, and is thus a strong clue that these were among the first humans to settle Europe, scientists said.

Dr. Delson agreed that the evidence “makes it possible that this individual is (or is descended from) a ‘hybrid’ between modern humans and Neanderthals, but as the authors note, such a conclusion cannot be reached from a single fossil, especially as hybrids between species of modern primates usually have some genetically related anatomical oddities.”

One concern is that the fossil skull is fairly small, with a somewhat lower braincase capacity than in modern humans. With only one specimen, it is hard to know whether this is normal for the general population, scientists said. And Dr. Delson said it would be interesting to test for DNA in the skull to support its possible hybrid status in a time of overlapping modern human-Neanderthal populations when early humans were making their way, as he phrased it, to “that small zoological backwater of Eurasia known as Europe.”

Excavations at Manot Cave are expected to continue through at least 2020, searching deeper for more fossils and artifacts from the migrating people. Israel, Dr. Hershkovitz said, “is like a Swiss cheese, lots of caves everywhere.”

Several caves in the vicinity of Manot were occupied for long times by Neanderthals between 65,000 and 50,000 years ago. In this respect, Dr. Hershkovitz said, Manot is an excellent place to search for possible hybrids, but they may be difficult to recognize from surface features. “Only DNA study will solve the problem,” he said.

<https://bitly.com/a/bitlinks/1EwjJND>

Health insurers using drug coverage to discriminate

In some US health plans, HIV drugs cost nearly \$3,000 more per year than in other plans; if left unchecked, this practice could partially undermine a central feature of the Affordable Care Act

Boston, MA - Some insurers offering health plans through the new federal marketplace may be using drug coverage decisions to discourage people with HIV from selecting their plans, according to a new study from Harvard T.H. Chan School of Public Health. The researchers found that these insurers are placing all HIV drugs in the highest cost-sharing category in their formularies (lists of the plans' covered drugs and costs), which ends up costing people with HIV several thousands more dollars per year than those enrolled in other plans.

The study appears online January 28, 2015 in the New England Journal of Medicine.

"Eliminating discrimination on the basis of preexisting conditions is one of the central features of the Affordable Care Act (ACA)," said Doug Jacobs, MD/MPH

candidate at the Harvard T.H. Chan School of Public Health and lead author of the study. "However, the use of formularies to increase costs and dissuade those with preexisting conditions such as HIV from enrolling in the plan threatens to at least partially undermine this goal of the ACA."

Jacobs and senior author Benjamin Sommers, assistant professor of health policy and economics, analyzed what they call "adverse tiering" - in which all drugs for certain conditions are placed in the highest cost-sharing tiers - in 12 states in the federal marketplace. They compared plans in six states that had been mentioned in a complaint to the U.S. Department of Health and Human Services (HHS) about adverse tiering (Delaware, Florida, Louisiana, Michigan, South Carolina, and Utah), and in the six most populous states without insurers in the HHS complaint (Illinois, New Jersey, Ohio, Pennsylvania, Texas, and Virginia). They compared cost-sharing for a commonly prescribed class of HIV medication - nucleoside reverse-transcriptase inhibitors, or NRTIs.

The researchers found that 25% of the plans examined used discriminatory drug tiering for NRTIs. The differences in out-of-pocket HIV drug costs between adverse-tiering plans (ATPs) and other plans were stark. People in ATPs on average paid three times more for HIV medications than people in non-ATP plans, with a nearly \$2,000 annual difference even for generic drugs. Even though annual premiums in the ATPs tended to be lower than other plans, the high cost of HIV drugs in the ATPs meant that, on average, a person with HIV would pay \$3,000 more for treatment each year than if he or she had instead enrolled in a non-ATP plan.

The researchers noted that insurers' use of "adverse tiering" puts significant and unexpected financial strain on those with chronic conditions. They added that, over time, the practice could lead to sicker people clustering in plans that offer more generous prescription drug benefits - which could in turn create a "race to the bottom" in insurers' drug plan designs as they try to avoid a large influx of sick enrollees that would negatively affect profits.

"The ACA has made a major positive change for people with preexisting conditions - they can now purchase insurance without paying higher premiums or getting denied coverage," said Jacobs. "But some insurance companies seem to be setting up formularies that continue to discriminate against people with chronic conditions, and policymakers should consider steps to prevent these discriminatory practices in the future."

"Using Drugs to Discriminate - Adverse Selection in the Insurance Marketplace," Douglas B. Jacobs and Benjamin D. Sommers, New England Journal of Medicine, January 28, 2015, DOI: 10.1056/NEJMp1411376

http://www.eurekalert.org/pub_releases/2015-01/ez-tf012815.php

The 2 faces of Mars

Mars has two differently shaped hemispheres: the lowlands of the northern hemisphere and the volcanic highlands of the southern hemisphere.

The two hemispheres of Mars are more different from any other planet in our solar system. Non-volcanic, flat lowlands characterise the northern hemisphere, while highlands punctuated by countless volcanoes extend across the southern hemisphere. Although theories and assumptions about the origin of this so-called and often-discussed Mars dichotomy abound, there are very few definitive answers. ETH Zurich geophysicists with Giovanni Leone are now providing a new explanation. Leone is the lead author of a paper recently published in the journal *Geophysical Research Letters*.

Using a computer model, the scientists have concluded that a large celestial object must have smashed into the Martian south pole in the early history of the Solar System. Their simulation shows that this impact generated so much energy that it created a magma ocean, which would have extended across what is today's southern hemisphere. The celestial body that struck Mars must have been at least one-tenth the mass of Mars to be able to unleash enough energy to create this magma ocean. The molten rock eventually solidified into the mountainous highlands that today comprise the southern hemisphere of Mars.

Volcanic activity stopped 3.5 billion years ago

In their simulation, the researchers assumed that the celestial body consisted to a large degree of iron, had a radius of at least 1,600 kilometres, and crashed into Mars at a speed of five kilometres per second. The event is estimated to have occurred around 4 to 15 million years after the Red Planet was formed. Mars' crust must have been very thin at that time, like the hard, caramelised surface of a crème brûlée. And, just like the popular dessert, hiding beneath the surface was a liquid interior.

When the celestial object impacted, it added more mass to Mars, particularly iron. But the simulation also found that it triggered strong volcanic activity. Around the equator in particular, numerous mantle plumes were generated as a consequence of the impact, which migrated to the south pole where they ended. Mantle plumes are magma columns that transport liquid material from the mantle to the surface. In the model, the researchers found that activity on Mars died down around 3.5 billion years, after which time the Red Planet experienced neither volcanic activity nor a magnetic field - this is consistent with observations and measurements.

Volcanic activity and topography modelled under realistic conditions

Earlier theories posited the opposite, namely that there must have been a gigantic impact or many smaller strikes against the northern hemisphere. The most important theory about the origin of the Mars dichotomy was formulated by two American researchers in 1984 in an article in the journal *Nature*. They postulated that a large celestial object struck the Martian north pole. In 2008 a different team revived this idea and published it once again in *Nature*.

This theory did not convince Leone: "Our scenarios more closely reflect a range of observations about Mars than the theory of a northern hemisphere impact," states Leone. The volcanoes on Mars are very unevenly distributed: they are common and widespread on the southern hemisphere, but are rare and limited to only a few small regions in the northern hemisphere. "Our model is an almost identical depiction of the actual distribution of volcanic identity," asserts Leone.

According to the researcher, no other model has been able to portray or explain this distribution before.

Their simulation was also able to reproduce the different topographies of the two hemispheres in a nearly realistic manner, says Leone. And he goes on to explain that the model - depending on the composition of the impact body chosen - is a virtually perfect representation of the size and shape of the hemispheres. One condition, however, is that the celestial body impacting Mars consist of 80 per cent iron; when the researchers simulated the impact with a celestial body made of pure silicate rock, the resulting image did not correspond to the reality of the dichotomy.

Magnetic field tipped the balance

Lastly, the model developed by the ETH researchers confirmed the date on which the magnetic field on Mars ceased to exist. The date calculated by the model corresponds to around 4.1 billion years ago, a figure previously proven by other scientists. The model also demonstrates why it ceased: a sharp decrease in heat flow from the core into the mantle and the crust in the first 400 million years after the impact. After a billion years, the heat flow was only one-tenth its initial value, which was too low to maintain even the volcanism. The model's calculations closely match previous calculations and mineralogical explorations.

The volcanic activity is related to the heat flow, explains Leone, though the degree of volcanic activity could be varied in the simulation and influenced by the strength of the impact. This, he states, is in turn linked to the size and composition of the celestial object. In other words, the larger it is, the stronger the volcanic activity is. Nevertheless, after one billion years the volcanic vents were extinguished - regardless of the size of the impact.

It has become increasingly clear to Giovanni Leone that Mars has always been an extremely hostile planet, and he considers it almost impossible that it ever had

oceans or even rivers of water. "Before becoming the cold and dry desert of today, this planet was characterised by intense heat and volcanic activity, which would have evaporated any possible water and made the emergence of life highly unlikely," asserts the planet researcher.

Leone G, Tackley PJ, Gerya TV, May DA, Zhu G (2014). Three-dimensional simulations of the southern polar giant impact hypothesis for the origin of the Martian dichotomy, Geophys. Res. Lett., 41, doi:10.1002/2014GL062261

http://www.eurekalert.org/pub_releases/2015-01/uab-dgl012815.php

Did genetic links to modern maladies provide ancient benefits?

A study finds that humanity's early ancestors had genetic variations associated with modern disease, and now the question is why

BUFFALO, N.Y. - Psoriasis, a chronic skin condition, can cause rashes that itch and sting.

So why would a genetic susceptibility to this and other ailments persist for hundreds of thousands of years, afflicting our ancient ancestors, and us?

That's the question scientists are asking after discovering that genetic variations associated with some modern maladies are extremely old, predating the evolution of Neanderthals, Denisovans (another ancient hominin) and contemporary humans. The study was published this month in *Molecular Biology and Evolution*.

"Our research shows that some genetic features associated with psoriasis, Crohn's disease and other aspects of human health are ancient," says senior scientist Omer Gokcumen, PhD, a University at Buffalo assistant professor of biological sciences. Some of humanity's early ancestors had the telltale features, called deletions, while others did not, mirroring the variation in modern humans, the scientists found. This genetic diversity may have arisen as far back as a million or more years ago in a common ancestor of humans, Denisovans and Neanderthals.

The discovery highlights the importance of balancing selection, a poorly understood evolutionary dance in which dueling forces drive species to retain a diverse set of genetic features. The research raises the possibility that the diseases in question - or at least a genetic susceptibility to them - "may have been with us for a long time," Gokcumen says.

Why this would happen is an open question, but one possibility is that certain traits that made humans susceptible to Crohn's and psoriasis may also have afforded an evolutionary benefit to our ancient ancestors.

Dueling forces shape evolution:

Though we often think of evolution as black and white - a trait is either good or bad - there are instances where the line is not so clear, Gokcumen says.

"The best example of this is sickle cell anemia," he explains. The disorder causes red blood cells to take on a curved, crescent-like shape, which leads to anemia (a

problem), but also protects against malaria by keeping parasites out of cells (an advantage). These opposing pressures create a balance where the copy of the gene that causes the sickle cell anemia remains in the population in malaria-ridden geographies. The new study hints that the ancient deletions that are associated with Crohn's disease and psoriasis may play similar - but likely more complex - roles in health.

"Crohn's disease and psoriasis are damaging, but our findings suggest that there may be something else - some unknown factor now or in the past - that counteracts the danger when you carry genetic features that may increase susceptibility for these conditions," Gokcumen says. "Both diseases are autoimmune disorders, and one can imagine that in a pathogen-rich environment, a highly active immune system may actually be a good thing even if it increases the chances of an auto-immune response."

Ancient genetic variations maintained due to opposing evolutionary pressures may be "underappreciated," says Yen-Lung Lin, a PhD candidate in UB's Department of Biological Sciences who is lead author in the study. "We're thinking forces that maintain variation might be more relevant to human health and biology than previously believed."

Important genetic variations predate Neanderthals

Gokcumen's team compared modern human genomes to those of other closely related species, including chimpanzees and two archaic hominins: Neanderthals and Denisovans, both of which evolved hundreds of thousands of years ago and whose genomes were sequenced by other scientists using ancient remains. Gokcumen's team identified chunks of DNA that exist in chimpanzees but that were later erased through evolutionary processes. These DNA segments are called deletions, and today, they are present in some human genomes and missing from others.

