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China will begin phasing out its ivory industry in an effort to fight poaching

This follows the country's one-year ban on ivory imports

By Lizzie Plaugic

For the first time ever, China has committed to phasing out its legal ivory industry, The Guardian reports. At an event this past week, workers destroyed 662kg of confiscated ivory in a symbolic gesture of the country's commitment to fighting African elephant poaching.

China hopes to eventually end the domestic manufacturing and sale of ivory in the country.

"We will strictly control ivory processing and trade until the commercial processing and sale of ivory and its products are eventually halted," Zhao Shucong, head of China's State Forestry Administration, reportedly said at the event.

This move follows China's decision earlier this year to impose a one-year ban on ivory imports in an effort to reduce illegal trading. Since 1989's international ivory trade ban, China has seized around 90,600 pounds of ivory, according to National Geographic.

At the event, Zhao outlined a 10-point plan to fight poaching, including stricter policing of wildlife trade online and offline, and running campaigns to discourage public demand, The Guardian reports.

A report this April found more than 500 instances of illegal ivory for sale online over a four-day period on Craigslist alone.

As much as 70 percent of the world's illegal ivory goes to China, where it is seen as a status symbol for a rising middle class, according to a report in The New York Times.

The demand for ivory in China is high, so part of phasing out the industry will have to include lowering consumer demand. A recent survey by the anti-trafficking group WildAid found 95 percent of respondents in China's three largest cities — Beijing, Shanghai, and Guangzhou — support an end to the country's ivory industry.

China hopes a reduction in the legal ivory market will also decrease black market demand.

But John Scanlon, secretary general of the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES), said that while China's decision is promising, the black market still remains a big driver of elephant poaching, which he called "one of the most destructive forms of wildlife crime."

A timeline for the phase-out has not yet been set.

http://www.eurekalert.org/pub_releases/2015-05/jhm-idi052815.php

Immunotherapy drug improves survival for common form of lung cancer

People with squamous-non-small cell lung cancer receiving nivolumab lived months longer than those receiving chemotherapy

In a head-to-head clinical trial comparing standard chemotherapy with the immunotherapy drug nivolumab, researchers found that people with squamous-non-small cell lung cancer who received nivolumab lived, on average, 3.2 months longer than those receiving chemotherapy. Squamous non-small cell lung cancer accounts for 25 to 30 percent of all lung malignancies.

Results of the trial, reported in the May 31 issue of the New England Journal of Medicine and presented at the American Society for Clinical Oncology 2015 annual meeting, also showed that after a year, the nivolumab group had nearly double the survival rate (42 percent) of the chemotherapy patients (24 percent).

"This solidifies immunotherapy as a treatment option in lung cancer," says Julie Brahmer, M.D., director of the Thoracic Oncology Program at the Johns Hopkins Kimmel Cancer Center.

"In the 20 years that I've been in practice, I consider this a major milestone," she adds, noting that the trial results helped achieve U.S. Food and Drug Administration approval in March to treat such patients whose lung cancer progressed, despite standard chemotherapy.

Brahmer emphasizes that the relatively small increase in median survival time with the use of the new immunotherapy drugs may be somewhat misleading in terms of overall impact of the medicines. "Patients who respond to immunotherapy tend to continue their responses for long durations, and these lengthier responses are cut off in calculations of median overall survival," she says. She suggests that one- and two-year survival data may provide more information about the effectiveness of these drugs than overall median survival rates.

Promising results of an earlier, initial, multicenter clinical trial of nivolumab, first reported in 2013 and directed by Brahmer, led to the current phase III trial of 260 patients treated at hospitals across the world.

Nivolumab is one of a group of so-called "checkpoint inhibitors" that work by disrupting a signaling system used by cancers to avoid detection and destruction by immune cells.

The system, says Brahmer, provides a kind of "handshake" or connection between receptors on immune cells, called PD-1, and their sister-proteins on tumor cells,

called PD-L1. Checkpoint inhibitors block that handshake, which alerts immune cells to cancer cells and target them for destruction.

For the new trial, hospitals enrolled patients with advanced, squamous non-small cell lung cancer whose disease had progressed despite initial chemotherapy. Researchers randomly selected 135 patients to receive nivolumab, sold under the name Opdivo, and 137 others to receive the chemotherapy drug docetaxel. Both drugs are delivered intravenously.

Median overall survival of patients receiving nivolumab was 9.2 months, compared with six months for patients who received docetaxel. At one year, 57 patients (42 percent) taking nivolumab were alive, compared with 33 patients (24 percent) taking docetaxel. Approximately 27 patients (20 percent) receiving nivolumab responded to the drug, compared with 12 (8.8 percent) who took docetaxel. The median disease-progression free survival was 3.5 months for those who took nivolumab and 2.8 months for docetaxel.

The researchers also reported that nivolumab reduced the relative risk of dying from lung cancer by 41 percent in those who took the immunotherapy drug, compared with those who took docetaxel.

Furthermore, they said the most severe side effects occurred more often in patients taking docetaxel (55 percent) than those taking nivolumab (6.9 percent). Patients on nivolumab experienced fatigue, decreased appetite, weakness, as well as colon, kidney or lung inflammation. Those taking docetaxel experienced hair loss, fatigue, nausea, diarrhea and low white blood cell counts, which decreases patients' ability to fight infections. "Immunotherapy can produce severe side effects, and it's important to be vigilant in efforts to manage them. However, it is less toxic than chemotherapy," says Brahmer.

She adds, "Generally, about 20 to 25 percent of patients with lung cancer are responding to checkpoint blockade inhibitors."

Because immunotherapy drugs tend to be expensive, costing more than $100,000 per year, per patient, Brahmer says there is even more urgency now to find out which patients are more likely to benefit. This includes determining whether the drugs should be used earlier on in cancer treatment, finding biomarkers to predict response, and combining immunotherapy with other treatments.

Funding for the study was provided by Bristol-Myers Squibb. Brahmer is an uncompensated adviser to Bristol-Myers Squibb. The terms of these arrangements are being managed by The Johns Hopkins University.

Scientists who contributed to the research include Karen Reckamp, City of Hope Comprehensive Cancer Center; Paul Baas, the Netherlands Cancer Institute; Lucio Crino, University Hospital of Perugia, Italy; Wilfried Ernst Eric Eberhardt, University Hospital Essen, Germany; Elena Poddubskaya, N.N. Blokhin Russian Cancer Research Center; Scott Antonia, the H. Lee Moffitt Cancer Center; Adam Pluzanski, the Centrum Onkologii-Instytut Im. Marii Sklodowskiej-Curie, Warsaw, Poland; Everett Vokes, University of Chicago; Esther Holdago, Hospital de Madrid in Spain; David Waterhouse, Oncology Hematology Care; Neal Ready, Duke University Medical Center; Justin Gainor, Massachusetts General Hospital; Osvaldo Aren Frontera, Centro Internacional de Estudios Clinicos in Santiago, Chile; Libor Havel, Nemocnice Na Bulovce, Czech Republic; Martin Steins, University Hospital Heidelberg, Germany; Marina Chaira Garassino, Fondazione IRCCS Instituto Nazionale Dei Tumori, Milan, Italy; Joachim Aerts, Erasmus MC Cancer Institute, the Netherlands; Manuel Domine, the Fundacion Jimenez Diaz, Spain; Luis Paz-Ares, Hospital Universitario Virgen Del Rocio, Seville, Spain; Martin Reck, the Lungen Clinic Grosshansdorf, Germany; Christine Baudelet, Christopher Harbison and Brian Lestini, Bristol-Myers Squibb; and David Spigel, Sarah Cannon Research Institute, Nashville, Tennessee.

http://www.eurekalert.org/pub_releases/2015-06/wsu-wrs052915.php

WSU researchers see link between hunter-gatherer cannabis use, fewer parasites

Study suggests unconscious use of 'medical marijuana'

VANCOUVER, Wash.--Washington State University researchers have found that the more hunter-gatherers smoke cannabis, the less they are infected by intestinal worms. The link suggests that they may unconsciously be, in effect, smoking medical marijuana.

Ed Hagen, a WSU Vancouver anthropologist, explored cannabis use among the Aka foragers to see if people away from the cultural and media influences of Western civilization might use plant toxins medicinally.

"In the same way we have a taste for salt, we might have a taste for psychoactive plant toxins, because these things kill parasites," he said.

In an earlier study, Hagen found that the heavier tobacco smokers among the Aka also had fewer helminths, parasitic intestinal worms.

He cautions, however, that the studies have their limits. While nicotine has been seen killing worms in livestock, that hasn't been directly demonstrated in humans. Cannabis kills worms in a petri dish, but researchers have not shown it killing worms in animals, Hagen said.

The Aka are a "pygmy" people of the Congo basin. As one of the world's last groups of hunter-gatherers, they offer anthropologists a window into a way of life accounting for some 99 percent of human history. They might also offer an alternative hypothesis to explain human drug use.

The prevailing explanation is that recreational drugs "hijack the pleasure centers of the brain," making people feel good. But they also trigger mechanisms that tell us we're consuming something toxic, tasting bitter and making us feel sick.

"So we thought, 'Why would so many people around the world be using plant toxins in this very 'recreational' way?" said Hagen. "If you look at non-human

animals, they do the same thing, and what a lot of biologists think is they're doing it to kill parasites."

The issue is significant on at least two fronts, write Hagen and his colleagues, with substance abuse and intestinal helminth infection being "two of the developing world's great health problems." Their study appears in the American Journal of Human Biology.