The study found that certain functionally important deletions that vary among modern humans likely originated in a common ancestor of humans, Neanderthals and Denisovans, possibly dating as far back as a million or more years ago. These unusually old deletions included ones that are common in Crohn's disease and psoriasis patients, as well as deletions linked to a person's ability to respond to a number of drugs, including growth hormone treatments.

In the past, scientists have conducted similar studies examining genetic variations that consist of a single unit of DNA called a nucleotide. The new research investigated longer sequences of DNA, taking advantage of recently available genomic data for modern and ancient hominins. The study demonstrates the power of leveraging such data to investigate different types of genetic differences among humans and to illuminate our species' genetic history.

Gokcumen and Lin's co-authors included Jerry Ajay in UB's Department of Computer Science and Engineering, Pavlos Pavlidis of the Institute of Molecular Biology and Biotechnology, part of the Foundation for Research and Technology-Hellas in Greece, and Emre Karakoc from Max Planck Institute for Evolutionary Biology.

http://www.eurekalert.org/pub_releases/2015-01/uow-sph012815.php

Why upper motor neurons degenerate in ALS

For first time scientists reveal mechanism underlying critical cell degeneration

For the first time, scientists have revealed a mechanism underlying the cellular degeneration of upper motor neurons, a small group of neurons in the brain recently shown to play a major role in ALS pathology.

ALS, or amyotrophic lateral sclerosis, is a fatal neuromuscular disorder marked by the degeneration of motor neurons, which causes muscle weakness and impaired speaking, swallowing and breathing that leads to paralysis and death. Defects in upper motor neurons, which send messages from the brain to the spinal cord to activate voluntary movement, may be a starting point for the disease.

In a new study supported by the Les Turner Foundation, published January 16 in Cerebral Cortex, Northwestern Medicine scientists begin to explain why upper motor neurons are vulnerable to degeneration. They developed a new mouse model for studying these cells, and found that increased stress in the endoplasmic reticulum (ER) is one culprit of upper motor neuron death.

"Now that we appreciate the importance of upper motor neurons, we need to develop therapies that improve their survival," said principal investigator Hande Ozdinler, assistant professor in the Ken and Ruth Davee Department of Neurology. "This study gives us a target to go after, bringing us one step closer to building effective treatment strategies."

The new model features mice without UCHL1 protein function - mutations in UCHL1 gene have previously been implicated in motor defects in human patients. Using in vitro and in vivo methods, the scientists discovered that loss of UCHL1 protein function affects protein regulation pathways, ER stress and upper motor neuron survival.

"In this model, the timing and extent of upper motor neuron degeneration is unprecedented," Ozdinler said. "All the other neurons in the brain remain healthy, which means that this model will be very useful for studying the health of the upper motor neurons." Upper motor neurons make up only about 150,000 of the 2 billion cells in the brain. "In mathematical terms, they're insignificant, but their function is so important. They act as the spokesperson of the brain by collecting, integrating, translating and transmitting brain's message to the spinal cord targets, and by doing so they initiate and modulate voluntary movement," Ozdinler said.

Ozdinler's lab has spearheaded research establishing that upper motor neurons are essential to ALS pathology. Previously, scientists thought that spinal motor neurons were more important in ALS pathology - that upper motor neuron death was a mere secondary effect. In 2012, her group showed that an early event in ALS is spine loss in the apical dendrites of upper motor neurons, where they make connections with other neurons in the brain. In 2013, the lab generated the first reporter line for upper motor neurons, to help scientists visualize them with a green fluorescent protein.

"Now that we have a model and reporter line, we have the tools to develop therapies directed at the upper motor neurons," Ozdinler said. "Survival requirements of these neurons cannot be ignored in ALS and in other diseases in which voluntary movement is impaired."

The findings of the study could have applications to other neurodegenerative diseases that may share ER stress as an underlying cause. "Parkinson's, Alzheimer's and ALS are different branches of the same tree," Ozdinler said. "Subpopulations of patients may be developing these diseases due to the same dysfunctional cellular pathways. Finding a therapy for the pathway could help all of these patients."

Ozdinler is a member of the Robert H. Lurie Comprehensive Cancer Center of Northwestern University, and the Cognitive Neurology and Alzheimer's Disease Research Center. Other Northwestern authors on the study are Javier Jara, Bar?? Genç, Dr. Emel Ulup?nar and Martha Bohn.

This study also was funded by National Institutes of Health (NIH) grant R01NS085161-01, the Les Turner ALS Foundation, Wenske Foundation, NUCATS, NIH/ National Institute of Neurological Disorders and Stroke grants NS49553, NS49553 and NS41590, NIH Mechanisms of Aging and Dementia Postdoctoral Training Program and ALSA Safenowitz fellowship.

http://www.eurekalert.org/pub_releases/2015-01/su-ssu012815.php

Stanford scientists use ocean waves to monitor offshore oil and gas fields

Passive probing of the seafloor using weak seismic waves generated by the ocean provides real-time monitoring of the subsurface

A technology developed by Stanford scientists for passively probing the seafloor using weak seismic waves generated by the ocean could revolutionize offshore oil and natural gas extraction by providing real-time monitoring of the subsurface while lessening the impact on marine life.

"We've shown that we can generate images of the subsurface nearly every day instead of taking snapshots just two or three times a year," said Biondo Biondi, professor of geophysics at Stanford's School of Earth Sciences.

Currently, many energy companies use a technique called time-lapse reflection seismology to monitor offshore oil and gas deposits to optimize production and look for hazards such as hidden gas pockets. Reflection seismology involves ships towing arrays of "air guns" that explode every 10 to 15 seconds to produce loud sound pulses. The pulses bounce off the seafloor and geological formations beneath, then journey back to the surface, where they are recorded by hydrophones. The data are then deciphered to reveal details about subsurface structures.

Each survey can cost tens of millions of dollars, and as a result they are only conducted two to three times a year. Environmental groups and marine biologists have expressed concerns about the use of air guns for contributing to noise pollution in the ocean that can disturb or even injure marine animals, including humpback whales and giant squid.

The new technique developed by Biondi and Sjoerd de Ridder, a student of Biondi's who is now a postdoctoral scientist at the University of Edinburgh, is different. It exploits naturally occurring seismic waves generated by Earth's oceans that are several orders of magnitude weaker than those produced by earthquakes.

Ambient seismicity

As ocean waves collide with one another, they create pressures on the sea floor, where they generate seismic waves that then propagate in every direction. Scientists have known about this "ambient seismic field" for nearly a century, but it was only recently that they understood ways to harness it.

"We knew the ambient seismic energy was there, but we didn't know what we could do with it," De Ridder said. "That understanding has only been developed in recent years. Our technique provides the first large-scale application to harness it for oil and gas production."

The technique that Biondi and De Ridder developed, called ambient seismic field noise-correlation tomography, or ASNT, uses sensors embedded in the seafloor. The sensors, which are typically installed by robotic submersibles, are connected to one another by cables and arranged into parallel rows that can span several kilometers of the seafloor. Another cable connects the sensor array to a platform in order to collect data in real time.

The sensors record ambient seismic waves traveling through Earth's crust. The waves are ubiquitous, continuously generated and traveling in every direction, but using careful signal-processing schemes they developed, Biondi and De Ridder can digitally isolate only those waves that are passing through one sensor and then another one downstream. When this is done repeatedly, and for multiple sensors

in the network, what emerges is a "virtual" seismic wave pattern that is remarkably similar to the kind generated by air guns.

Less disruptive

Because the ASNT technique is entirely passive, meaning it does not require a controlled explosion or a loud air gun blast to create a seismic wave signature, it can be performed for a fraction of the cost of an active-reflection-seismology survey and should be far less disruptive to marine life, the scientists say. Since 2007, Biondi and De Ridder have been testing and refining their technique in a real-world laboratory in Europe. The scientists worked with the energy companies BP and ConocoPhillips to study recordings from existing sensor arrays in the Valhall and Ekofisk oil fields in the North Sea that are capable of recording ambient seismic waves.

The proof-of-concept experiment has been successful, and the scientists have demonstrated that they can image the subsurface at Valhall down to a depth of nearly 1,000 feet. "We've now shown that our technique can very reliably and repeatedly retrieve an image of the near-surface," De Ridder said. "Our hope is that they can also reveal changes in the rocks that could signal an impending problem."

The Stanford scientists outlined their technique and detailed some of their results from Valhall, as well as from Ekofisk, in a series of technical papers, the latest of which was recently published in the journal *Geophysical Research Letters*.

This research was mostly funded by the Stanford Exploration Project affiliate program, with the contribution of a grant from the Global Climate and Energy Project (GCEP).

<http://www.medscape.com/viewarticle/836626>

When a Patient With a Black Eye Claims Everything Is Fine: Detecting Domestic Violence

When Domestic Violence Appears in Your Office

Batya Swift Yasgur, MA, LMSW

It's a Monday morning, and you notice something unusual about a patient who comes to your office with flu-like symptoms. She has a black eye, which she says was caused by bumping into a closet. On the basis of past conversations, you're sure she was assaulted by her husband, and you know you have an ethical responsibility to report suspected intimate partner violence - and in many states, a legal obligation to do so - but what happens if the patient denies it?

That quandary is not uncommon. [Medscape's 2014 Ethics Survey](#) asked physicians, "Have you ever suspected domestic abuse of a patient but not reported it or investigated further?" Eleven percent of the more than 21,000 respondents said yes. Some elaborated:

- *"I have always made sure to ask the patient in private, but if they lied to me, there's nothing I can do."*
- *"Often the suspicion is based on multiple chronic complaints, and not objective findings. If the patient does not endorse abuse, you have no grounds for instigating investigation."*
- *"I suspected it, but the patient denied it. I observed closely but found no additional evidence, so I did not report it."*
- *"The patient told me her boyfriend beat her up and that if I reported it, he would kill her three children, whom he was watching at the time of her ER visit. I had to promise this so that the patient would agree to be treated. Sad."*

One expert says that in such cases, while you shouldn't give up and move on, at the same time you don't want to come across as overbearing.

"Do take 'no' for an answer," says Barbara Gerbert, PhD, professor emeritus, University of California at San Francisco, Division of Behavioral Science. "Your job isn't to badger the patient until she breaks down and admits it. Gently backing off is important, so that she feels safe. But keep asking, at every visit." It may take years before the woman acknowledges the abuse, and even longer before she's willing to leave the relationship. Keeping phone numbers and resources in the bathroom will give the patient the opportunity to peruse them in private. "I call this the 'planting seeds approach,'" Dr Gerbert says.

In states where it's mandatory to report suspicious injuries, such as California, some doctors are very open with patients about their legal obligation. "I've never had a patient clam up or shut down because of this. In fact, any time I've had to report, the woman has appreciated it," says Catherine Gutfreund, MD, of the Department of Family Medicine, and Domestic Violence Lead Physician, Kaiser Permanente, Santa Rosa, California.

In many cases, telling the woman that the injury must be reported can remove the denial and open up the discussion.

Domestic Violence Knows No Bounds

Dr Gerbert recounts a story of a colleague whose "deluxe" ob/gyn practice served an upscale, affluent community. "She kept informational cards with phone numbers of shelters and hotlines in the private bathroom adjacent to the examining room and was surprised that she had to replace those cards weekly."

As this story illustrates, intimate partner violence cuts across all socioeconomic, racial, and ethnic populations. "It's a mistake to assume that this issue affects primarily patients from lower socioeconomic groups," says Dr Gerbert.

The benefits of intervention go beyond the individual patient. "Domestic violence affects the whole family," Dr Gutfreund points out. "Children in homes where a parent is being abused suffer serious adverse effects in childhood and adulthood, even if they themselves aren't currently being abused."

And because domestic violence typically increases stress-related chronic illness, such as headaches and dyspepsia, it also burdens the healthcare system and increases overall costs.

"Learning to screen for and address intimate partner violence in the medical setting is a skill that must be mastered, not only for the sake of the patient but also for the sake of other family members, and society as a whole," says Dr Gutfreund.

Who Should Be Screened?

In 2013, the US Preventive Services Task Force (USPSTF) issued a recommendation for clinicians to screen women of childbearing age for intimate partner violence, and refer women who screen positive to intervention services.^[1] Although the USPSTF only mentions women of childbearing age, women of any age are at risk, says Peter Cronholm, MD, MSCE, Associate Professor of Family Medicine and Community Health, University of Pennsylvania, Philadelphia. And men are also at risk. Studies have shown that 1 in 3 women and 1 in 4 men have experienced some form of physical violence by an intimate partner during their lifetime.^[2]

This raises the question, when does a normal argument become "violence"? Obviously, there will be disagreements between couples and tempers may flare, but "a relationship where one person is controlling, putting down, emotionally manipulating, or pressuring the partner into doing things they don't want to do is abusive," says Dr Gerbert. "Threatening to hurt the partner, children, or pets or throwing objects are all forms of violence."

Dr Gutfreund agrees. "The adage 'Sticks and stones may break my bones but words will never hurt me' is absolutely wrong when it comes to emotional abuse."

"First, Do No Harm": How to Screen

Many physicians are "nervous about the potential to do harm by inquiring," says Dr Cronholm. "They worry that screening might put patients at risk. But there's been no evidence that screening women has brought demonstrable harm."

The key, according to experts, is to screen and intervene skillfully. And that includes the cardinal rule of never asking in front of the potential perpetrator.

In most cases, you'll have no trouble conducting your screening when the patient is alone. But sometimes the partner refuses to leave. In cases like these, Dr Gerbert says, you'll have to come up with some creative ways to get him (or her) out of the room.

Some physicians have a signal with their staff, Dr Gerbert reports. "They say to the nurse, 'Please take Mr Smith down the hall and have him fill out some additional forms.' Or they say directly to Mr Smith, 'I always conduct the physical exam in private.'"

If you can't assure privacy at this particular visit, schedule a follow-up or order laboratory tests, adds Dr Cronholm.

Include the Question in the Social History

Many patients respond better if your inquiry is included with other routine questions. "I ask this in the social history, rather than the checklist for health maintenance," notes Erin Marcus, MD, associate professor of clinical medicine and public health, University of Miami Miller School of Medicine.

"I ask the patient about their occupation and their home setting. Then I look for an appropriate opening to ask whether they feel safe in their home," says Dr Marcus, who is also co-director of the University of Miami/Jackson Memorial Hospital Internal Medicine Residency Training Clinic.

That's a smart approach, Dr Cronholm says. "Funneling the question into other routine questions not only systematizes but also normalizes the issue," he explains. "I ask about alcohol use, smoking, and use of seatbelts, and then inquire about safety."

Any physician encounter can be an opportunity to address possible domestic abuse. For example, Dr Gutfreund includes some screening questions when children come for well check-ups: "We ask how the child is developing and how things are at home," she says. Often these seemingly innocent questions can prompt the parent to disclose some bigger issues.

Use Screening Tools

Odds are that patients who won't open up to you in person may do so in writing. "There's good data that people will disclose intimate partner violence on paper- or computer-based screeners," Dr Cronholm notes. The USPSTF has validated six brief tools that clinicians can use in their practice.^[1]

Dr Gutfreund adds, "We append questions to the nine-item Patient Health Questionnaire (PHQ-9) - the brief tool used for depression screening - regarding past and present domestic violence."

Physical Signs of Abuse

Obviously, bruising and injuries are the biggest red flags. "When you see these things, asking and labeling the phenomenon as physical harm can open the door," Dr Gerbert advises. "You can say, 'I see you have bruising around your neck. Did someone try to hurt you?' And you can label it without saying for sure that it's due to violence."

But don't ignore less dramatic presentations. Patients with frequent vague, nonspecific complaints or chronic conditions without an adequate physical explanation are often victims of domestic violence. Make sure you take the time to question these patients as well.

And although we've focused mainly on women thus far, men also may be abused: in heterosexual, homosexual, or transgender relationships, and across all social and economic levels. But it can be more difficult to elicit information about intimate partner abuse from men, Dr Gutfreund says, because they typically feel greater shame at being abused. (Resources for men are provided at the end of this article.)

Experts agree, however, on three things that must be done in cases of suspected intimate partner violence: provide emotional support, referrals, and resources.

Emotional Support

Being empathetic is critical, according to Dr Gerbert. "You can say, 'I've seen other women who were being hurt at home. In fact, one third of American women have experienced this.' This communicates that you're not shocked and that she's not alone."

Low self-esteem is prevalent in victims of violence, who tend to feel isolated and ashamed, so experts say it's very important to be nonjudgmental. Moreover, and not surprisingly, people are less likely to open up when they feel they're being judged.

Convey, too, that you respect the patient's timing in opening up to you. "On average, it takes a woman years to leave an abusive relationship. If she feels you're not pushing her, she will feel more supported." Always offer empathy and validation, helping her feel that you're on her team and will be there for her.

When to Refer

It's important to get to know local community resources, such as domestic violence prevention programs, hotlines, mental health centers, specialized therapists, and women's shelters. Sometimes, it's enough to provide your patients with these names.

But on other occasions, more extreme measures may be called for. In situations like these, Dr Gerbert advises a "warm handoff." While the patient is sitting in your office and the partner isn't around, pick up the phone, call the social worker or other support system, and hand the phone to the patient right then and there. In very urgent situations, it may be possible to have the woman hospitalized somewhere that the perpetrator can't find her. That gives the patient her privacy and a place to stay until she can safely go somewhere else.

Provide Resources

Commit the phone numbers for domestic violence hotlines or crisis lines to memory; the number for the National Domestic Violence Hotline is (800) 799-SAFE (7233). Have handouts, including hotline phone numbers, and other information available in multiple languages, Dr Gutfreund says.

Remember, adds Dr Gerbert, that many patients can't come home with a pile of papers because this could inflame the perpetrator. So be creative in how you present the materials. For example, a hotline number can be provided on a piece of paper small enough to slip into a lipstick container, pillbox, or tampon container.

Help the patient explore potential options for leaving her abuser, including an escape route; a safe place to stay; and an "emergency escape kit," including money, car keys, and important documents.

Know Your State's Laws

Reporting procedures may differ by state. For example, should the violence be reported to the police in the county where it occurred, in the city where it occurred, or in the city where you practice? And familiarize yourself with the Violence Against Women Act,^[3] to reassure undocumented immigrants that they won't be turned in to the Department of Homeland Security and deported if they're referred to a domestic abuse program or a women's shelter.

Make sure you document all discussions with the patient about domestic violence and all actions taken, such as referrals to other professionals or reports to the police. Suggestions for documentation are provided in the resources listed below.

Conclusion

High-profile celebrity cases, such as that of professional football player Ray Rice, who assaulted his then-fiancée, have placed domestic violence prominently on the map and can be helpful in broaching the subject with patients. Using these and other talking points can facilitate proactive screening and targeted intervention, and enable physicians to play an important role in alleviating this destructive societal problem.

Resources

Dudgeon A, Evanson TA. Intimate partner violence in rural U.S. areas: what every nurse should know. *Am J Nurs.* 2014;114:26-35.

Family Violence Prevention Fund. *Compendium of state statutes and policies on domestic violence and health care.*

http://www.acf.hhs.gov/sites/default/files/fysb/state_compendium.pdf Accessed November 10, 2014.

American Academy of Orthopaedic Surgeons/American Association of Orthopaedic Surgeons. *Family violence state statutes.* <http://www.aaos.org/about/abuse/ststatut.asp> Accessed November 10, 2014.

Helpguide.org. *Help for abused men.* <http://www.helpguide.org/articles/abuse/help-for-abused-men.htm> Accessed November 10, 2014.

US Department of Justice, Office of Justice Programs, National Institute of Justice.

Documenting domestic violence: how health care providers can help victims.

<https://www.ncjrs.gov/pdffiles1/nij/188564.pdf> Accessed November 10, 2014.

Lentz L. 10 tips for documenting domestic violence. *Nursing.* 2010;40:53-55.

Moyer VA; U.S. Preventive Services Task Force. Screening for intimate partner violence and abuse of elderly and vulnerable adults: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med.* 2013;158:478-486.

References

1. Moyer VA; U.S. Preventive Services Task Force. Screening for intimate partner violence and abuse of elderly and vulnerable adults: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med.* 2013;158:478-486. [Abstract](#)

2. Black MC, Basile KC, Breiding MJ, et al. *The National Intimate Partner and Sexual Violence Survey: 2010 summary report.* Atlanta: National Center for Injury Prevention and Control, Centers for Disease Control and Prevention; 2011.

http://www.cdc.gov/violenceprevention/pdf/nisvs_report2010-a.pdf Accessed November 10, 2014.

3. US Government Printing Office. *113th Congress of the United States of America. Violence Against Women Reauthorization Act of 2013.* <http://www.gpo.gov/fdsys/pkg/BILLS-113s47enr/pdf/BILLS-113s47enr.pdf> Accessed November 10, 2014.

<http://bit.ly/TwO9M4J>

23 Kids' Peanut Allergies Were Cured, At Least Temporarily

A probiotic may be the key to fighting allergies to peanut proteins

By Erin Blakemore smithsonian.com

Peanut allergies are increasingly common and extremely dangerous, especially for kids. But a team of Australian researchers say they were able to cure the potentially fatal allergy - at least temporarily - in a small group of Australian children.

In Melbourne, Australia, researchers treated a group of 28 kids with peanut allergies with a probiotic and peanut protein and a control group of 28 allergic kids with a placebo. Over the next 18 months, the researchers increased the dose of peanut protein for the test group. By the end of the trial, 23 of kids in that group - about 80 percent of them - were able to eat peanuts without any reaction at all.

"These findings provide the first vital step towards developing a cure for peanut allergy and possibly other food allergies," Mimi Tang, a pediatric allergist immunologist who led the study, told the Australian Associated Press.

The probiotic/peanut treatment could be big news for parents of the 1.4 percent of American children who have peanut allergies, but researchers are urging caution. They note that one of the children in the control group was able to eat peanut products after the trial concluded, even though that child only received a placebo. Scientists have no idea if the effects of the study will be permanent - and since the study did cause "serious reactions" in some children during treatment, they warn parents not to try replicating the experiment at home.

<http://bit.ly/1wOlxYN>

Chemists Confirm the Existence of New Type of Bond

A “vibrational” chemical bond predicted in the 1980s is demonstrated experimentally

Jan 20, 2015 | By Amy Nordrum | Véalo en español

Chemistry has many laws, one of which is that the rate of a reaction speeds up as temperature rises. So, in 1989, when chemists experimenting at a nuclear accelerator in Vancouver observed that a reaction between bromine and muonium - a hydrogen isotope - slowed down when they increased the temperature, they were flummoxed.

Donald Fleming, a University of British Columbia chemist involved with the experiment, thought that perhaps as bromine and muonium co-mingled, they formed an intermediate structure held together by a “vibrational” bond - a bond that other chemists had posed as a theoretical possibility earlier that decade. In this scenario, the lightweight muonium atom would move rapidly between two heavy bromine atoms, “like a Ping Pong ball bouncing between two bowling balls,” Fleming says. The oscillating atom would briefly hold the two bromine atoms together and reduce the overall energy, and therefore speed, of the reaction. (With a Fleming working on a bond, you could say the atomic interaction is shaken, not stirred.)

At the time of the experiment, the necessary equipment was not available to examine the milliseconds-long reaction closely enough to determine whether such vibrational bonding existed. Over the past 25 years, however, chemists' ability to track subtle changes in energy levels within reactions has greatly improved, so Fleming and his colleagues ran their reaction again three years ago in the nuclear accelerator at Rutherford Appleton Laboratory in England. Based on calculations from both experiments and the work of collaborating theoretical chemists at Free University of Berlin and Saitama University in Japan, they concluded that muonium and bromine were indeed forming a new type of temporary bond. Its vibrational nature lowered the total energy of the intermediate bromine-muonium structure - thereby explaining why the reaction slowed even though the temperature was rising.

The team reported its results last December in *Angewandte Chemie International Edition*, a publication of the German Chemical Society. The work confirms that vibrational bonds - fleeting though they may be - should be added to the list of known chemical bonds. And although the bromine-muonium reaction was an “ideal” system to verify vibrational bonding, Fleming predicts the phenomenon also occurs in other reactions between heavy and light atoms.

<http://bit.ly/1uPi4OP>

Three Stanford Graduates Are Matching Unused Prescriptions With Patients Who Need Them

Unopened drugs - billions of dollars worth - are trashed in this country each year. What if they instead went to the 50 million who can't afford them?

By Megan Gambino smithsonian.com

Adam Kircher was a healthcare consultant for McKinsey and Company. Kiah Williams was leading the Clinton Foundation's childhood obesity initiative, and George Wang, an expert in the nation's drug donation laws, was working on several legislative initiatives around the country, when all three Stanford graduates quit their jobs in 2011 to found SIRUM.