Researchers are unsure when the Aka might have first smoked cannabis or when it arrived on the continent. It may have come with traders from the Indian subcontinent around the first century A.D., but Hagen and his colleagues say it might not have been smoked until European colonization in the 17th Century.

Hagen surveyed almost all of the nearly 400 adult Aka along the Lobaye River in the Central African Republic and found roughly 70 percent of the men and 6 percent of the women used cannabis. The polling was supported by bioassays of the men that found high enough levels of THCA, a metabolic byproduct of cannabis's active ingredient, to indicate that 68 percent of them had recently smoked.

Stool samples collected from the men to gauge their worm burden found some 95 percent of them were infected with helminths. But those who consumed cannabis had a significantly lower rate of infection. A year after being treated with a commercial antihelmintic, the cannabis users were reinfected with fewer worms.

While the Aka deliberately consume a tea of a local plant, motunga, to fight parasitic infections, they do not think of cannabis or tobacco as medicine, Hagen said. This suggests they are unconsciously using cannabis to ward off parasites, he said.

Hagen's co-authors on the study are Casey Roulette, who did the research as part of his WSU PhD, and Pasteur Institute researchers Mirdad Kazanji and Sébastien Breurec.

http://www.eurekalert.org/pub_releases/2015-06/uos-pit052815.php

Patient information too high for patients' literacy: New research

Over 90 per cent of educational materials written for kidney disease patients is higher than an average patient's literacy

More than 90 per cent of educational materials written for kidney disease patients is higher than an average patient's literacy, according to a new study published in the June issue of the National Kidney Foundation's American Journal of Kidney Diseases.

"Our study suggests most patient information materials are not fit for their intended purpose, and that organisations are producing materials that may be too difficult for their intended audience to understand," said Angela Webster, lead researcher and an Associate Professor Clinical Epidemiology at the University of Sydney.

The average adult patient has an 8th grade literacy level but over 20 per cent of patients read at or below a 5th grade level. Of patients over the age of 65, 40 per cent read at or below a 5th grad level.

In the study, researchers looked at 80 English-language educational materials that were designed to be printed and read by patients in the United States, Australia and the United Kingdom.

These free educational materials were analyzed using both the Lexile Analyzer and the Flesch-Kincaid Grade Level formula.

Analysis suggested that most materials required a minimum of a 9th grade health literacy level. Only 5 per cent of materials were pitched at the recommended 5th grade level.

"These findings suggested that patient information materials aimed at patients with chronic kidney disease are pitched well above the average patient's literacy level, so that most patients wouldn't be able to read and understand the health messages," Webster said.

Providing patients with reading materials outside their level of understanding could make it difficult to follow medication directives, dietary restrictions, and necessary lifestyle modifications for disease management.

Poor health literacy is a particular problem for elderly, ethnic minority, and socially disadvantaged people, all of whom are more likely to have chronic kidney disease.

People with low health literacy are less likely to feel engaged with their healthcare providers, and are less likely to participate in their treatment decisions and have significantly higher mortality and morbidity rates.

Materials that are written above a patient's health literacy level can contribute to poor management and outcomes.

"Developing patient education materials that are appropriate for all literacy levels is a challenge, but a very important challenge for improving health outcomes.

All organisations need to make a thorough assessment on the readability of their patient information materials," said Thomas Manley, Director of Scientific Activities for the National Kidney Foundation.

"Conducting formal readability testing, as suggested by the study authors, along with use of patient reviewers from a variety of educational and cultural backgrounds may provide important feedback to enhance the value of materials across a larger spectrum of health literacy levels."

http://nyti.ms/1Q3en3a

Medicine's Hidden Roots in an Ancient Manuscript

The first time Grigory Kessel held the ancient manuscript, its animal-hide pages more than 1,000 years old, it seemed oddly familiar.

By MARK SCHROPE JUNE 1, 2015

Credit Courtesy of the Owner

A Syriac scholar at Philipps University in Marburg, Germany, Dr. Kessel was sitting in the library of the manuscript's owner, a wealthy collector of rare scientific material in Baltimore. At that moment, Dr. Kessel realized that just three weeks earlier, in a library at Harvard University, he had seen a single orphaned page that was too similar to these pages to be coincidence.

The manuscript he held contained a hidden translation of an ancient, influential medical text by Galen of Pergamon, a Greco-Roman physician and philosopher who died in 200 A.D. It was missing pages and Dr. Kessel was suddenly convinced one of them was in Boston.

Dr. Kessel's realization in February 2013 marked the beginning of a global hunt for the other lost leaves, a search that culminated in May with the digitization of the final rediscovered page in Paris.

Scholars are just beginning to pore over the text, the oldest known copy of Galen's "On the Mixtures and Powers of Simple Drugs." It may well provide new insights into medicine's roots and into the spread of this new science across the ancient world. "On so many levels it's important," said Peter Pormann, a Graeco-Arabic expert at the University of Manchester who now leads a study of the text.

The manuscript held by Dr. Kessel that day was a palimpsest: older text covered up by newer writing. It was a common practice centuries ago, a medieval form of recycling. In this case, 11th-century Syrian scribes had scraped away Galen's medical text and had overwritten hymns on the parchment.

The hymn book itself is of interest, but for now it is the original text, all but invisible to the naked eye and known as the undertext, that has captured the imagination of scholars.

For centuries, Galen's "Simple Drugs" was required reading for aspiring physicians, the summation of ancient knowledge about medicine, patient care and pharmaceutical plants. Galen described a root that cures "roughness of the throat" and recommended hemp as an earache remedy that "does not produce flatulence" (though it "dries out the semen").

Much of "Simple Drugs" was eventually translated into Syriac, a form of Aramaic used by Middle Eastern Christian communities. The undertext of the manuscript in Baltimore, most likely from the ninth century A.D., is a copy of the first Syriac translation, itself painstakingly completed in the sixth century A.D. by Sergius of Reshaina, a Syriac physician and priest. "Today, it doesn't look to be special when somebody translates one language to another, but in those days, it was indeed a great achievement," Dr. Kessel said. "He had to create vocabulary, to find Syriac words to correspond to this Greek medical vocabulary."

One leaf of the Syriac Galen Palimpsest remains at St. Catherine's in the Sinai Desert in Egypt, which has the world's oldest continuously operating library. **Mark Schrope**

By the sixth century, Syriac-speaking Christians were spreading east from Turkey through Syria, Iraq and Iran. They needed translations of Greek scholarly work, partly to support missionary work like running hospitals.

"Simple Drugs" was a large work, an 11-book treatise. Sergius's translations of Galen's text were copied and recopied for centuries, and eventually became a bridge for moving the medical expertise of the ancient Greeks to Islamic societies. Syriac texts were much easier than Greek ones to translate into Arabic.

As Muslim influence grew in the Near and Middle East, Christian populations dwindled, and so did Syriac. "These great Christian cultures that used it suffered so much," said Columba Stewart, the executive director of the Hill Museum and Manuscript Library in Collegeville, Minn.

"By the time you have modern scholarship, these ancient Syriac cultures are just a vestige of their former selves — often quite isolated from Western culture, so there's not a lot of awareness."

Revealing Reading

Little is known of the history of the manuscript in Baltimore, formally known as the Syriac Galen Palimpsest, from its recycling in the 11th century until the 1920s, when it was sold to a private collector in Germany. After that, the manuscript fell again from public view until 2002, when it was purchased by a collector in a private sale. He has not been publicly identified.

In 2009, the Galen Palimpsest was lent to the Walters Art Museum for spectral imaging of its leaves by an independent group of specialists, which would reveal

the erased Galen undertext. Each page is photographed digitally at extremely high resolution with varying colors and configurations of light, which in various ways illuminate the inks, grooves from writing and parchment itself. Computer algorithms exploit these variations to maximize the visibility of the undertext.

The resulting images went online under a "creative commons" license, meaning that anyone can use the material free for any noncommercial purpose. Once the images were online, William Noel, who was the curator of manuscripts and rare books at the museum, began organizing members of the tiny community of scholars who study Syriac scientific texts to study the new material.

One of them was Dr. Kessel, who was on a fellowship at Harvard's Dumbarton Oaks Research Library in Washington. Eventually, Michael B. Toth, a systems engineer who managed the imaging work arranged for him to see the Galen Palimpsest for himself.

"I couldn't even imagine how it looked," Dr. Kessel said. "Then when I saw the manuscript, I had the kind of déjà vu impression that I had already seen it. And then I recalled the single folio in the Harvard library."

Filling the Gaps

By analyzing the page size, handwriting and other features, as well as the visible text, Dr. Kessel was able to determine that the Harvard leaf did indeed fill one of the gaps in the Galen Palimpsest. But six more were apparently missing. Dr. Kessel set out to find them. He began with a list of 10 libraries known to have ancient Syriac material, combing through online catalogs when available to look for clues such as the right dimensions or vague references to undertext. Sometimes, he traveled to the libraries himself.

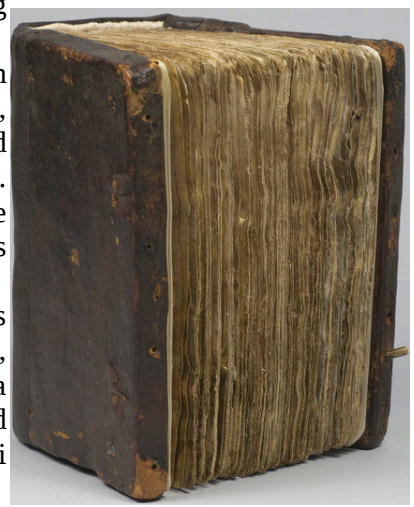
It was not long before Dr. Kessel had good news. He found one missing page in a catalog from the Sacred and Imperial Monastery of the God-Trodden Mount of Sinai. It is known more commonly as St. Catherine's in the Sinai Desert in Egypt, which has the world's oldest continuously operating library.

Another leaf turned up at the National Library of France in Paris. And at the Vatican's vast library in Rome, he The seventh missing page is believed to have been blank and was probably discarded.

An Intriguing Link

No one knew how much of "Simple Drugs" might be hidden in the Galen Palimpsest. The only other known Syriac copy resides at the British Library in London and includes only Books 6 to 8. Translations of these later books in the series are the most common, because they contain more specific medicinal information and details about the properties of plants.

But as their preliminary studies progressed, Dr. Kessel and his colleagues spotted words from Books 2 and 4 in one of the loose leaves. The full text of "Simple Drugs" is known to scholars, but only from more recent translations in languages other than Syriac. "This was something absolutely unexpected," he said.



The bound Syriac Galen Palimpsest. **Credit Courtesy of the Owner**

Siam Bhayro, a specialist in early Jewish studies at the University of Exeter in England, had believed that Sergius must have translated the earlier books, but there had been no proof. When he heard that Dr. Kessel might have found pages from the early translations, "I was almost dancing up and down," he said.

Another of Dr. Kessel's intriguing discoveries was a note in Arabic on the first leaf, indicating that the manuscript — by then a hymn book concealing Galen's text — had been donated to the brothers of the Sinai monastery, a reference to St. Catherine's.

How it left the monastery is unclear: Particularly in the early 20th century, some of the library's holdings were borrowed legitimately, while others were stolen by visitors hoping to make private sales.

The independent imaging team is now finishing the work necessary to add the rediscovered leaves to the digital collection. But translating and studying the Syriac text revealed in the images will take much longer, perhaps five years or more. That work is now underway because of a recent $1.5 million grant from the United Kingdom's Arts and Humanities Research Council.

Scholars are eager to compare the Syriac material to existing copies of "Simple Drugs" written in Greek, all of which appear to be centuries younger than the Galen Palimpsest and much further removed from the original.

As texts went through multiple rounds of copying, they underwent significant changes. A scribe might remove parts that seemed unimportant or add material based on new knowledge. Comparing the Galen Palimpsest and the British Syriac copy, for instance, could offer telling insights into how the ancient Greeks treated the ill and how these remedies spread across the Middle East.

"Some of the stuff is not entirely scientific by our standards," even if it enabled progress, Dr. Petit said. Indeed, little of Galen's advice would stand up to modern scrutiny. Like other ancient physicians, he believed health was controlled by the balance of four "humors" in the body and recommended certain stones for their cleansing powers.

"The Galenic system is completely bonkers," Dr. Bhayro said.

Still, it was the best thinking available in an era in which the very idea of medical science was relatively new. “It’s likely to be a central text once it’s fully deciphered,” said Dr. Pormann of the University of Manchester. “We might discover things we really can’t dream of yet.”

http://www.eurekalert.org/pub_releases/2015-06/p-ppp052615.php

PharmaMar's PM1183 plus doxorubicin shows remarkable activity in small cell lung cancer

Treatment induced objective responses in 67% of patients, including 10% of them where all signs of cancer disappeared

Chicago and Madrid - PharmaMar today announced data from a Phase 1b study of the transcriptional inhibitor PM1183 in combination with doxorubicin in second line therapy in patients with small cell lung cancer (SCLC) showing that the treatment induced objective responses in 67% of the patients, including 10% of them where all signs of cancer disappeared (complete responses). Every patient with SCLC denominated primary chemotherapy-sensitive (their chemotherapy-free interval (CTFI) is more than 90 days) responded to treatment, including 18% of complete responses. In primary chemotherapy-resistant patients, where cancer was progressing within 90 days or less of previous chemotherapy, a remarkable 30% achieved a response. Notably, the treatment resulted in durable responses, with an overall progression-free survival (PFS) of 4.6 months, which was 3.6 months in resistant patients. The most common adverse drug reaction was reversible myelosuppresion but no cardiotoxicity or drug-related deaths were observed.

"The rate, depth and length of responses that we have observed with this treatment in the second-line setting are remarkable, even in those patients that are usually considered harder to treat", said Dr. Martin Forster, University College Hospital, London, UK. "Small cell lung cancer is an unmet clinical need with very few recent advances and the scientific community is committed to help new develop effective therapies."

The lead author Dr. Martin Forster, University College Hospital, London, UK. will present the full data today at the 51st Annual Meeting of the American Society of Clinical Oncology (ASCO) (Abstract#7509, Monday, June 1 from 8:00 AM to 11:30 AM at S Hall A Poster Board 256). This study will be further discussed later today at a Poster Discussion Session on Targeted Therapies In Unselected Patients from 1:15 PM - 2:30 PM at E Hall D2

"No therapies have been approved in the last 17 years for small cell lung cancer, so we are very excited about the results obtained with PM1183 in these patients. The novel mechanism of action and lack of platinum cross-resistance of PM1183 are an advantage for treating these tumors." pointed out Arturo Soto, Director of Clinical Development at PharmaMar.

About the Phase 1b study with PM1183 and doxorubicin

PM1183 is an inhibitor of transcription by specifically targeting the enzyme RNA polymerase II (in its active state) for degradation , thereby blocking the expression of certain genes important for tumor progression. This targeting of the transcriptional machinery is also coupled to a DNA repair pathway called nucleotide excision repair (NER), which is important to repair DNA breaks. A recent preclinical study has shown that SCLC may be particularly sensitive to transcription inhibitors , and PM1183 plus doxorubicin demonstrated a synergistic and robust anticancer effect in SCLC mouse models .

This Phase 1b study is an expansion cohort of approximately 20 evaluable SCLC patients that have failed after one chemotherapy-containing prior line to assess in second line treatment the remarkable activity of the combination treatment (71% of objective partial responses) previously observed during the escalation phase.

After 12 months of follow up, the overall response rate as measured by RECIST criteria was 67% and a complete response was achieved by 10% of the patients. Durable responses were observed with an overall PFS of 4.6 months (4.8 months in sensitive patients and 3.6 months in resistant patients).

CTFI was the only variable with statistically significant (p=0.001) correlation with response - all sensitive patients responded (95%CI: 71-100%) and a remarkable 30% of resistant patients also showed a response.

The response rate observed with the combination of PM1183 and DOX in second line is comparable to those observed with first line chemotherapy treatments in this same population.

Reversible myelosuppression was the most frequent adverse drug reaction observed. There were no unexpected or drug-related deaths. DOX dose may be adapted, with or without CSF prophylaxis, to reduce associated myelosuppression.

About small cell lung cancer

SCLC is a very aggressive cancer that usually presents with distant metastases and has already spread at the time of diagnosis, thus limiting the role of traditional approaches and posing a worse prognosis compared to other lung cancer types. The 5-year survival rate is about 5% . About 18% of all the lung cancer cases diagnosed are SCLC, and only in the US more than 34,000 new cases are recorded every year. This tumor is strongly associated with tobacco smoking, posing an important public health problem . After failure to treatment with a platinum-based therapy in first line, there are almost no therapeutic alternatives, and the approval of the last drug for this disease took place a few decades ago.

About PM1183 (lurbinectedin)

PM1183 is an investigational drug from the class of inhibitors of the enzyme RNA polymerase II, which is crucially involved in transcription. By targeting transcription, the drug inhibits the expression of factors important for tumor progression, and impairs the DNA repair system called NER, thereby enhancing tumor cell killing. PM1183 (lurbinectedin) is currently being investigated in different tumor types, including a Phase 3 study for platinum-resistant ovarian cancer, a Phase 2 study for BRCA1/2-associated metastatic breast cancer and a Phase 1b study for SCLC.

http://www.bbc.com/news/health-32958504

Have we cured cancer?

Have we cured cancer?

By James Gallagher Health editor, BBC News website

You will have been left with that impression if you walked past a newspaper stand this morning. The short answer, if you're in a hurry, is no.

But something truly exciting is happening - the field of immunotherapy is coming of age. It will not be a universal "cure" but immunotherapy is fast becoming a powerful new weapon alongside chemotherapy, radiotherapy and surgery.

Defender

Your immune system is your body's internal guardian and protector as it purges anything that is not "you". It has a series of checks and brakes in the system that prevent the immune system turning on healthy tissue (this is what goes wrong in autoimmune diseases like multiple sclerosis). But cancer is a corrupted version of healthy tissue and can masquerade as normal to dodge our immune defences.

It performs the chemical equivalent of shouting "move along, nothing to see here". And it does this by producing proteins on its surface that perform a "chemical handshake" with immune system cells to switch them off.

The immunotherapy drugs that have got people excited are like an oven-mitt that covers one of the hands, preventing the handshake. The field has been developing for some time, but the explosion of front page newspaper headlines was triggered by data presented at the American Society of Clinical Oncology (ASCO).

UK-led research showed that 60% of advanced melanoma skin cancers shrank when two immunotherapies were given in combination. The dual treatment stopped some of these deadliest cancers progressing for nearly a year.

Significant advance

The ASCO announcement came two days after another immunotherapy trial showed some lung cancer patients had their life expectancy doubled by immunotherapy drugs. Smaller trials in a wide range of other cancers have also been presented - suggesting immunotherapy will have a role in many tumour-types. Exciting? Certainly. A cure? No.

As Prof Karol Sikora, the dean of the University of Buckingham's medical school, told the BBC: "You would think cancer was being cured tomorrow. "It's not the case, we've got a lot to learn." So what are the words of caution?