The four-year-old startup, Supporting Initiatives to Redistribute Unused Medicine - or SIRUM, for short - connects pharmacies, drug manufacturers, nursing homes and other health facilities with excess, unexpired prescriptions to safety-net clinics that can dole out the medications to patients needing them for free. The company is providing this service in California, Oregon and Colorado and hopes to expand its operations into the 39 other states where drug donation is legal. The three founders share their story with Smithsonian.com.

Let's start with the problem. What problem are you trying to fix?

Williams: We are trying to solve two problems simultaneously.

Medication is second only to insurance premiums as America's highest out-of-pocket healthcare cost. As a result, one in four working-age adults in the United States skip taking prescription medication due to cost. Society ends up paying a much higher price when patients skip medications and let diseases go untreated. Taxpayers ultimately foot costlier bills for worse conditions and pay for avoidable emergency room visits.

At the same time, as patients struggle to afford medications, America is destroying about \$5 billion worth of unused, unexpired medicine each year.

Nurses, doctors and pharmacists at healthcare institutions across the U.S. spend countless valuable hours popping out perfectly good pills and squeezing out creams and solutions into trash cans. These wasted medications get incinerated, dumped and flushed and ultimately end up in our air and water supplies, where they pose significant environmental and health hazards.

So, what exactly is SIRUM?

Wang: SIRUM is a non-profit designed to solve those two inefficiencies in our healthcare system by matching the surplus that exists with the need that persists. By saving medicine, and delivering it to where it can do the greatest good, SIRUM saves lives, reduces waste and cuts healthcare costs.

Using an online platform and the same modern logistics that make it possible for anyone anywhere in the U.S. to order an Amazon item today and receive it tomorrow, we connect the untapped surplus of drugs from manufacturers, pharmacies and health facilities with the needs of safety-net clinics.

You've called SIRUM the "Match.com of medicine." How does it work exactly?

Kircher: SIRUM's online platform allows donor and recipient organizations to easily upload medicine surpluses or needs they have. Our system then connects compatible donor and recipient organizations and coordinates all donation logistics, including producing itemized drug manifests, and handling all shipping and tracking. Donations are made directly from donor to recipient, creating a fast, efficient donation process with low overhead costs and no middlemen. Once a recipient organization receives a donation, pharmacists or doctors verify the integrity of each donated medication and dispense them to patients in need.

Are there any legal or logistical limitations to your redistribution of medicines? What laws are in place to allow for these transfers?

Wang: Laws typically known as "Good Samaritan" laws exist in 42 states protecting drug donation or redistribution to at least some extent. SIRUM is the only organization in the nation that has created and leveraged the infrastructure needed to operate donation programs in-line with these laws and take full advantage of them.

How did you come up with this concept?

Kircher: I developed the idea for SIRUM in 2005 after witnessing the destruction caused by the 2004 Indonesian tsunami - and the way in which inefficient donation logistics prevented critical medicine from getting to the Indonesians who desperately needed them. An industrial engineering master's degree student at Stanford at the time, I hypothesized that an online peer-to-peer, matchmaking service could reduce the fulfillment time of donated medications from 9 months to a matter of days. Aware of recent legislative changes that for the first time enabled and legally protected medicine donation in 40 states, George and Kiah took my idea out of academia and applied it to donors and clinics directly and domestically in the U.S.

How would you describe your success to date?

Williams: Since starting full-time at SIRUM in 2011, we have created from the ground up, in California, what is now the largest drug redistribution program in the country. Since inception, SIRUM has facilitated the redistribution of 1 million pills worth about \$3 million wholesale directly to safety-net clinics to help serve about 20,000 patients in need. That amounts to two tons of medicine diverted away from our waste streams - and thousands of tons more waste avoided by

forgoing the production of the 1 million pills these safety-net clinics would have otherwise had to purchase anew. SIRUM currently operates programs in California, Colorado and Oregon, with over 200 donor and recipient organizations participating.

As you see it, what is the potential impact SIRUM could have on healthcare?

Williams: Our ultimate vision is to get every one of those \$5 billion worth of medications being wasted to a patient in need. Even if we just stopped the \$700 million of drug waste happening in long-term care facilities alone, we estimate we could fill about 10 million prescriptions.

But it's not just the cost of purchasing medications that we can affect. We could also reduce those secondary costs we incur when we let our most vulnerable go without the medications they need - the emergency room visits, the incarcerations, the lost productivity. And finally, we could save families from having to decide between other basic needs, like fresh food or clothing, and medications - they could have both.

How do you plan to scale your company? What's next?

Kircher: We are currently exploring pilot programs in a few of the other states with Good Samaritan laws while also growing our new programs in Colorado and Oregon, and our flagship program in California. Although we currently mostly work with long-term care facilities, like nursing homes, we are always seeking out donation partners in other parts of the pharmaceutical supply chain, like pharmacies, wholesalers and manufacturers.

<http://www.bbc.com/news/health-31019097>

Ebola outbreak: Virus mutating, scientists warn

Scientists tracking the Ebola outbreak in Guinea say the virus has mutated.

By Tulip Mazumdar Global health reporter

Researchers at the Institut Pasteur in France, which first identified the outbreak last March, are investigating whether it could have become more contagious. More than 22,000 people have been infected with Ebola and 8,795 have died in Guinea, Sierra Leone and Liberia.

Scientists are starting to analyse hundreds of blood samples from Ebola patients in Guinea. They are tracking how the virus is changing and trying to establish whether it's able to jump more easily from person to person

"We know the virus is changing quite a lot," said human geneticist Dr Anavaj Sakuntabhai. "That's important for diagnosing (new cases) and for treatment. We need to know how the virus (is changing) to keep up with our enemy."

It's not unusual for viruses to change over a period time. Ebola is an RNA virus - like HIV and influenza - which have a high rate of mutation. That makes the virus more able to adapt and raises the potential for it to become more contagious.

"We've now seen several cases that don't have any symptoms at all, asymptomatic cases," said Anavaj Sakuntabhai. "These people may be the people who can spread the virus better, but we still don't know that yet. A virus can change itself to less deadly, but more contagious and that's something we are afraid of."

Latest figures

There were fewer than 100 new cases in a week for the first time since June 2014. In the week to 25 January there were 30 cases in Guinea, four in Liberia and 65 in Sierra Leone. The World Health Organization says the epidemic has entered a "second phase" with the focus shifting to ending the epidemic.

But Prof Jonathan Ball, a virologist at the University of Nottingham, says it's still unclear whether more people are actually not showing symptoms in this outbreak compared with previous ones. "We know asymptomatic infections occur... but whether we are seeing more of it in the current outbreak is difficult to ascertain," he said. "It could simply be a numbers game, that the more infection there is out in the wider population, then obviously the more asymptomatic infections we are going to see."

Another common concern is that while the virus has more time and more "hosts" to develop in, Ebola could mutate and eventually become airborne.

There is no evidence to suggest that is happening. The virus is still only passed through direct contact with infected people's body fluids.

"No blood borne virus, for example HIV or Hepatitis B, has ever shown any indication of becoming airborne. The mutation would need to be major," said infectious disease expert Professor David Heyman.

Virologist Noel Tordo from the Institut Pasteur is in the Guinea capital Conakry. He said: "At the moment, not enough has been done in terms of the evolution of the virus both geographically and in the human body, so we have to learn more. But something has shown that there are mutations. "For the moment the way of transmission is still the same. You just have to avoid contact (with a sick person). "But as a scientist you can't predict it won't change. Maybe it will."

Researchers are using a method called genetic sequencing to track changes in the genetic make-up of the virus. So far they have analysed around 20 blood samples from Guinea. Another 600 samples are being sent to the labs in the coming months.

A previous similar study in Sierra Leone showed the Ebola virus mutated considerably in the first 24 days of the outbreak, according to the World Health Organization. It said: "This certainly does raise a lot of scientific questions about transmissibility, response to vaccines and drugs, use of convalescent plasma.

"However, many gene mutations may not have any impact on how the virus responds to drugs or behaves in human populations."

'Global problem'

The research in Paris will also help give scientists a clearer insight into why some people survive Ebola, and others don't. The survival rate of the current outbreak is around 40%. It's hoped this will help scientists developing vaccines to protect people against the virus. Researchers at the Institut Pasteur are currently developing two vaccines which they hope will be in human trials by the end of the year.

One is a modification of the widely used measles vaccine, where people are given a weakened and harmless form of the virus which in turn triggers an immune response. That response fights and defeats the disease if someone comes into contact with it. The idea, if it proves successful, would be that the vaccine would protect against both measles and Ebola.

"We've seen now this is a threat that can be quite large and can extend on a global scale," said Professor James Di Santo, and immunologist at the Institut.

"We've learned this virus is not a problem of Africa, it's a problem for everyone." He added: "This particular outbreak may wane and go away, but we're going to have another infectious outbreak at some point, because the places where the virus hides in nature, for example in small animals, is still a threat for humans in the future. "The best type of response we can think of... is to have vaccination of global populations."

http://www.eurekalert.org/pub_releases/2015-01/mg-rsp012815.php

Research study published - Corn oil helps lower cholesterol more than extra virgin olive oil

A study indicates corn oil significantly reduces cholesterol more than extra virgin olive oil with favorable changes in both total and LDL cholesterol

OAK BROOK TERRACE, Ill. - A study published in the January/February 2015 issue of the Journal of Clinical Lipidology indicates corn oil significantly reduces cholesterol more than extra virgin olive oil with favorable changes in both total and low-density lipoprotein (LDL) cholesterol.

"The study results suggest corn oil has significantly greater effects on blood cholesterol levels than extra virgin olive oil, due, in part, to the natural cholesterol-blocking ability of plant sterols," said lead researcher Dr. Kevin C Maki, PhD, of Biofortis, the clinical research arm of Merieux NutriSciences.

"These findings add to those from prior research supporting corn oil's positive heart health benefits, and align with recommendations to replace saturated fats with unsaturated fats, such as those found in corn oil."

Cardiovascular disease remains the number one cause of death in the United States. Existing research supports the notion that diets containing at least 5-10

percent of calories from polyunsaturated fatty acids (PUFAs) from vegetable oils, are associated with lower risk for heart disease. Additionally, corn oil has a unique combination of healthy fatty acids and plant sterols, which research suggests help lower cholesterol. Corn oil has four times more plant sterols than olive oil and 40 percent more than canola oil. Based on 2013 USDA analysis of corn oil and comparison of other cooking oils, corn oil has a plant sterols content of 135.6 mg/serving vs. 30.0 mg/serving for olive oil. Plant sterols are plant-based substances naturally present in fruits, vegetables, nuts, seeds, cereals, legumes and vegetable oils, such as corn oil. To the extent that plant sterols play a part in reducing blood cholesterol levels, they could have an important role in a heart healthy diet.

Among the 54 healthy men and women in the feeding study, consumption of foods made with corn oil resulted in significantly lower levels of LDL (bad) cholesterol and total cholesterol than the same foods made with extra virgin olive oil. Corn oil lowered LDL cholesterol by 10.9 percent compared to extra virgin olive oil's 3.5 percent reduction, and total cholesterol decreased by 8.2 percent with corn oil compared to 1.8 percent for extra virgin olive oil. Study participants received four tablespoons of corn oil or extra virgin olive oil in the foods provided every day, consistent with the recommended Dietary Guidelines for Americans. All foods were provided to the study participants as part of a weight maintenance diet.

The randomized, double-blind, controlled crossover clinical trial, funded in part by ACH Food Companies, Inc., assessed the effects of dietary oils on fasting lipoprotein lipids. The study compared the effects of corn and extra virgin olive oil on LDL cholesterol (primary outcome variable), total cholesterol, HDL cholesterol (good cholesterol), Non-HDL cholesterol, Triglycerides and the total to HDL cholesterol ratio. Study participants had fasting LDL cholesterol \geq 130 mg/dL and $<$ 200 mg/dL. Fasting blood samples, along with other clinical measurements, were taken from all participants during visits to the clinical study center before and after each treatment phase of the study.

About ACH Food Companies, Inc.

ACH Food Companies, Inc. manufactures, markets and sells a premier branded portfolio of cooking oils, spices and seasonings and baking ingredients in the consumer and foodservice channels in the US, Canada, Puerto Rico and Mexico, all of which are either #1 or #2 brands in their categories. When it comes to baking, ACH features such trusted and loved brands as Fleischmann's® yeast, Fleischmann's® Simply Homemade® bread mixes, Argo® corn starch and baking powder, and Karo® corn syrup. As one of the largest branded consumer oil manufacturers and marketers in North America, the cornerstone of ACH's portfolio features Mazola® oils, the leading corn oil brand in the USA and Canada, and Capullo® oil, the leading premium canola oil brand in Mexico. ACH is the second largest manufacturer

and marketer of spices and seasonings in North America, including Spice Islands® spices and extracts, Durkee®spices, dry sauces and gravies, Weber® Seasonings and Sauces, Tone's® spices, French's® dry sauces, Mazola© brand bouillons, and Patak's® Indian Foods - the leading brand of Indian sauces, pastes and shelf-stable meals in North America.