For starters, these drugs do not work equally in everyone. Some people do spectacularly well, some do ok, and some do not respond at all.

The reason why is still unclear. Are cancers susceptible during just a short window in their development? Is it down to the type or quantity of proteins the tumours produce on their surface? We don't yet know. Also, the therapies are likely to be very expensive, which means targeting the drugs on those who will respond will be key.

Long-term side effects are another a big uncertainty. Will the change to the immune system increase the risk of autoimmune diseases? So far the side effects seem to appear only during treatment, but long-term follow of patients who do respond has not taken place. The research outside of melanoma and lung cancer is also still at a very early stage.

This is not a sudden breakthrough, or even the first set of really promising immunotherapy data. The melanoma trial used a combination of two drugs - ipilimumab and nivolumab. Ipilimumab is already recommended as the primary treatment for advanced melanoma in the UK.

So what we are seeing is a series of advances in a field that holds huge promise for the future. That's exciting without throwing in the "cure" word.

http://www.eurekalert.org/pub_releases/2015-06/uoc--psl052915.php

Poor sleep linked to toxic buildup of Alzheimer's protein, memory loss

Berkeley neuroscientists connect a deficit of restorative slumber to an accumulation of beta-amyloid

Sleep may be a missing piece in the Alzheimer's disease puzzle.

Scientists at the University of California, Berkeley, have found compelling evidence that poor sleep - particularly a deficit of the deep, restorative slumber needed to hit the save button on memories - is a channel through which the beta-amyloid protein believed to trigger Alzheimer's disease attacks the brain's long-term memory.

"Our findings reveal a new pathway through which Alzheimer's disease may cause memory decline later in life," said UC Berkeley neuroscience professor Matthew Walker, senior author of the study to be published Monday, June , in the journal Nature Neuroscience.

Excessive deposits of beta-amyloid are key suspects in the pathology of Alzheimer's disease, a virulent form of dementia caused by the gradual death of brain cells. An unprecedented wave of aging baby boomers is expected to make Alzheimer's disease, which has been diagnosed in more than 40 million people, one of the world's fastest-growing and most debilitating public health concerns.

The good news about the findings, Walker said, is that poor sleep is potentially treatable and can be enhanced through exercise, behavioral therapy and even electrical stimulation that amplifies brain waves during sleep, a technology that has been used successfully in young adults to increase their overnight memory.

"This discovery offers hope," he said. "Sleep could be a novel therapeutic target for fighting back against memory impairment in older adults and even those with dementia."

The study was co-led by UC Berkeley neuroscientists Bryce Mander and William Jagust, a leading expert on Alzheimer's disease. The team has received a major National Institutes of Health grant to conduct a longitudinal study to test their hypothesis that sleep is an early warning sign or biomarker of Alzheimer's disease. While most research in this area has depended on animal subjects, this latest study has the advantage of human subjects recruited by Jagust, a professor with joint appointments at UC Berkeley's Helen Wills Neuroscience Institute, the School of Public Health and the Lawrence Berkeley National Laboratory.

"Over the past few years, the links between sleep, beta-amyloid, memory, and Alzheimer's disease have been growing stronger," Jagust said. "Our study shows that this beta-amyloid deposition may lead to a vicious cycle in which sleep is further disturbed and memory impaired."

Using a powerful combination of brain imaging and other diagnostic tools on 26 older adults who have not been diagnosed with dementia, researchers looked for the link between bad sleep, poor memory and the toxic accumulation of beta-amyloid proteins.

"The data we've collected are very suggestive that there's a causal link," said Mander, lead author of the study and a postdoctoral researcher in the Sleep and Neuroimaging Laboratory directed by Walker. "If we intervene to improve sleep, perhaps we can break that causal chain."

A buildup of beta-amyloid has been found in Alzheimer's patients and, independently, in people reporting sleep disorders. Moreover, a 2013 University of Rochester study found that the brain cells of mice would shrink during non-rapid-eye-movement (non-REM) sleep to make space for cerebrospinal fluids to wash out toxic metabolites such as beta-amyloid.

"Sleep is helping wash away toxic proteins at night, preventing them from building up and from potentially destroying brain cells," Walker said. "It's providing a power cleanse for the brain."

Specifically, the researchers looked at how the quantity of beta-amyloid in the brain's medial frontal lobe impairs deep non-REM sleep, which we need to retain and consolidate fact-based memories.

In a previous study, Mander, Jagust and Walker found that the powerful brain waves generated during non-REM sleep play a key role in transferring memories from the hippocampus - which supports short-term storage for information - to longer-term storage in the frontal cortex. In elderly people, deterioration of this frontal region of the brain has been linked to poor-quality sleep.

For this latest study, researchers used positron emission tomography (PET) scans to measure the accumulation of beta-amyloid in the brain; functional Magnetic Resonance Imaging (fMRI) to measure activity in the brain during memory tasks; an electroencephalographic (EEG) machine to measure brain waves during sleep; and statistical models to analyze all the data.

The research was performed on 26 older adults, between the ages of 65 and 81, who showed no existing evidence of dementia or other neurodegenerative, sleep or psychiatric disorders. First, they each received PET scans to measure levels of beta-amyloid in the brain, after which they were tasked with memorizing 120 word pairs, and then tested on how well they remembered a portion of them.

The study participants then slept for eight hours, during which EEG measured their brain waves. The following morning, their brains were scanned using fMRI as they recalled the remaining word pairs. At this point, researchers tracked activity in the hippocampus, where memories are temporarily stored before they are transferred to the prefrontal cortex.

"The more you remember following a good night of sleep, the less you depend on the hippocampus and the more you use the cortex," Walker said. "It's the equivalent of retrieving files from the safe storage site of your computer's hard drive, rather than the temporary storage of a USB stick."

Overall, the results showed that the study participants with the highest levels of beta-amyloid in the medial frontal cortex had the poorest quality of sleep and, consequently, performed worst on the memory test the following morning, with some forgetting more than half of the information they had memorized the previous day.

"The more beta-amyloid you have in certain parts of your brain, the less deep sleep you get and, consequently, the worse your memory," Walker said. "Additionally, the less deep sleep you have, the less effective you are at clearing out this bad protein. It's a vicious cycle.

"But we don't yet know which of these two factors - the bad sleep or the bad protein - initially begins this cycle. Which one is the finger that flicks the first domino, triggering the cascade?" Walker added.

And that's what the researchers will determine as they track a new set of older adults over the next five years.

"This is a new pathway linking Alzheimer's disease to memory loss, and it's an important one because we can do something about it," Mander said.

Other co-authors and researchers on the study are Shawn Marks, Jacob Vogel, Jared Saletin and Vikram Rao at UC Berkeley, Brandon Lu at the California Pacific Medical Center and Sonia Ancoli-Israel at the University of California, San Diego. The study was funded by grants from the National Institute of Aging.

http://www.eurekalert.org/pub_releases/2015-06/tjnj-idp052815.php

Is diabetes protective against amyotrophic lateral sclerosis?

A study of patients in Denmark suggests that type 2 diabetes may be associated with a reduced risk for the fatal neurodegenerative disease amyotrophic lateral sclerosis (ALS), according to an article published online by JAMA Neurology.

Recent reports have suggested a protective association between vascular risk factors, such as obesity or higher body mass index (BMI), higher cholesterol levels and hyperlipidemia with ALS incidence and survival. Patients with type 2 diabetes have, on average, higher BMI, elevated blood lipid levels and defective energy metabolism. However, the association between diabetes and ALS has not been widely explored.

Marianthi-Anna Kioumourtzoglou, Sc.D., of the Harvard T.H. Chan School of Public Health, Boston, and coauthors, examined the association between diabetes, obesity and ALS using data from Danish National Registers for 3,650 patients diagnosed with ALS between 1982 and 2009. The average age at diagnosis was 65.4 years. They were compared with 365,000 healthy control patients.

The authors also identified 9,294 patients with diabetes at least three years prior to the index date (the date of ALS diagnosis or the same date for the matched controls), 55 of whom were subsequently diagnosed with ALS. The average age of the first diabetes-related diagnosis was 59.7 years.

The study found that diabetes, but not obesity, was associated with a reduced risk of ALS. The association with diabetes was affected by both age at ALS diagnosis and age at diabetes diagnosis, with older age at diagnosis for either disease associated with lower risk for ALS.

"We conducted a nationwide, population-based study and observed an overall protective association between diabetes and ALS diagnosis, with the suggestion that type 2 diabetes is protective and type 1 diabetes is a risk factor. Although the mechanisms underlying this association remain unclear, our findings focus further attention on the role of energy metabolism in ALS pathogenesis," the study concludes.

(JAMA Neurol. Published online June 1, 2015. doi:10.1001/jamaneurol.2015.0910. Available pre-embargo to the media at http://media.jamanetwork.com.)

http://www.eurekalert.org/pub_releases/2015-06/byu-ela060115.php

Extra love and support doesn't make up for being a helicopter parent

It's time for helicopter parents to land and stay grounded.

New research by professors at Brigham Young University revealed that parental warmth cannot neutralize the consequences of helicopter parenting. Additionally, a lack of warmth makes the negative effects worse. Such negative effects include lower self-worth and higher risk behavior, such as binge drinking.

"From our past work, we thought there might be something positive about helicopter parenting under certain conditions, but we're just not finding it," study author Larry Nelson said.

The study, published in Emerging Adulthood, is a follow-up to 2012 research on helicopter parenting that found the children of helicopter parents are less engaged in school.

Now they've found that helicopter parenting combined with an absence of parental warmth is especially detrimental to young adults' well-being.

Researchers defined helicopter parenting as parents' over-involvement in the lives of their children.

This includes making important decisions for them, solving their problems and intervening in their children's conflicts.

Warmth is measured by parental availability to talk and spend time together.

Nelson and Padilla-Walker examined data from 438 undergraduate students in four universities nationwide (not including Brigham Young University).

The students self-reported on their parents' controlling behavior and warmth, then on their own self-esteem, risk behaviors and academics.

Results showed that the lack of warmth intensifies both the decrease in self-worth and increase in risk behaviors in the young-adult children of helicopter parents. High levels of parental warmth reduced the negative effects, but did not eliminate them completely.

The findings suggest that loving parents can't justify their helicoptering tendencies; too much control is too much, no matter the parents' affection and support.

"Overall, stepping in and doing for a child what the child developmentally should be doing for him or herself, is negative," Nelson said.

"Regardless of the form of control, it's harmful at this time period."

The authors note that helicopter parenting is relatively uncommon and not as damaging as forms of control that are harsh, punitive or manipulative.

Nelson warned that helicopter parents shouldn't overcompensate by removing themselves completely from their children's lives.

Young adults deserve more autonomy, but still need parental support.

"Lack of control does not mean lack of involvement, warmth and support," Nelson said.

http://www.eurekalert.org/pub_releases/2015-06/vcu-rsm060115.php

Researchers synthesize magnetic nanoparticles that could offer alternative to rare Earth magnets

A team of scientists at Virginia Commonwealth University has synthesized a powerful new magnetic material that could reduce the dependence of the United States and other nations on rare earth elements produced by China.

"The discovery opens the pathway to systematically improving the new material to outperform the current permanent magnets," said Shiv Khanna, Ph.D., a commonwealth professor in the Department of Physics in the College of Humanities and Sciences.

The new material consists of nanoparticles containing iron, cobalt and carbon atoms with a magnetic domain size of roughly 5 nanometers. It can store information up to 790 kelvins with thermal and time-stable, long-range magnetic order, which could have a potential impact for data storage application.

When collected in powders, the material exhibits magnetic properties that rival those of permanent magnets that generally contain rare earth elements. The need to generate powerful magnets without rare earth elements is a strategic national problem as nearly 70 to 80 percent of the current rare earth materials are produced in China. The team's findings will appear in the article "Experimental evidence for the formation of CoFe2C phase with colossal magnetocrystalline-anisotropy," in a forthcoming issue of Applied Physics Letters.

Permanent magnets, specifically those containing rare earth metals, are an important component used by the electronics, communications and automobile industries, as well as in radars and other applications.

Additionally, the emergence of green technology markets - such as hybrid and electric vehicles, direct drive wind turbine power systems and energy storage systems - have created an increased demand for permanent magnets.

However, China is the main supplier of world rare earth demands and has tried to impose restrictions on their export, creating an international problem.

The current paper is a joint experimental theoretical effort in which the new material was synthesized, characterized and showed improved characteristics following the theoretical prediction.

"This is good science along with addressing a problem with national importance," said Ahmed El-Gendy, a former postdoctoral associate in the Department of Chemistry in the College of Humanities and Sciences and a co-author of the paper. Everett Carpenter, Ph.D., a professor in the Department of Chemistry and director of the VCU's Nanoscience and Nanotechnology Program, said the new material is "already showing promise, even for applications beyond permanent magnets."

The research was supported by ARPA-e REACT project 1574-1674 and the U. S. Department of Energy (DOE) through grant DE-SC0006420.

http://www.eurekalert.org/pub_releases/2015-06/uonc-nee052915.php

New evidence emerges on the origins of life

University of North Carolina researchers provide evidence on how the genetic code developed in 2 stages and how primordial chemicals were able to evolve into the first cells on Earth

CHAPEL HILL, NC - In the beginning, there were simple chemicals. And they produced amino acids that eventually became the proteins necessary to create single cells. And the single cells became plants and animals. Recent research is revealing how the primordial soup created the amino acid building blocks, and there is widespread scientific consensus on the evolution from the first cell into plants and animals. But it's still a mystery how the building blocks were first assembled into the proteins that formed the machinery of all cells. Now, two long-time University of North Carolina scientists - Richard Wolfenden, PhD, and Charles Carter, PhD - have shed new light on the transition from building blocks into life some 4 billion years ago.

"Our work shows that the close linkage between the physical properties of amino acids, the genetic code, and protein folding was likely essential from the beginning, long before large, sophisticated molecules arrived on the scene," said Carter, professor of biochemistry and biophysics at the UNC School of Medicine. "This close interaction was likely the key factor in the evolution from building blocks to organisms."

Their findings, published in companion papers in the Proceedings of the National Academy of Sciences, fly in the face of the problematic "RNA world" theory, which posits that RNA - the molecule that today plays roles in coding, regulating, and expressing genes - elevated itself from the primordial soup of amino acids and cosmic chemicals to give rise first to short proteins called peptides and then to single-celled organisms.

Wolfenden and Carter argue that RNA did not work alone; in fact, it was no more likely that RNA catalyzed peptide formation than it was for peptides to catalyze RNA formation.

The finding adds a new layer to the story of how life evolved billions of years ago.

Its name was LUCA

The scientific community recognizes that 3.6 billion years ago there existed the last universal common ancestor, or LUCA, of all living things presently on Earth. It was likely a single-cell organism. It had a few hundred genes. It already had complete blueprints for DNA replication, protein synthesis, and RNA transcription. It had all the basic components - such as lipids - that modern

organisms have. From LUCA forward, it's relatively easy to see how life as we know it evolved.

Before 3.6 billion years, however, there is no hard evidence about how LUCA arose from a boiling caldron of chemicals that formed on Earth after the creation of the planet about 4.6 billion years ago. Those chemicals reacted to form amino acids, which remain the building blocks of proteins in our own cells today.

"We know a lot about LUCA and we are beginning to learn about the chemistry that produced building blocks like amino acids, but between the two there is a desert of knowledge," Carter said. "We haven't even known how to explore it."

The UNC research represents an outpost in that desert.

"Dr. Wolfenden established physical properties of the twenty amino acids, and we have found a link between those properties and the genetic code," Carter said. "That link suggests to us that there was a second, earlier code that made possible the peptide-RNA interactions necessary to launch a selection process that we can envision creating the first life on Earth."

Thus, Carter said, RNA did not have to invent itself from the primordial soup. Instead, even before there were cells, it seems more likely that there were interactions between amino acids and nucleotides that led to the co-creation of proteins and RNA.

Complexity from simplicity

Proteins must fold in specific ways to function properly. The first PNAS paper, led by Wolfenden, shows that both the polarities of the twenty amino acids (how they distribute between water and oil) and their sizes help explain the complex process of protein folding - when a chain of connected amino acids arranges itself to form a particular 3-dimensional structure that has a specific biological function.

"Our experiments show how the polarities of amino acids change consistently across a wide range of temperatures in ways that would not disrupt the basic relationships between genetic coding and protein folding," said Wolfenden, Alumni Distinguished Professor of Biochemistry and Biophysics. This was important to establish because when life was first forming on Earth, temperatures were hot, probably much hotter than they are now or when the first plants and animals were established.

A series of biochemical experiments with amino acids conducted in Wolfenden's lab showed that two properties - the sizes as well as the polarities of amino acids - were necessary and sufficient to explain how the amino acids behaved in folded proteins and that these relationships also held at the higher temperatures of Earth 4 billion years ago.

The second PNAS paper, led by Carter, delves into how enzymes called aminoacyl-tRNA synthetases recognized transfer ribonucleic acid, or tRNA. Those enzymes translate the genetic code.

"Think of tRNA as an adapter," Carter said. "One end of the adapter carries a particular amino acid; the other end reads the genetic blueprint for that amino acid in messenger RNA. Each synthetase matches one of the twenty amino acids with its own adapter so that the genetic blueprint in messenger RNA faithfully makes the correct protein every time."

Carter's analysis shows that the two different ends of the L-shaped tRNA molecule contained independent codes or rules that specify which amino acid to select. The end of tRNA that carried the amino acid sorted amino acids specifically according to size.

The other end of the L-shaped tRNA molecule is called the tRNA anticodon. It reads codons, which are sequences of three RNA nucleotides in genetic messages that select amino acids according to polarity.

Wolfenden and Carter's findings imply that the relationships between tRNA and the physical properties of the amino acids - their sizes and polarities - were crucial during the Earth's primordial era. In light of Carter's previous work with very small active cores of tRNA synthetases called Urzymes, it now seems likely that selection by size preceded selection according to polarity. This ordered selection meant that the earliest proteins did not necessarily fold into unique shapes, and that their unique structures evolved later.

Carter said, "Translating the genetic code is the nexus connecting pre-biotic chemistry to biology." He and Wolfenden believe that the intermediate stage of genetic coding can help resolve two paradoxes: how complexity arose from simplicity, and how life divided the labor between two very different kinds of polymers: proteins and nucleic acids.

"The fact that genetic coding developed in two successive stages - the first of which was relatively simple - may be one reason why life was able to emerge while the earth was still quite young," Wolfenden noted.

An earlier code, which enabled the earliest coded peptides to bind RNA, may have furnished a decisive selective advantage. And this primitive system could then undergo a natural selection process, thereby launching a new and more biological form of evolution.

"The collaboration between RNA and peptides was likely necessary for the spontaneous emergence of complexity," Carter added. "In our view, it was a peptide-RNA world, not an RNA-only world."