Biofortis, a Merieux NutriSciences company, is a leading global clinical nutrition research team serving industry leading clients from the food, ingredient and dietary supplement industry segments.

http://www.eurekalert.org/pub_releases/2015-01/uom-lai012915.php

Love and intimacy in later life - new study reveals active sex lives of the over 70s

Older people are continuing to enjoy active sex lives well into their seventies and eighties, according to new research from The University of Manchester and NatCen Social Research.

More than half (54%) of men and almost a third (31%) of women over the age of 70 reported they were still sexually active, with a third of these men and women having frequent sex - meaning at least twice a month - according to data from the latest wave of the English Longitudinal Study of Ageing (ELSA).

The paper, lead authored by Dr. David Lee, an Age UK Research Fellow at The University of Manchester's School of Social Sciences and entitled Sexual health and wellbeing among older men and women in England, is published in the American academic journal, Archives of Sexual Behavior.

It is the first study on sexual health of its kind to include people over the age of 80 and uncovers a detailed picture of the sex lives of older men and women in England, finding that a sizeable minority remain sexually active in their old age. Contrary to popular misconceptions, it finds that overall health and conflicting partnership factors were more closely linked to decreasing sexual activity and functioning, rather than simply increasing age. Of the more than 7000 people who responded to the questionnaire, very few (less than 3%) declined to answer direct questions about their sexual activities and problems.

Dr Lee said: "This is the first nationally-representative study to include people over the age of 80 when asking older men and women in England about their sexual health. "We hope our findings improve public health by countering stereotypes and misconceptions about late-life sexuality, and offer older people a reference against which they may relate their own experiences and expectations. "Our ongoing research is also highlighting the diversity of late-life sexualities, and trying to impose youthful norms of sexual health on older people would be over-simplistic and even unhelpful.

"It is however important that health professionals act on this and are more open about discussing sexual health with older people - it can't simply be assumed to be

an irrelevance." Problems most frequently reported by sexually active women related to becoming sexually aroused (32%) and achieving orgasm (27%), while for men it was erectile difficulties (39%).

Chronic health conditions and poor self-rated health seemed to have more obvious negative impacts on the sexual health of men compared to women.

Men were more concerned about their sexual activities and function than women and, with increasing age, these concerns tended to become more common.

Sexually active women were less dissatisfied with their overall sex lives than men, and also reported decreasing levels of dissatisfaction with increasing age.

The study also found that many septuagenarians and octogenarians were still affectionate towards their partners, with 31% of men and 20% of women reporting frequent kissing or petting. Among those who reported any sexual activity in the past three months, 1% of men and 10% of women reported they felt obligated to have sex.

Caroline Abrahams, Charity Director at Age UK, said: "The fact this is the first time that people over 80 years old have been included in this kind of research highlights how often the public health needs of older people, including sexual health, are ignored or overlooked.

"With an ageing population it is important that providers of sexual health services understand the needs of older people in both clinical settings and when developing information and advice. These recent findings now need to be used to improve sexual health advice and information for older people."

<http://www.manchester.ac.uk/discover/news/article?id=13745>

<http://www.medscape.com/viewarticle/838479>

A New Virus in the Midwest - The Bourbon Virus

Hello. This is Paul Auwaerter with Medscape Infectious Diseases, speaking from Johns Hopkins Division of Infectious Diseases.

Paul G. Auwaerter, MD

Astute clinicians and continued expertise in new molecular diagnostic techniques mean that another virus has been identified. This new virus now deserves consideration in trying to understand what might be afflicting people with an acute febrile illness.

The story goes back to Kansas last summer. A patient, a middle-aged man, was admitted with a fever, flu-like symptoms, muscle aches, liver function test abnormalities, and also an abnormal complete blood cell count. Appropriately, the physicians were initially concerned about a tick-borne disease, such as *Ehrlichia* or Rocky Mountain spotted fever. But the man didn't respond to the typical antibiotic - that is, doxycycline.

So, astute clinicians sent blood, through their state health department, to the Centers for Disease Control - mainly [to look] for a virus identified over 2 years ago called the Heartland virus,^[1,2] which probably is transmitted by the Lone Star tick. That tick has been identified with cases described nearby in Missouri and Tennessee, causing a very similar clinical story.

But the Heartland virus wasn't found. Instead, a new virus was found^[3] - a member of the orthomyxovirus family, a DNA virus, and from a subcategory that wasn't previously known and described to cause human disease. That virus has been named, so far, the Bourbon virus - a classy name, after the county, Bourbon County, in Kansas, from which it was identified. In fact, that county was named after Bourbon County in Kentucky.

At the moment, not much more is known about the virus other than that it was acquired during the summertime. The thought is that it could be another vector-borne illness. Whether it is tick-borne, mosquito-borne, or something else, it is a new virus, the Bourbon virus.

There hasn't yet been a published report of this, so information has spread mainly through press releases and interviews with the Kansas State Health Department.^[3] It is something to be on the watch for as next season comes around. If you have a patient with an unexplained illness, this pathogen might need to be on your list as well. Thanks very much for listening.

1. *Notes from the Field: Heartland Virus Disease - United States, 2012-2013. Morb Mortal Wkly Rep.* 2014;63:270-271.

2. *Savage HM, Godsey MS, Lambert A, et al. First detection of Heartland virus (Bunyaviridae: Phlebovirus) from field collected arthropods. Am J Trop Med Hyg.* 2013;89:445-452.

3. *KDHE Office of Communications (22 December 2014). KDHE and CDC Investigate New Virus. KDHE Office of Communications.*

http://www.kdheks.gov/news/web_archives/2014/12222014.htm Accessed January 16, 2015.

http://www.eurekalert.org/pub_releases/2015-01/ehs-afi012215.php

Added fructose is a principal driver of type 2 diabetes

Clinical experts reporting in Mayo Clinic Proceedings urge drastic reductions in the consumption of foods and beverages containing added sugars, particularly added fructose

Rochester, MN Recent studies have shown that added sugars, particularly those containing fructose, are a principal driver of diabetes and pre-diabetes, even more so than other carbohydrates. Clinical experts writing in Mayo Clinic Proceedings challenge current dietary guidelines that allow up to 25% of total daily calories as added sugars, and propose drastic reductions in the amount of added sugar, and especially added fructose, people consume.

Worldwide, approximately one in ten adults has type 2 diabetes, with the number of individuals afflicted by the disease across the globe more than doubling from 153 million in 1980 to 347 million in 2008. In the United States, 29 million adults (one in eleven) have type 2 diabetes and another 86 million (more than one in three) have pre-diabetes.

"At current levels, added-sugar consumption, and added-fructose consumption in particular, are fueling a worsening epidemic of type 2 diabetes," said lead author James J. DiNicolantonio, PharmD, a cardiovascular research scientist at Saint Luke's Mid America Heart Institute, Kansas City, MO. "Approximately 40% of U.S. adults already have some degree of insulin resistance with projections that nearly the same percentage will eventually develop frank diabetes."

The net result of excess consumption of added fructose is derangement of both overall metabolism and global insulin resistance say the authors. Other dietary sugars not containing fructose seem to be less detrimental in these respects.

Indeed, several clinical trials have shown that compared to glucose or starch, isocaloric exchange with fructose or sucrose leads to increases in fasting insulin, fasting glucose, and the insulin/glucose responses to a sucrose load. "This suggests that sucrose (in particular the fructose component) is more harmful compared to other carbohydrates," added Dr. DiNicolantonio. Dr. DiNicolantonio and his co-authors, James H O'Keefe, MD, Saint Luke's Mid America Heart Institute, Kansas City, MO, and Sean C. Lucan, MD, MPH, MS, a family physician at Montefiore Medical Center, Albert Einstein College of Medicine, Bronx, NY, examined animal experiments and human studies to come to their conclusions.

Data from recent trials suggest that replacing glucose-only starch with fructose-containing table sugar (sucrose) results in significant adverse metabolic effects. Adverse effects are broader with increasing baseline insulin resistance and more profound with greater proportions of added fructose in the diet. The totality of the evidence is compelling to suggest that added sugar, and especially added fructose (usually in the form of high-fructose corn syrup and table sugar), are a serious and growing public health problem, according to the authors.

The 2010 Dietary Guidelines for Americans say it is acceptable for some people to consume up to 19% of calories from added sugars, and the Institute of Medicine permits up to 25% of total calories from added sugars. In contrast, the World Health Organization recommends that added sugars should make up no more than 10% of an entire day's caloric intake, with a proposal to lower this level to 5% or less for optimal health. Such levels would be more in line with what the authors would recommend and similarly restrictive to existing American Heart Association (AHA) recommendations - to consume no more than six teaspoons

(24 grams) of sugar per day for women and no more than nine teaspoons (36 grams) of sugar per day for men.

While fructose is found naturally in some whole foods like fruits and vegetables, consuming these foods poses no problem for human health. Indeed, consuming fruits and vegetables is likely protective against diabetes and broader cardiometabolic dysfunction, explained DiNicolantonio and colleagues. The authors propose that dietary guidelines should be modified to encourage individuals to replace processed foods, laden with added sugars and fructose, with whole foods like fruits and vegetables. "Most existing guidelines fall short of this mark at the potential cost of worsening rates of diabetes and related cardiovascular and other consequences," they wrote.

The authors also think there should be incentives for industry to add less sugars, especially fructose-containing varieties, to food-and-beverage products. And they conclude that at "an individual level, limiting consumption of foods and beverages that contain added sugars, particularly added fructose, may be one of the single most effective strategies for ensuring one's robust future health."

http://www.eurekalert.org/pub_releases/2015-01/mcow-pgl012815.php

Parkinson's gene linked to lung cancer

Researchers at the Medical College of Wisconsin (MCW), in collaboration with other colleagues of the Genetic Epidemiology of Lung Cancer Consortium (GELCC), have identified a gene that is associated with lung cancer.

The findings are published in American Journal of Human Genetics. Through whole exome sequencing, researchers identified a link between a mutation in PARK2, a gene associated with early-onset Parkinson's disease, and familial lung cancer.

The researchers sequenced the exomes (protein coding region of the genome) of individuals from a family with multiple cases of lung cancer. They then studied the PARK2 gene in additional families affected by lung cancer.

"While this specific mutation is very rare in the general population, there was a significant association between the PARK2 mutation we studied and the families with multiple cases of lung cancer," said Donghai Xiong, PhD, assistant professor of pharmacology and toxicology at MCW and the lead author on the paper.

"These results implicate this specific mutation as a genetic susceptibility factor for lung cancer, and provide an additional rationale for further investigations of this gene and this mutation for evaluation of the possibility of developing targeted therapies against lung cancer in individuals with PARK2 variants," added Ming You, MD, PhD, the Joseph F. Heil Jr. Professor of Oncogenesis at MCW and Director of the MCW Cancer Center.

http://www.eurekalert.org/pub_releases/2015-01/si-woi012915.php

Walking on ice takes more than brains

Salk scientists discover how a 'mini-brain' in the spinal cord aids in balance

LA JOLLA - Walking across an icy parking lot in winter - and remaining upright - takes intense concentration. But a new discovery suggests that much of the balancing act that our bodies perform when faced with such a task happens unconsciously, thanks to a cluster of neurons in our spinal cord that function as a "mini-brain" to integrate sensory information and make the necessary adjustments to our muscles so that we don't slip and fall.

In a paper published January 29, 2015 in the journal *Cell*, Salk Institute scientists map the neural circuitry of the spinal cord that processes the sense of light touch. This circuit allows the body to reflexively make small adjustments to foot position and balance using light touch sensors in the feet. The study, conducted in mice, provides the first detailed blueprint for a spinal circuit that serves as control center for integrating motor commands from the brain with sensory information from the limbs. A better understanding of these circuits should eventually aid in developing therapies for spinal cord injury and diseases that affect motor skills and balance, as well as the means to prevent falls for the elderly.

"When we stand and walk, touch sensors on the soles of our feet detect subtle changes in pressure and movement. These sensors send signals to our spinal cord and then to the brain," says Martyn Goulding, a Salk professor and senior author on the paper. "Our study opens what was essentially a black box, as up until now we didn't know how these signals are encoded or processed in the spinal cord. Moreover, it was unclear how this touch information was merged with other sensory information to control movement and posture."