The National Institutes of Health funded this work. Dr. Wolfenden holds a joint appointment in the department of chemistry in the College of Arts and Sciences at UNC-Chapel Hill.

http://www.climatecentral.org/news/ocean-warming-species-change-19051

Ocean Species Set for Reshuffle Unseen in 3 Million Years

The world's oceans could face a massive reshuffling by the end of the century – the likes of which hasn't been seen in as many as 3 million years – due to warming waters.

By Brian Kahn

Changes are already afoot in the oceans. Roughly 93 percent of the heat trapped by human greenhouse gas emissions is ending up in the world's seas and already contributing to changes from slowing plankton growth to recent incursions of tuna near Alaska, thousands of miles from their normal range.

If greenhouse gas emissions continue to build, that heat could create wholesale changes for the vast majority of the world's oceans (which, of course, make up the vast majority of the world).

The findings come from a new study published in Nature Climate Change, which looks at future climate projections and the distant past when 60-foot sharks prowled the oceans, sea levels were 100 feet higher and the globe was about 11°F hotter. Oh, and humans weren't around, either.

There's one major similarity between our current period and 3 million years ago, a period known as the Pliocene: the amount of greenhouse gas concentrations in the atmosphere. It's a trait that makes it a powerful comparison for what the next century may have in store unless humans cut their greenhouse gas emissions.

"What we have found is that if we constrain global warming by less than 2°C, ocean changes will be relatively benign on the global scale," Grégory Beaugrand said. "But if we are above this threshold, we will have a huge reorganization of marine biodiversity."

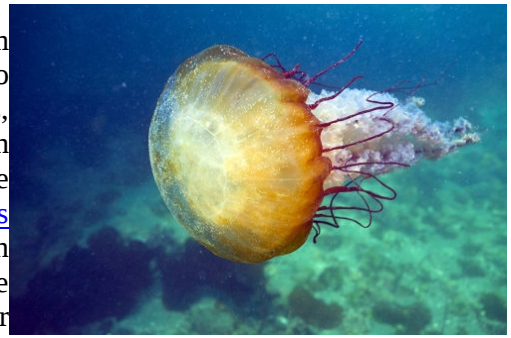
Beaugrand is an ocean researcher at the French National Center for Science Research and lead author of the new report, which shows that if greenhouse gas emissions continue unabated, up to 70 percent of world's oceans could see biodiversity shifts unprecedented in modern times.

The findings show that species could vacate the tropics as warming water send species poleward with nothing in place to fill their void.

For species already living near the poles, they would face a wave of invaders that could outcompete them for resources. Warming waters would eventually make any suitable habitat disappear. The outcome in both cases is the distinct possibility of extinction.

"It (the study) demonstrates the capacity for huge marine biomes to be fundamentally reorganized and disturbed, and shows us there are real differences between moderate warming and severe warming," Sarah Moffitt, a postdoctoral researcher at the University of California, Davis' Bodega Marine Laboratory, said.

The study takes a macro-level view so its unclear how specific species would react to the changes.



A brown sea nettle drifting through Monterey Harbor in California. **NOAA/flickr**

But everything in the sea from crustaceans to cetaceans would have to contend with these shifting conditions, the effects of which would be felt on land as well. Fisheries and aquaculture contributed $274 billion to the world's GDP in 2012, the most recent year with data available. As species move or die off, people that rely on them for livelihoods will have to respond.

"We will have species that will disappear but some others will take their place," Beaugrand said.

"But fishermen, usually they are adapted to a certain type of species. They will have to re-adapt to a new type of species and adaptation is expensive."

Signs of warming are already floating across the oceans.

Recent sightings of tuna off the Alaska coast, thousands of miles from their usual habitat, made headlines earlier this year as record-breaking warm water spread up the West Coast.

The cause is likely natural but it could be a sign of things to come.

More broadly, warming waters can slow plankton growth, which form the base of the marine food chain, resulting in less food for fish.

"What's going on now does affect people and their fishing but not very rapidly," Lisa Suatoni, a senior scientist with the Natural Resources Defense Council's ocean program, said.

"It's at a rate where adapting the fishing fleet can occur. But if we go into greater warming, then we're going to see really radical changes."

Suatoni authored a study earlier this year looking at the vulnerability of U.S. coastal communities to ocean acidification.

The new findings don't address acidification, which Beaugrand said is unlikely to play as large a role as warming in biodiversity shifts.

"Most species do not control their inner temperature so for 99.9 percent of the species on earth, temperature is a very, very important factor because they are generally in equilibrium with their outer temperature," Beaugrand said. "Temperature is a master parameter."

http://www.eurekalert.org/pub_releases/2015-06/e-ibd060115.php

Infant brains develop years faster than we thought

Scientists from the University of Louvain have discovered that a key element of infant brain development occurs years earlier than previously thought.

The way we perceive faces -- using the right hemisphere of the brain -- is unique and sets us apart from non-human primates. It was thought that this ability develops as we learn to read, but a new study published in the journal eLife shows that in babies as young as four months it is already highly evolved.

"Just as language is impaired following damage to the brain's left hemisphere, damage to the right hemisphere can impair our ability to distinguish faces so it is critical to understand how it develops," says co-author Bruno Rossion, Principal Investigator at the University of Louvain.

Researchers used a cap fitted with electrodes to monitor the brain activity of 15 babies as they sat on their mothers' laps and watched a rapid succession of images over 20 seconds. They were shown 48 images of faces that differed in viewpoint, colour, lighting, and background, interspersed with 200 images of animals, plants, and man-made objects.

Each image was shown for only 166 milliseconds, the same rate used for adult studies. Compared to other images, the appearance of a face was shown to coincide with a specific spike in stimulation of the right hemisphere of the brain. The difference between the right and the left hemisphere was even more pronounced than in the same study with adults, confounding previous assumptions.

"Given the enormous resources devoted to digital face recognition, the babies' brain accomplishment is not trivial," says Rossion. "The success of this research method in babies demonstrates that it can be used in all ages to improve our understanding of how we develop the ability to perceive complex images."

Humans far outperform computer algorithms in categorizing natural visual images. The face is such a frequent and socially important stimulus in human development that it is ideal for studying how we develop the ability to visually categorise objects.

A fundamental element of face perception is our ability to tell individuals apart. The authors can now use the same methods to define when this emerges and how it develops with age.

"Parents and carers are already aware of how quickly babies' brains develop but, until now, gathering evidence has been hard due to the limitations of the methods used," says Rossion.

The paper 'Rapid categorization of natural face images in the infant right hemisphere' can be freely accessed online at http://dx.doi.org/10.7554/eLife.06564. Contents, including text, figures, and data, are free to re-use under a CC BY 4.0 license.

http://www.eurekalert.org/pub_releases/2015-06/uoa-gco060115.php

Genetic causes of cerebral palsy trump birth causes

University of Adelaide researchers have discovered cerebral palsy has an even stronger genetic cause than previously thought, leading them to call for an end to unnecessary caesareans and arbitrary litigation against obstetric staff.

In an authoritative review published in the American Journal of Obstetrics & Gynecology, members of the Australian Cerebral Palsy Research Group, based at the University of Adelaide's Robinson Research Institute, argue that up to 45% of cerebral palsy cases can have genetic causes.

This builds on research published in February this year by the group which found at least 14% of cerebral palsy cases are likely to be caused by a genetic mutation. And the group expects the percentage of genetically caused cerebral palsy cases will continue to increase as genetic sequencing techniques evolve.

The University of Adelaide's Emeritus Professor Alastair MacLennan, leader of the research group, says the realisation by courts that many cases of cerebral palsy cannot be prevented by differences in labour management should reduce the adverse influence of obstetric litigation.

"For many years it was assumed, without good evidence, cerebral palsy was caused by brain damage at birth through lack of oxygen. This belief along with the temptation to blame the insured, and the high cost of caring for children with cerebral palsy, has fuelled litigation against obstetric staff," says Emeritus Professor MacLennan.

"Numerous recent studies have shown that despite an increase in caesarean deliveries over 50 years, which have risen from 5% to 34% in Australia, there has been no overall change in cerebral palsy rates.

"Some of the increase in caesareans appears to be due to defensive obstetrics and fear of litigation - there are lower rates of caesareans in countries with a "no-fault insurance scheme" like New Zealand, where rates are 23%.

"It's estimated that $300 million is paid on cerebral palsy settlements in Australia each year. I hope that our research will help end unfounded cerebral palsy related litigation," he says.

Several more years of research are needed but the research group believes that eventually cerebral palsy genetic testing before, during and after pregnancy will be introduced.

"It is now becoming apparent that cerebral palsy is an umbrella diagnosis for children with non-progressive disorders of movement control and posture, and that there are many types and antenatal influences including genetic causes," says the University of Adelaide's Professor Jozef Gecz, Head of Neurogenetic Research, Robinson Research Institute.

"Cerebral is akin to many other neurodevelopmental disorders such as intellectual disability, autism and epilepsy, co-morbidities that are often seen with cerebral palsy, and they too have many genetic causes," he says.

"Many children who have received a diagnosis of cerebral palsy may have an inherited or spontaneous genetic cause and this is exciting because we can now focus research on the beginning of pregnancy and not so unfruitfully on the circumstances of birth," says Dr Suzanna Thompson, co-author on the paper and paediatric neurologist at the Women's and Children's Hospital, Adelaide.

http://www.eurekalert.org/pub_releases/2015-06/ru-mce060215.php

Microendoscope could eliminate unneeded biopsies

Rice University device nearly doubled sensitivity of esophageal cancer screenings

In a clinical study of patients in the United States and China, researchers found that a low-cost, portable, battery-powered microendoscope developed by Rice University bioengineers could eventually eliminate the need for costly biopsies for many patients undergoing standard endoscopic screening for esophageal cancer.

The research is available online in the journal Gastroenterology and was co-authored by researchers from nearly a dozen institutions that include Rice, Baylor College of Medicine, the Chinese Academy of Medical Sciences and the National Cancer Institute.

The clinical study, which involved 147 U.S. and Chinese patients undergoing examination for potentially malignant squamous cell tumors, explored whether Rice's low-cost, high-resolution fiber-optic imaging system could reduce the need for unnecessary biopsies when used in combination with a conventional endoscope -- the worldwide standard of care for esophageal cancer diagnoses.

The study involved patients from two U.S. and two Chinese hospitals: Mt. Sinai Medical Center in New York, the University of Texas MD Anderson Cancer Center in Houston, the Cancer Institute and Hospital of the Chinese Academy of Medical Sciences in Beijing and First University Hospital in Jilin, China.

In the study, all 147 patients with suspect lesions were examined with both a traditional endoscope and Rice's microendoscope. Biopsies were obtained based upon the results of the traditional endoscopic exam.

A pathology exam revealed that more than half of those receiving biopsies -- 58 percent -- did not have high-grade precancer or cancer. The researchers found that the microendoscopic exam could have spared unnecessary biopsies for about 90 percent of the patients with benign lesions.

"For patients, biopsies are stressful and sometimes painful," said lead researcher Rebecca Richards-Kortum, Rice's Stanley C. Moore Professor of Bioengineering, professor of electrical and computer engineering and director of Rice 360°: Institute for Global Health Technologies. "In addition, in low-resource settings, pathology costs frequently exceed endoscopy costs. So the microendoscope could both improve patient outcomes and provide a significant cost-saving advantage if used in conjunction with a traditional endoscope."

When examined under a microscope, cancerous and precancerous cells typically appear different from healthy cells. The study of cellular structures is known as histology, and a histological analysis is typically required for an accurate diagnosis of both the type and stage of a cancerous tumor.

To determine whether a biopsy is needed for a histological exam, health professionals often use endoscopes, small cameras mounted on flexible tubes that can be inserted into the body to visually examine an organ or tissue without surgery. Rice's high-resolution microendoscope uses a 1-millimeter-wide fiber-optic cable that is attached to the standard endoscope. The cable transmits images to a high-powered fluorescence microscope, and the endoscopist uses a tablet computer to view the microscope's output. The microendoscope provides images with similar resolution to traditional histology and allows endoscopists to see individual cells and cell nuclei in lesions suspected of being cancerous.

By providing real-time histological data to endoscopists, Rice's microendoscope can help rule out malignancy in cases that would otherwise require a biopsy.

"While traditional endoscopy can rule out malignancy and eliminate the need for biopsies for some patients, in a significant number of cases the difference between malignant and benign lesions only becomes apparent through a histological analysis," said study co-author Dr. Sharmila Anandasabapathy, professor of medicine and gastroenterology at Baylor College of Medicine and director of Baylor Global Initiatives and the Baylor Global Innovation Center.

Richards-Kortum's lab specializes in the development of low-cost optical imaging and spectroscopy tools to detect cancer and infectious disease at the point of care. Her research group is particularly interested in developing technology for low-resource settings, and the microendoscope was developed as part of that effort. It is battery-operated, inexpensive to operate and requires very little training. Results from the clinical study verified that both experienced and novice endoscopists could use the microendoscope to make accurate assessments of the need for a biopsy.

Clinical studies of Rice's microendoscope are either planned or underway for a dozen types of cancer including cervical, bladder, oral and colon cancers.

"More than half of cancer deaths today occur in the developing world, often in low-resource areas," Anandasabapathy said. "The World Health Organization and other important international bodies have called for increased global focus on noncommunicable diseases like cancer, and Rice's microendoscope is a great

example of what the right kind of technology can do to change health care in low-resource countries."

Additional study co-authors include Timothy Quang, Dongsuk Shin and Richard Schwarz, all of Rice; James Godbold, Marion-Anna Protano, Michelle Lee, Josephine Mitcham, Erin Moshier, Alexandros Polydorides and Courtney Hudson, all of Mount Sinai Medical Center; Junsheng Cui, Hong Xu, Fan Zhang and Weiran Xum, all of the First Hospital of Jilin University; Guiqi Wang and Liyan Xue of the Cancer Institute and Hospital, Chinese Academy of Medical Sciences; Sanford Dawsey of the National Cancer Institute; Mark Pierce of Rutgers University; Manoop Bhutani of the University of Texas MD Anderson Cancer Center; Neil Parikh of Yale University; and Chin Hur of Massachusetts General Hospital.

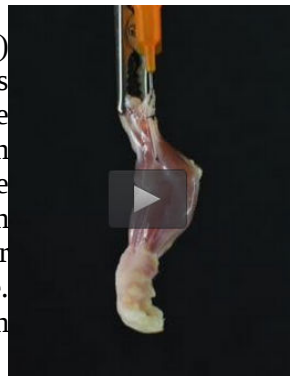
A copy of the paper, "Low-Cost High-Resolution Microendoscopy for the Detection of Esophageal Squamous Cell Neoplasia: An International, Multicenter Trial," is available at: http://www.gastrojournal.org/article/S0016-5085(15)00675-7/abstract

http://www.eurekalert.org/pub_releases/2015-06/mgh-mgt060215.php

Mass. General team develops transplantable bioengineered forelimb in an animal model

Experimental technique used to create whole organs appears feasible for creation of complex tissues

A team of Massachusetts General Hospital (MGH) investigators has made the first steps towards development of bioartificial replacement limbs suitable for transplantation. In their report, which has been published online in the journal Biomaterials, the researchers describe using an experimental approach previously used to build bioartificial organs to engineer rat forelimbs with functioning vascular and muscle tissue. They also provided evidence that the same approach could be applied to the limbs of primates



Over a period of 52 hours, infusion of a detergent solution removes cells from a rat forelimb, leaving behind the cell-free matrix scaffolding onto which new tissues can be regenerated. VIDEO **Bernhard Jank, M.D., Ott Laboratory, Massachusetts General Hospital Center for Regenerative Medicine**

"The composite nature of our limbs makes building a functional biological replacement particularly challenging," explains Harald Ott, MD, of the MGH Department of Surgery and the Center for Regenerative Medicine, senior author of the paper. "Limbs contain muscles, bone, cartilage, blood vessels, tendons, ligaments and nerves - each of which has to be rebuilt and requires a specific supporting structure called the matrix. We have shown that we can maintain the matrix of all of these tissues in their natural relationships to each other, that we can culture the entire construct over prolonged periods of time, and that we can repopulate the vascular system and musculature."

The authors note that more than 1.5 million individuals in the U.S. have lost a limb, and although prosthetic technology has greatly advanced, the devices still have many limitations in terms of both function and appearance. Over the past two decades a number of patients have received donor hand transplants, and while such procedures can significantly improve quality of life, they also expose recipients to the risks of life-long immunosuppressive therapy. While the progenitor cells needed to regenerate all of the tissues that make up a limb could be provided by the potential recipient, what has been missing is the matrix or scaffold on which cells could grow into the appropriate tissues.

The current study uses technology Ott discovered as a research fellow at the University of Minnesota, in which living cells are stripped from a donor organ with a detergent solution and the remaining matrix is then repopulated with progenitor cells appropriate to the specific organ. His team and others at MGH and elsewhere have used this decellularization technique to regenerate kidneys, livers, hearts and lungs from animal models, but this is the first reported use to engineer the more complex tissues of a bioartificial limb.

The same decellularization process used in the whole-organ studies - perfusing a detergent solution through the vascular system - was used to strip all cellular materials from forelimbs removed from deceased rats in a way that preserved the primary vasculature and nerve matrix. After thorough removal of cellular debris - a process that took a week - what remained was the cell-free matrix that provides structure to all of a limb's composite tissues. At the same time, populations of muscle and vascular cells were being grown in culture.

The research team then cultured the forelimb matrix in a bioreactor, within which vascular cells were injected into the limb's main artery to regenerate veins and arteries. Muscle progenitors were injected directly into the matrix sheaths that define the position of each muscle. After five days in culture, electrical stimulation was applied to the potential limb graft to further promote muscle formation, and after two weeks, the grafts were removed from the bioreactor. Analysis of the bioartificial limbs confirmed the presence of vascular cells along blood vessel walls and muscle cells aligned into appropriate fibers throughout the muscle matrix.

Functional testing of the isolated limbs showed that electrical stimulation of muscle fibers caused them to contract with a strength 80 percent of what would be seen in newborn animals. The vascular systems of bioengineered forelimbs transplanted into recipient animals quickly filled with blood which continued to circulate, and electrical stimulation of muscles within transplanted grafts flexed

the wrists and digital joints of the animals' paws. The research team also successfully decellularized baboon forearms to confirm the feasibility of using this approach on the scale that would be required for human patients.

Ott notes that, while regrowing nerves within a limb graft and reintegrating them into a recipient's nervous system is one of the next challenges that needs to be faced, the experience of patients who have received hand transplants is promising. "In clinical limb transplantation, nerves do grow back into the graft, enabling both motion and sensation, and we have learned that this process is largely guided by the nerve matrix within the graft. We hope in future work to show that the same will apply to bioartificial grafts. Additional next steps will be replicating our success in muscle regeneration with human cells and expanding that to other tissue types, such as bone, cartilage and connective tissue."

http://www.eurekalert.org/pub_releases/2015-06/nu-rpe060215.php

Researchers pinpoint epicenter of brain's predictive ability

Researchers find that limbic tissue, which also helps to create emotions, is at the top of the brain's prediction hierarchy

In recent years, scientists have discovered the human brain works on predictions, contrary to the previously accepted theory that it reacts to the sensations it picks up from the outside world. Experts say humans' reactions are in fact the body adjusting to predictions the brain is making based on the state of our body the last time it was in a similar situation.