While the brain's role in cerebral achievements such as philosophy, mathematics and art often take center stage, much of what the nervous system does is to use information gathered from our environment to guide our movements. Walking across that icy parking lot, for instance, engages a number of our senses to prevent us from falling. Our eyes tell us whether we're on shiny black ice or damp asphalt. Balance sensors in our ear canals keep our heads level with the ground. And sensors in our muscles and joints track the changing positions of our arms and legs.

Every millisecond, multiple streams of information, including signals from the light touch transmission pathway that Goulding's team has identified, flow into the brain. One way the brain handles this data is by preprocessing it in sensory way stations such as the eye or spinal cord. The eye, for instance, has a layer of neurons and light sensors at its back that performs visual calculations - a process known as "encoding" - before the information goes on to the visual centers in the

brain. In the case of touch, scientists have long thought that the neurological choreography of movement relies on data-crunching circuits in the spinal cord. But until now, it has been exceedingly difficult to precisely identify the types of neurons involved and chart how they are wired together.

In their study, the Salk scientists demystified this fine-tuned, sensory-motor control system. Using cutting-edge imaging techniques that rely on a reengineered rabies virus, they traced nerve fibers that carry signals from the touch sensors in the feet to their connections in the spinal cord. They found that these sensory fibers wire together in the spinal cord with another group of neurons known as ROR α neurons, named for a specific type of molecular gate found on each cell's nucleus. The ROR α neurons in turn connect with neurons in the motor region of brain, suggesting they might serve as a critical link between the brain and the feet. When Goulding's team disabled the ROR α neurons in the spinal cord using genetically modified mice developed at Salk, they found that these mice were substantially less sensitive to movement across the surface of the skin or to a sticky piece of tape placed on their feet. Despite this, the animals were still able to walk and stand normally on flat ground.

However, when the researchers had the animals walk across a narrow, elevated beam, a task that required more effort and skill, the animals struggled, performing more clumsily than animals with intact ROR α neurons. The scientists attribute this to the animals' reduced ability to sense when a foot was slipping off the edge and respond accordingly with small adjustments in foot position and balance - motor skills similar to those necessary for balancing on ice or other slippery surfaces.

Another important characteristic of the ROR α neurons is that they don't just receive signals from the brain and the light touch sensors, but also directly connect with neurons in the ventral spinal cord that control movement. Thus, they are at the center of a "mini-brain" in the spinal cord that integrates signals from the brain with sensory signals to make sure the limbs move correctly.

"We think these neurons are responsible for combining all of this information to tell the feet how to move," says Steeve Bourane, a postdoctoral researcher in Goulding's lab and first author on the new paper. "If you stand on a slippery surface for a long time, you'll notice your calf muscles get stiff, but you may not have noticed you were using them. Your body is on autopilot, constantly making subtle corrections while freeing you to attend to other higher-level tasks." The team's study represents the beginning of a new wave of research that promises to provide precise and comprehensive explanations for how the nervous system encodes and integrates sensory information to generate both conscious and unconscious movement.

"How the brain creates a sensory percept and turns it into an action is one of the central questions in neuroscience," adds Goulding. "Our work is offering a really robust view of neural pathways and processes that underlie the control of movement and how the body senses its environment. We're at the beginning of a real sea change in the field, which is tremendously exciting."

Other authors on the paper were Katja S. Grossmann, Olivier Britz, Antoine Dalet, Marta Garcia Del Barrio, Floor J. Stam, Lidia Garcia-Campmany and Stephanie Koch, all of the Salk Institute.

The research was funding by National Institutes of Health (Grants NS080586, NS086372 and NS072031), the Catharina Foundation, the Humboldt Foundation and Joan and Irwin Jacobs, through Salk's Innovation Grants Program.

http://www.eurekalert.org/pub_releases/2015-01/afps-tks012915.php

Transgender kids show consistent gender identity across measures *Gender identity of transgender children is deeply held and is not the result of confusion about gender identity*

A study with 32 transgender children, ages 5 to 12, indicates that the gender identity of these children is deeply held and is not the result of confusion about gender identity or pretense. The study, led by psychological scientist Kristina Olson of the University of Washington, is one of the first to explore gender identity in transgender children using implicit measures that operate outside conscious awareness and are, therefore, less susceptible to modification than self-report measures. The findings will be published in *Psychological Science*, a journal of the Association for Psychological Science.

Olson started the research project, partly out of her interest in how children think about social groups, but also because she'd witnessed the challenges of a close friend with a transgender child.

"Seeing how little scientific information there was, basically nothing for parents, was hard to watch," Olson said. "Doctors were saying, 'We just don't know,' so the parents have to make these really big decisions: Should I let my kid go to school as a girl, or should I make my kid go to school as a boy? Should my child be in therapy to try to change what she says she is, or should she be supported?"

The idea that young children, who haven't gone through puberty, can truly be transgender has met with public skepticism and some experts believe the best approach is to encourage "gender-variant" children to be comfortable with their biological gender. In recent years, however, more doctors, parents, and mental health professionals have begun to advocate for allowing children to live as their identified gender. Olson wanted to better understand gender identity in transgender children, taking a scientific approach to investigating whether their gender identity is deeply held, confused, or simply pretense, as some have argued.

Olson and co-authors Nicholas Eaton at Stony Brook University and Aidan Key of Gender Diversity, a Seattle organization that provides training and runs support groups for families of gender-nonconforming children, specifically focused their study on transgender children who were living as their identified gender in all aspects of their lives, who came from supportive home environments, and who had not yet reached puberty. The participants and their cisgender (non-transgender) siblings were recruited through support groups, conferences, and word of mouth.

Finally, the researchers recruited cisgender children from a database of families interested in participating in developmental psychology research studies. These cisgender children were age-matched to the transgender participants for analytical comparisons.

To get a comprehensive sense of the children's gender identity, Olson and colleagues used self-report measures that asked children to reflect on aspects of their gender in combination with implicit measures designed to gauge the strength of the children's more automatic gender associations.

For example, one of the implicit measures, based on the commonly used Implicit Association Test (IAT), assessed the speed with which they associated gender - male and female - with descriptors related to the concepts of "me" and "not me." The test is based on the theory that people are faster to respond to pairings that are more strongly associated in memory. The IAT has been used in many studies to investigate implicit attitudes related to various attributes, including gender and race, and brief versions of the IAT that use pictures instead of words have been validated for use with children.

Overall, data from the various measures indicated that transgender children's responses were indistinguishable from those of two groups of cisgender children. On the IAT measuring children's gender identity, transgender children showed a strong implicit identification with their expressed gender. When the researchers looked at the data according to the children's expressed gender, they saw that the data from transgender girls showed the same pattern as the data from cisgender girls and the data from transgender boys showed the same pattern as data from cisgender boys.

And Olson and colleague saw the exact same pattern of findings when they looked at data from an IAT test that tapped into the children's gender preferences. Transgender children also showed the same pattern of results as cisgender children on the explicit measures included in the study. For example, transgender girls, just like cisgender girls, preferred to be friends with other girls and they tended to prefer toys and foods that other girls liked.

"While future studies are always needed, our results support the notion that transgender children are not confused, delayed, showing gender-atypical responding, pretending, or oppositional - they instead show responses entirely typical and expected for children with their gender identity," the researchers write. "The data reported in this paper should serve as further evidence that transgender children do indeed exist and that this identity is a deeply held one," they conclude. Olson hopes to recruit up to 100 additional transgender children and follow them into adulthood to observe how the support they have received influences their development and whether it translates into more positive outcomes than in today's transgender adults, launching the first large-scale, nationwide, longitudinal study of transgender children in the United States.

"We have absolutely no idea what their lives will look like, because there are very few transgender adults today who lived as young kids expressing their gender identity," Olson said. "That's all the more reason why this particular generation is important to study. They're the pioneers."

<http://bit.ly/1CoDgic>

Portable mind-reader gives voice to locked-in people

Once only possible in an MRI scanner, vibrating pads and electrode caps could soon help locked-in people communicate on a day-to-day basis

29 January 2015 by Clare Wilson

YOU wake up in hospital unable to move, to speak, to twitch so much as an eyelid. You hear doctors telling your relatives you are in a vegetative state – unaware of everything around you – and you have no way of letting anyone know this is not the case. Years go by, until one day, you're connected to a machine that allows you to communicate through your brain waves. It only allows yes or no answers, but it makes all the difference – now you can tell your carers if you are thirsty, if you'd like to sit up, even which TV programmes you want to watch.

In recent years, breakthroughs in mind-reading technology have brought this story close to reality for a handful of people who may have a severe type of locked-in syndrome, previously diagnosed as being in a vegetative state. So far, most work has required a lab and a giant fMRI scanner. Now two teams are developing devices that are portable enough to be taken out to homes, to help people communicate on a day-to-day basis. The technology might also be able to identify people who have been misdiagnosed.

People with "classic" locked-in syndrome are fully conscious but completely paralysed apart from eye movements. Adrian Owen of Western University in London, Canada, fears that there is another form of the condition where the paralysis is total. He thinks that a proportion of people diagnosed as being in a vegetative state – in which people are thought to have no mental awareness at all

– are actually aware but unable to let anyone know. "The possibility is that we are missing people with some sort of complete locked-in syndrome," he says.

Owen's group and others are on a mission to give a voice to as many such people as possible. He is also asking ethicists how to respond if such people, once they can communicate, express a wish to die (see "[What if they want to die?](#)").

People most often enter a vegetative state after emerging from a coma. Instead of fully awakening, they enter a twilight zone between the two states. Their eyes may sometimes open, but their gaze wanders randomly and they do not respond to attempts to communicate, a key measure of consciousness. There is no official tally, but Derick Wade, a neurological rehabilitation consultant at Oxford University Hospitals has estimated that there are about 6000 people in the UK in a persistent vegetative state.

Owen's group has previously shown that a proportion of these people can in fact understand and follow instructions. The group made headlines in 2010 when they demonstrated this using an fMRI scanner, which shows brain activity. They asked people to imagine they were playing tennis or walking around their home. Not only did the scans show that about one in five of those tested could think about the different activities on cue, but three people so far have been able to use the different patterns of brain activity that these thoughts produced to answer simple yes or no questions.

One man tested, for instance, who had been classed as in a vegetative state for 12 years after a car crash, correctly answered questions about his name and those of his carer and a relative. He went on to signal that he was not in pain – and that he liked watching ice hockey on TV. "They were important questions for his family," says Owen. "It's about quality of life."

Brain scanning is a laborious process, though, so it is no good for helping people communicate frequently or easily. The size and cost of fMRIs mean that most care homes do not have them. To make the technology more accessible, Owen's team has been developing a version of the technique that uses an electrode cap to record the brain's electrical signals, or EEG. Because an EEG can only read surface brain activity, they had to find different mental tasks. Their first approach was to ask people to think about squeezing a hand or wiggling their toes.

The team showed in 2011 that three out of 16 people classed as being in a vegetative state could generate discernibly different patterns of brain activity in response to these commands. But Owen thinks this could still miss some people with awareness, as even people without brain injuries find the task difficult: one quarter of healthy volunteers he tested couldn't do it. "It's quite hard to imagine squeezing your hands," he says.

Now he has a new version, which involves placing vibrating devices on someone's arms, and asking them to pay attention to one vibrator or the other as they are asked questions, Owen told the Barts Neuroscience Symposium in London last week. Focusing on sensory information like vibration seems to be easier to read on an EEG than imagined movements, he says. "Tactile stimulation works very well." Still, it's early days and Owen's work is unpublished as yet. "We have had some successes," is all he will say for now.

Owen has reason for caution as his work is not without its critics. They worry that the publicity surrounding his work is giving false hope to families caring for people who are truly in a vegetative state. They also claim that the people with whom Owen has communicated had been misdiagnosed and were actually in a minimally conscious state (MCS). Such people can show fluctuating signs of awareness, such as being able to open their eyes on request. If this is true, then they don't need a mind-reader to communicate, just better diagnosis in the first place, to alert carers to ways they could communicate.

Yet Owen insists that his work shows that around 20 per cent of people classed as being in a vegetative state really have some kind of awareness – and that his latest version of the EEG "mind-reader" will help to find them. EEG communication devices already exist for people who have classic locked-in syndrome. They can look at letters as they flash on a screen and select them with a burst of mental activity, picked up by electrodes.

And a company in Austria called g.tec has developed an EEG device using vibrating pads for the wrists for people who have difficulty communicating. It went on sale in Europe at the end of last year. A group led by Steven Laureys of the University of Liège in Belgium has tested it on people with classic locked-in syndrome and found that asking users to count how many buzzes they feel on each wrist helps the electrodes pick up brain activity more clearly, and might make it possible for the device to enable people who are completely locked-in to communicate.

Despite the critics, Owen seems upbeat: "I think we may be able to send people home with some variation of an EEG. We will get there."

What if they want to die?

Adrian Owen has not used his methods of communicating with locked-in people (see main story) to ask someone if they want to end their life. "We don't have the [ethical] frameworks in place for dealing with that question," he says. He is, however, working with ethicists to draw up the first set of guidelines for such cases, which he hopes to have ready next year.

Going down this road would not be classed as euthanasia. People who are completely paralysed are kept alive by artificial means. This is classed as medical

treatment, and can be refused by anyone who is mentally competent. Treatment can already be withdrawn if a person's relatives think it is what they would have wanted.

There are well established ways of deciding if someone is of sound mind – but analysing brain activity is not currently conducive to them. "It's like trying to evaluate decision-making capacity in an abbreviated game of 20 questions," says ethicist [Andrew Peterson](#) of Western University in London, Canada, who is helping to write the guidelines.

Even with Owen's communication methods, there is likely to be some uncertainty. There will be cases where relatives disagree with the patient or with each other over whether treatment should continue. "This is not going to end up being a medical issue, this is going to end up a legal issue," says Owen.

<http://bit.ly/1CoDgic>

Why “Expensive” Medicines Might Actually Work Better *Perceived cost might influence drugs' benefits*

By Erin Blakemore

Do expensive drugs work better than cheap ones? Hold onto your wallet - new research suggests that how much you think a drug costs could impact how much you benefit from it...even if it's a placebo.

A team of Parkinson's researchers and neurologists were curious about how cost contributes to the perceived impacts of treatments. They told 12 Parkinson's patients that they would give them two different formulations of the same drug - one that cost \$100 per dose and one that cost a whopping \$1,500 per shot. After telling them whether they were being given the “cheap” or “expensive” drug, researchers injected the subjects with harmless saline solution instead. Once the drug “wore off,” they injected them with the other solution before subjecting them to a barrage of neurological tests.

The result was impressive indeed: patients who though they'd been given the “expensive” drug first showed a 28 percent improvement in motor skills. And after the researchers revealed that the drugs were actually placebos, the patients who confessed to expecting the “expensive” drug to do better ended up being the same ones who exhibited the biggest benefits.

Though the results could be skewed by the fact that the tests were performed on Parkinson's patients, who are known to release more dopamine in response to placebos, researchers think the results might apply to others, as well. “People who receive the shots thinking they received a drug may have an ‘expectation of reward’ response,” explains study lead Alberto J. Espay in a news release. That expectation can cause the brain to release dopamine in an amount that's similar to that generated by the reward itself, he notes.

Since the study relied on deception to get its results, the team had their plans reviewed and approved before they lied to patients about the placebo injections. But did patients really need to be deceived to feel the placebo effect? In a 2010 study, a team of researchers found that a group of patients who took pills clearly labeled “placebo” to treat irritable bowel syndrome were twice as likely to feel relief as those who didn’t receive treatment at all. Perhaps the mere act of being treated - or the specter of a big bill - is powerful in and of itself.

<http://nyti.ms/1z4myCb>

For Athletes, the Time of an Event Can Affect Performance

Athletes have long sought ways to gain even a small edge that can make the difference between getting a medal and finishing in the middle of the pack, like altitude training or even performance-enhancing drugs.

By Gina Kolata

Now British researchers are reporting that something completely legal and much less damaging to the body can dwarf the effects of drugs like EPO or testosterone. What really matters, they say, is whether the time of an event is in sync with an athlete’s body clock.

The most extreme example involves people who naturally go to bed late and wake up late. Even trying as hard as they can, they are as much as 26 percent slower when they sprint in the morning as in the evening. Individuals, like runners or cyclists, and people playing team sports, like soccer or football, would be affected. “Quite a remarkable finding,” said Carlyle Smith, a circadian rhythm expert and emeritus professor at Trent University in Canada who was not involved in the research.

The results, published Thursday in the journal *Current Biology*, diverge sharply from those of earlier studies that found that performance peaks in the evening. The lead researcher, Roland Brandstaetter of the University of Birmingham, said the previous research had measured athletes together - those who woke early, those who woke late, and those in between. When Dr. Brandstaetter lumped his athletes together he, too, found that, as a group, they performed best in the evening. It was only when he divided the athletes into groups according to their circadian rhythms that profound differences emerged.

The study was small - the researchers tested 20 competitive field hockey players and 22 competitive squash players six times a day.

The early risers tended to wake up, on average, around 7 a.m. on weekdays and 7:30 on weekends; intermediate risers got up about 8 on weekdays and 9:10 on weekends; and the late risers awoke about 9:30 on weekdays and 11 on weekends. The researchers evaluated their performances with measures involving sprinting

tests and, for the squash players, a test of concentration and alertness in which the athletes had to hit a ball into a small area.

The early risers had their peak performances at midday, the intermediate group did best in the afternoon and the late risers did best in the evening. Everyone did the worst at 7 a.m. Dr. Brandstaetter said some earlier studies had examined as many as 20 athletes while others had as few as six to eight.

Scientists not involved in the research said the findings make intuitive sense.

“Every athlete knows that there are times of day when they perform best,” said Dr. Benjamin D. Levine, the director of the Institute for Exercise and Environmental Medicine at Texas Health Presbyterian Hospital in Dallas.

But researchers also said that the large differences in performance that the study found needed to be replicated. Dr. Levine said future studies should also involve larger groups of elite athletes and more rigorous performance tests that accurately reflect each athlete’s chosen sports.

Kenneth P. Wright Jr., the director of the sleep and chronobiology lab at the University of Colorado, Boulder, said the findings seemed consistent with what is known about biological clocks. Researchers have long known that an individual’s natural circadian rhythm controls body temperature, heart rate, reaction time and concentration, so it might be expected that individual biological clocks would affect athletic performance.

The good news for athletes is that circadian clocks can be tweaked. Dr.

Brandstaetter says he deliberately alters his depending on what he plans to do, adjusting factors like light, activity and meal times. He normally does not get up early or late, but somewhere in between. But he makes himself an early riser for work and becomes a late riser when he is on vacation. He is now working with athletes, doing what he calls “circadian coaching.” The idea is to change the natural biological clocks of those who are naturally late risers when their sporting events - like marathons - start early in the day.

Of course, there is more to athletic performance than physiology, exercise researchers noted. “One of the biggest problems in athletic performance research is that we cannot replicate the highly motivated and competitive situations in the laboratory,” said Hirofumi Tanaka, an exercise researcher at the University of Texas in Austin.

Yet, he adds, “there is no question that circadian rhythms affect sports performance.” That is one reason athletes worry about jet lag, which can disrupt circadian rhythms “and become a performance killer.”

As for coaches and team owners, Dr. Smith said, “It would be handy to know the phenotype of all of your team members. You could predict who would be playing well at various times of day.” “Chronomoneyball,” he quipped.

http://www.eurekalert.org/pub_releases/2015-01/lsh-lhn013015.php

LSU Health New Orleans makes discovery key to preventing blindness and stroke devastation

Gene interactions that determine whether cells live or die in such conditions as age-related macular degeneration and ischemic stroke

New Orleans, LA - Research led by Nicolas Bazan, MD, PhD, Boyd Professor, Ernest C. and Yvette C. Villere Chair of Retinal Degeneration Research, and Director of the Neuroscience Center of Excellence at LSU Health New Orleans, has discovered gene interactions that determine whether cells live or die in such conditions as age-related macular degeneration and ischemic stroke. These common molecular mechanisms in vision and brain integrity can prevent blindness and also promote recovery from a stroke.

The paper is published online in *Cell Death & Differentiation*, a Nature journal at <http://www.nature.com/cdd/journal/vaop/ncurrent/full/cdd2014233a.html>.

"Studying the eye and the brain might hold the key to creating therapeutic solutions for blindness, stroke and other seemingly unrelated conditions associated with the central nervous system," notes Dr. Bazan. "The eye is a window to the brain."

Dr. Bazan and his research team discovered Neuroprotectin D1 (NPD1), which is made from the essential fatty acid, docosahexaenoic acid (DHA). Previous work showed that while it protected cells, the molecular principles underlying this protection were not known.

"During the last few years, my laboratory has been immersed in studying gene regulation," Dr. Bazan says. "We have uncovered a novel control that makes definitive decisions about whether a retina or brain cell will survive or die when threatened with disease onset. The gene mechanism that we discovered is the interplay of two genes turned on by the messenger Neuroprotectin D1."

Age-related macular degeneration (AMD) is a devastating disease that targets the retina of the elderly and destroys cells in charge of receiving photons and transferring light signals to the brain for decoding. The causal mechanisms of this disease remain elusive. The retinal pigment epithelium (RPE) is a single layer of cells that accomplishes multiple functions, such as providing survival molecules that prevent photoreceptors from dying.

The research team worked with human RPE cells and an experimental model of ischemic stroke. They discovered novel mechanisms in cells with the ability to activate pathways that crosstalk one to another and then assemble consolidated responses that decide cell fate. The researchers found that the powerful messenger, NPD1, is produced on-demand in the brain and retina and that it elicits a network

of positive signals essential for the well-being of vision and cognition. They showed that NPD1 bioactivity governs key gene interactions decisive in cell survival when threatened by disease or injury. They demonstrated that not only does NPD1 protect photoreceptors, but it also promotes remarkable neurological recovery from the most frequent form of stroke in humans.

In addition to Dr. Bazan, the LSU Health New Orleans Neuroscience Center research team included Drs. Jorgelina M. Calandria, Aram Asatryan, Veronica Balaszczuk, Eric Knott, Bok Kyoo Jun, Pranab K. Mukherjee and Ludmila Belayev.

This work was supported by National Institutes of Health (NIH) - grants R01 EY005121 (National Eye Institute) and P30 GM103340 (National Institute of General Medical Sciences) - and by the Eye Ear Nose and Throat Foundation of New Orleans, LA.

http://www.eurekalert.org/pub_releases/2015-01/uo-e-dch013015.php

DNA clock helps to get measure of people's lifespans

Scientists have identified a biological clock that provides vital clues about how long a person is likely to live.

Researchers studied chemical changes to DNA that take place over a lifetime, and can help them predict an individual's age. By comparing individuals' actual ages with their predicted biological clock age, scientists saw a pattern emerging.

People whose biological age was greater than their true age were more likely to die sooner than those whose biological and actual ages were the same.

Four independent studies tracked the lives of almost 5,000 older people for up to 14 years. Each person's biological age was measured from a blood sample at the outset, and participants were followed up throughout the study.

Researchers found that the link between having a faster-running biological clock and early death held true even after accounting for other factors such as smoking, diabetes and cardiovascular disease.

Scientists from the University of Edinburgh, in collaboration with researchers in Australia and the US, measured each person's biological age by studying a chemical modification to DNA, known as methylation.

The modification does not alter the DNA sequence, but plays an important role in biological processes and can influence how genes are turned off and on.

Methylation changes can affect many genes and occur throughout a person's life.

Dr Riccardo Marioni, of the University of Edinburgh's Centre for Cognitive Ageing and Cognitive Epidemiology, said: "The same results in four studies indicated a link between the biological clock and deaths from all causes. At present, it is not clear what lifestyle or genetic factors influence a person's biological age. We have several follow-up projects planned to investigate this in detail."

The study's principal investigator, Professor Ian Deary, also from the University of Edinburgh's Centre for Cognitive Ageing and Cognitive Epidemiology, said: "This new research increases our understanding of longevity and healthy ageing. It is exciting as it has identified a novel indicator of ageing, which improves the prediction of lifespan over and above the contribution of factors such as smoking, diabetes, and cardiovascular disease."

The study is published in the journal Genome Biology and was conducted by researchers from the University of Edinburgh, University of Queensland, Harvard University, University of California, Los Angeles (UCLA), Boston University, the Johns Hopkins University Lieber Institute for Brain Development and the U.S. National Heart, Lung and Blood Institute.

<http://bbc.in/1uOkeJG>

'Cold plasma' kills off norovirus

***Cold plasma consists of ionised gas molecules at room temperature
Norovirus, the most common cause of gastroenteritis in the world, can be killed
with "cold plasma," researchers in Germany have reported.***

By Alex Berezow Science writer

The virus, which elicits vomiting and diarrhoea, has gained international notoriety for causing outbreaks on cruise ships. However, such incidents represent merely a fraction of the tens of millions of cases that occur around the world each year. The research appears in mBio journal.

Preventing norovirus outbreaks is complicated by the fact that the virus is highly resistant to several different chemical disinfectants. Bleach, a chlorine-based solution, is currently the most effective treatment, but researchers are seeking more convenient alternatives. One such alternative is cold plasma, also known as non-thermal plasma. This "fourth state of matter" consists of ionised gas molecules at room temperature. These ions can destroy many kinds of microbes, but their effect on viruses was less clear.

Handheld gadget

A team of scientists led by Dr Birte Ahlfeld and Prof Günter Klein at the University of Veterinary Medicine in Hannover examined the effect of cold plasma on a strain of norovirus isolated from a human faecal sample taken during an outbreak at a military base in Germany. Cold plasma treatment led to a roughly 20- to 50-fold reduction in the number of virus particles. The viruses were destroyed because cold plasma consists of highly noxious ions, called reactive nitrogen and oxygen species, which exhibit potent antimicrobial activity. Moreover, the cold plasma generator, which produces the ions by applying an electric field to ambient air, could be designed as a handheld device. Alternatively, commonly contaminated surfaces, such as salad bars, could have cold plasma generators built into them. "A spread of norovirus can be inhibited at crucial

points, which as we know from our previous studies are all surfaces with frequent contact to human skin or hands," Dr Klein said. "Handheld devices can be used to disinfect different surfaces or a plasma box for hands or cutlery or plates is possible."

Growing problem

Other researchers shared Dr Klein's enthusiasm. Brendan Niemira and Dr David Kingsley, food safety experts at the US Department of Agriculture who were not involved with the research, said: "Cold plasma is a waterless technology, so there wouldn't be any solutions to apply or to rinse off. "That reduces water usage throughout the process, and might be more advantageous for continuous cleaning applications, such as for conveyor belts, materials handling surfaces, etc." A further advantage, they noted, is that storage of large volumes of sanitiser on site would no longer be necessary.

Mr Niemira and Dr Kingsley believe that treating surfaces with a combination of bleach and cold plasma may eventually become the gold standard of norovirus decontamination.

Over the decades, norovirus research has been greatly hindered by the inability to grow the virus in the laboratory. However, in late 2014, scientists at the University of Florida reported a breakthrough after they successfully cultured the virus in a complex in vitro system that utilised B-cells.

Besides decontaminating surfaces, cold plasma may have other medical applications. For instance, its use in treating dental caries has recently completed phase II clinical trials in the United States.

http://www.eurekalert.org/pub_releases/2015-01/bu-mmr013015.php

Meteorite may represent 'bulk background' of Mars' battered crust

NWA 7034, a meteorite found a few years ago in the Moroccan desert, is like no other rock ever found on Earth.

PROVIDENCE, R.I.-- It's been shown to be a 4.4 billion-year-old chunk of the Martian crust, and according to a new analysis, rocks just like it may cover vast swaths of Mars.

In a new paper, scientists report that spectroscopic measurements of the meteorite are a spot-on match with orbital measurements of the Martian dark plains, areas where the planet's coating of red dust is thin and the rocks beneath are exposed. The findings suggest that the meteorite, nicknamed Black Beauty, is representative of the "bulk background" of rocks on the Martian surface, says Kevin Cannon, a Brown University graduate student and lead author of the new paper.

The research, co-authored by Jack Mustard from Brown and Carl Agee from the University of New Mexico, is in press in the journal *Icarus*.

When scientists started analyzing Black Beauty in 2011, they knew they had something special. Its chemical makeup confirmed that it was a castaway from Mars, but it was unlike any Martian meteorite ever found. Before Black Beauty, all the Martian rocks found on Earth were classified as SNC meteorites (shergottites, nakhlites, or chassignites). They're mainly igneous rocks made of cooled volcanic material. But Black Beauty is a breccia, a mashup of different rock types welded together in a basaltic matrix. It contains sedimentary components that match the chemical makeup of rocks analyzed by the Mars rovers. Scientists concluded that it is a piece of Martian crust -- the first such sample to make it to Earth.

Cannon and Mustard thought Black Beauty might help to clear up a longstanding enigma: the spectral signal from SNC meteorites never quite match with remotely sensed spectra from the Martian surface. "Most samples from Mars are somewhat similar to spacecraft measurements," Mustard said, "but annoyingly different." So after acquiring a chip of Black Beauty from Agee, Cannon and Mustard used a variety of spectroscopic techniques to analyze it. The work included use of a hyperspectral imaging system developed by Headwall photonics, a Massachusetts-based company. The device enabled detailed spectral imaging of the entire sample.

"Other techniques give us measurements of a dime-sized spot," Cannon said.

"What we wanted to do was get an average for the entire sample. That overall measurement was what ended up matching the orbital data."

The researchers say the spectral match helps put a face on the dark plains, suggesting that the regions are dominated by brecciated rocks similar to Black Beauty. Because the dark plains are dust-poor regions, they're thought to be representative of what hides beneath the red dust on much of the rest of the planet. "This is showing that if you went to Mars and picked up a chunk of crust, you'd expect it to be heavily beat up, battered, broken apart and put back together," Cannon said.

That the surface of Mars would be rich in Black Beauty-like breccias makes a lot of sense, given what we know about Mars, the researchers say.

"Mars is punctured by over 400,000 impact craters greater than 1 km in diameter ...," they write. "Because brecciation is a natural consequence of impacts, it is expected that material similar to NWA 7034 has accumulated on Mars over time."

In other words, Mustard says, the bulk of rocks on the surface of Mars probably look a lot like Black Beauty: "dark, messy and beautiful."

<http://bit.ly/1z4hFnX>

Meet the Friendly Virus That Might Actually Be Good For You *Many people carry it, but it doesn't make you sick and could actually fight against viruses like HIV and Ebola*

By Marissa Fessenden

A virus called GB Virus-C has, apparently, infected more than a billion people alive today. But, fortunately, the cost of being infected with this virus is so low that researchers don't think it causes any illness. In fact, it might stave them off, [reports NPR's Richard Harris](#).

GBV-C infects white blood cells and dampens the body's immune response. "It's not severe — it's not enough that it makes people immune-suppressed," Jack Stapleton, an infectious disease specialist at the University of Iowa, told NPR, "but it does reduce the inflammatory response of immune cells." The virus can be transmitted sexually, through blood and from an infected mother.

All this resembles HIV, and, in fact, people infected with HIV are also likely to have GBV-C. But that might be a good thing. Some studies have shown that GBV-C [slows the progression of HIV infection](#).

Researchers don't know exactly how GBV-C could do that, but they suspect that the virus reduces inflammation and thus staves off AIDS. If that's the mechanism, it might also work in other viral diseases — say, Ebola. Though the number of new cases this month in the worst-affected countries [was the lowest since late June](#), the Red Cross [says](#) the virus is appearing in new regions and that West Africa may not be rid of it this year. Harris reports:

Hypothetically, this virus might also reduce inflammation in some people fighting off a roaring Ebola infection. "It's something you would predict," Stapleton says.

"Although often what you predict doesn't happen, so I wouldn't have predicted it." But if that's the case, perhaps drugs that act in a similar manner would help as well.

The idea isn't just theoretical. A [study last summer](#) that gathered plasma from Ebola patients in order to study the genetics of Ebola viruses also yielded some information about GBV-C. A pathologist, David O'Connor of the University of Wisconsin in Madison, found 13 samples from people who had both Ebola and GBV-C. Six of those people died, but seven survived. [Given that the death rate in this latest outbreak has been 70 percent](#), that's a notable outcome. The work is [published in the Journal of Virology](#).

It may be that the co-infection slowed Ebola's progression, just as it does HIV's, and gave the people a chance to fight off the deadly virus. But larger numbers would be needed to state that with any certainty. Still, while O'Connor is cautious about these results, he could see a future where it might be worth testing deliberate infection with GBV-C. "The thinking is," he told NPR, "this infects

hundreds of millions of people around the world today; we knowingly transmit it in blood transfusions. It's essentially a safe virus."

We think. Another study has found that the virus might be more common in people with [non-Hodgkin lymphoma](#), raising the possibility that GBV-C could be connected with some negative health effects. Again, that association isn't strong enough to say much for sure. But it is worth seeing if GBV-C is as good as it seems.

http://www.eurekalert.org/pub_releases/2015-02/uoo-fvr012915.php

Fewer viral relics may be due to a less bloody evolutionary history

Humans have fewer remnants of viral DNA in their genes compared to other mammals, a new study has found.

This decrease could be because of reduced exposure to blood-borne viruses as humans evolved to use tools rather than biting during violent conflict and the hunting of animals.

Despite natural defence systems, a retrovirus occasionally infects a mammal's egg or sperm, and the virus's genetic code gets incorporated into the animal's own genome. This viral 'fossil' then passes down from generation to generation: we all carry remnants of DNA from viruses that infected our ancestors millions of years ago. These 'endogenous retroviruses' (ERVs) appear not to cause us any harm, even though they are known to result in diseases such as cancer in other animals. A team of researchers from the University of Oxford and Plymouth University, UK, and the Aaron Diamond AIDS Research Center, USA, wondered if there was a combination of factors unique to humans that explained why these viral fossils in our genomes remain benign. They counted the number of times that retroviruses appear to have been integrated into an animal's genome in humans, comparing humans with 39 other mammalian species, including chimpanzees, dolphins and giant pandas.

Reporting their results in the journal *Retrovirology*, the researchers compared the genetic signature of the two edges of the virus. These edges are identical when the virus first invades the genome, but as they acquire random mutations over time, they slowly begin to diverge. By tracking this divergence, the research team could measure how long the retrovirus had spent in an animal's genome.

Using this measure, they found that, compared to other animals, far fewer retroviruses were incorporated into the genome for humans and other apes over the last 10 million years. Even compared to animals very similar to us, humans are unusual in not having acquired any new types of retroviruses into their DNA over the last 30 million years.

One reason for the reduction in retroviral incorporation into the human genome might be a change in behaviour as humans evolved: fewer bloody fights and less

exposure to infected meat meant that compared to other animals, our ancestors became less likely to encounter blood, a major route for viral infection.

'Considering us simply as a primate species, the proportion of human individuals that are infected with retroviruses is much less than among our relatives such as chimpanzees,' said Dr Robert Belshaw from Plymouth University.

However, lead researcher Dr Gkikas Magiorkinis from Oxford University's Department of Zoology said: 'We have shown in the past that Hepatitis C, a virus transmitted mainly through blood, was spread massively after World War II.

There is no doubt that the past trend of reduced blood contacts has been reversed in the last century, and this has severe consequences for viral infections.'

The work was supported by The Wellcome Trust and the Medical Research Council.

<http://bit.ly/18GDd4S>

Sugar Beets Make Hemoglobin

It's the latest veggie discovered to produce the protein best known for its role in blood

Jan 20, 2015 | By Amy Nordrum

Hemoglobin is best known as red blood cells' superstar protein—carrying oxygen and other gases on the erythrocytes as they zip throughout the bodies of nearly all vertebrates. Less well known is its presence in vegetables, including the sugar beet, in which Nélida Leiva-Eriksson recently discovered the protein while working on her doctoral thesis at Lund University in Sweden. In fact, many land plants—from barley to tomatoes—contain the protein, says Raúl Arredondo-Peter, an expert on the evolution of plant hemoglobins, or leghemoglobins, at the Autonomous University of the State of Morelos in Mexico. "Hemoglobins are very ancient proteins," he notes. Scientists first discovered them in the bright-red nodules of soybean roots in 1939 but have yet to determine the proteins' role in plants in most cases. One popular idea is that hemoglobin binds with and delivers nitric oxide to cells, sending signals to regulate growth.

Researchers are now investigating ways to leverage leghemoglobins. For example, Robert Hill, a plant biologist at the University of Manitoba, found that genetically engineering alfalfa to produce more of the proteins boosted the crop's survival rate during a flood from 20 to 80 percent. Plant hemoglobins might even serve as a blood substitute for humans someday—an idea that Arredondo-Peter says is conceivable but far off because they do not carry and release oxygen at the same rates as human hemoglobins. Or they could be exploited to trick our senses: food scientists at Stanford University are experimenting with plant hemoglobins as an ingredient in veggie burgers to make them taste more like bloody steaks.