Now, University Distinguished Professor Lisa Feldman Barrett at Northeastern has reported finding the epicenter of those predictions.

In an article published in Nature last week, Barrett contends that limbic tissue, which also helps to create emotions, is at the top of the brain's prediction hierarchy. She coauthored the paper with W. Kyle Simmons, of the Laureate Institute for Brain Research in Tulsa, Oklahoma.

"The unique contribution of our paper is to show that limbic tissue, because of its structure and the way the neurons are organized, is predicting," Barrett said. "It is directing the predictions to everywhere else in the cortex, and that makes it very powerful."

For example, when a person is instructed to imagine a red apple in his or her mind's eye, Barrett explained that limbic parts of the brain send predictions to visual neurons and cause them to fire in different patterns so the person can "see" a red apple.

Barrett is a faculty member in the Department of Psychology and is director of the Interdisciplinary Affective Science Laboratory. A pioneer in the psychology of emotion and affective neuroscience, she has challenged the foundation of affective science by showing that people are the architects of their own emotional experiences.

In the Nature paper, Barrett summarized research on the cellular composition of limbic tissue, which shows that limbic regions of the brain send but do not receive predictions. This means that limbic regions direct processing in the brain. They don't react to stimulation from the outside world. This is ironic, Barrett argues, because when scientists used to believe that limbic regions of the brain were the home of emotion, they were seen as mainly reactive to the world.

Common sense tells you that seeing is believing, but really the brain is built for things to work the other way around: you see (and hear and smell and taste) what you believe. And believing is largely based on feeling. In her paper, Barrett shows that your brain is not wired to be a reactive organ. It's wired to ask the question: "The last time I was in a situation like this, what sensations did I encounter, and how did I act?" And the sensations that seem to matter most are the ones that are inside your own body, which are called "interoceptions."

"What your brain is trying to do is guess what the sensation means and what's causing the sensations so it can figure out what to do about them," Barrett said. "Your brain is trying to put together thoughts, feelings, and perceptions so they arrive as needed, not a second afterwards."

http://www.eurekalert.org/pub_releases/2015-06/f-asb052715.php

Autism struck by surprise

A new study shows that social and sensory overstimulation drives autistic behaviors.

The study, conducted on rats exposed to a known risk factor in humans, supports the unconventional view of the autistic brain as hyper-functional, and offers new hope with therapeutic emphasis on paced and non-surprising environments tailored to the individual's sensitivity.

For decades, autism has been viewed as a form of mental retardation, a brain disease that destroys children's ability to learn, feel and empathize, thus leaving them disconnected from our complex and ever-changing social and sensory surroundings. From this perspective, the main kind of therapeutic intervention in autism to date aims at strongly engaging the child to revive brain functions believed dormant. Researchers at the Swiss Federal Institute of Technology in Lausanne (EPFL) completed a study that turns this traditional view of autism completely around.

The study demonstrates that, in rats exposed to a known autism risk factor, unpredictable environmental stimulation drives autistic symptoms at least as much as an impoverished environment does, and that predictable stimulation can prevent these symptoms. The study is also evidence for a drastic shift in the

clinical approach to autism, away from the idea of a damaged brain that demands extensive stimulation. Instead, autistic brains may be hyper-functional and thus require enriched environments that are non-surprising, structured, safe, and tailored to a particular individual's sensitivity.

"The valproate rat model used is highly relevant for understanding autism, because children exposed to valproate in the womb have an increased chance of presenting autism after birth," says Prof. Henry Markram, co-author of the study and father of a child with autism. Accordingly, rats exposed to valproate in early embryonic development demonstrate behavioral, anatomical and neurochemical abnormalities that are comparable to characteristics of human autism.

The scientists here show that if rats are exposed to this prenatal autism risk factor and reared in a home environment that is calm, safe, and highly predictable with little surprise -- while still rich in sensory and social engagement -- do not develop symptoms of emotional over-reactivity such as fear and anxiety, nor social withdrawal or sensory abnormalities.

"We were amazed to see that environments lacking predictability, even if enriched, favored the development of hyper-emotionality in rats exposed to the prenatal autism risk factor", says Henry Markram.

The study critically shows that in certain individuals, non-predictable environments lead to the development of a wider range of negative symptoms, including social withdrawal and sensory abnormalities. Such symptoms normally prevent individuals from fully benefiting from and contributing to their surroundings, and are thus the targets of therapeutic success. The study identifies drastically opposite behavioral outcomes depending on levels of predictability in the enriched environment, and suggests that the autistic brain is unusually sensitive to predictability in rearing environment, but to different extent in different individuals. The results were received with enthusiasm by the autism community, which consistently reports the high sensitivity of people with autism to change and to sensory stimulation.

The study is strong evidence for the Intense World Theory of Autism, proposed in 2007 by neuroscientists Kamila Markram and Henry Markram, both co-authors on the present study. This theory is based on recent research suggesting that the autistic brain, in both humans and animal models, reacts differently to stimuli. It proposes that an interaction -- between an individual's genetic background with biologically toxic events early in embryonic development -- triggers a cascade of abnormalities that create hyper-functional brain microcircuits, the functional units of the brain. Once activated, these hyper-functional circuits could become autonomous and affect further brain functional connectivity and development. These would lead to an experience of the world as intense, fragmented, and overwhelming; while differences in severity between persons with autism would stem from the system affected and the timing of the effect. The authors acknowledge the need to test these ideas in humans.

If children with autism are indeed more neurobiologically sensitive to the environment than other children as a result of early brain hyper-function, then predictable environmental stimulation tailored to an individual's specific hyper-sensitivity could significantly improve quality of life, by preventing or ameliorating the debilitating autistic symptoms of sensory overload and anxiety or fears, and allow the child to flourish.

"A stable, structured environment rich in stimuli could help children with autism, by providing a safe haven from an overload of sensory and emotional stimuli. In contrast, an environment with many unpredictable, changing stimuli could make their symptoms worse, raising anxiety and fear and making these children retract into a bubble," says Kamila Markram.

"Importantly, such constructive interactions with a safe and predictable world at key developmental sensitive periods early on could enhance coping and succeeding in subsequent less structured or unfamiliar contexts, and give place to a harmonious individual development," says Monica Favre, first author of the study.

This study has immediate implications for clinical and research settings, because enhanced brain processing and sensitivity to environmental surprises need to be considered as possible defining characters of autism. This breakthrough suggests that if brain hyper-function can be diagnosed soon after birth, at least some of the debilitating effects of a supercharged brain can be prevented, not by environmental enrichment per se, but by highly specialized environmental stimulation that is safe, consistent, controlled, announced and only changed very gradually at the pace determined by each child.

http://www.eurekalert.org/pub_releases/2015-06/jhm-rba060215.php

Re-inflating balloon after carotid stenting appears to double risk of stroke and death

Findings lead Johns Hopkins surgeons to call for end to routine 'ballooning' once stent is in place

After reviewing outcomes from thousands of cases, researchers at Johns Hopkins report that patients with blocked neck arteries who undergo carotid stenting to prop open the narrowed blood vessels fare decidedly worse if their surgeons re-inflate a tiny balloon in the vessel after the mesh stent is in place.

Although the overall risk of stroke and death is low in patients who undergo carotid stenting, the common practice of "ballooning" the vessel after the wire

mesh is inserted can double the risk of death and stroke during or shortly after the procedure, according to findings published online May 30 in the Journal of Vascular Surgery.

"Ballooning after placing the stent appears to cause the very complication it's intended to prevent," says study senior author Mahmoud Malas, M.D., M.H.S., an associate professor of surgery at the Johns Hopkins University School of Medicine. "Surgeons should avoid doing it. Period."

The carotid arteries, which run on both sides of the neck and ferry oxygen-rich blood from the heart to the brain, can become narrowed and stiff from buildup of fat and calcium deposits over time. The condition, known as carotid stenosis, is responsible for half of the nearly 800,000 strokes that occur in the United States each year, according to the Centers for Disease Control and Prevention.

Patients with severe blockages typically undergo surgery to scrape off the fatty deposits from the walls of the vessel, the preferred approach that carries notably lower stroke risk but is not recommended for people too sick to withstand traditional surgery. Such patients are often offered minimally invasive stent placement to flatten and stabilize the built up debris inside the clogged vessels.

To place the stent, surgeons thread a catheter through the groin and up into the neck artery. Once inside, surgeons typically insert a tiny surgical balloon and inflate it to compress the fatty deposits, open up the vessel, and make room for the stent.

Once the stent is in place, however, it is common practice to re-inflate the balloon to expand the wire mesh and firm up its position against the artery walls. But the new Johns Hopkins study shows re-inflating the balloon once the stent is in place fuels stroke risk.

A previous study led by Malas showed post-stent ballooning could cause another serious complication marked by a precipitous drop in blood pressure and breathing problems.

For the new study, the team analyzed stroke and death risk in more than 3,700 patients, ages 19 to 89, who had carotid stenting between 2005 and 2014 in hospitals across the United States and whose outcomes were reported in the Vascular Quality Initiative, a national repository of vascular surgery outcomes. One group of patients had pre-stent ballooning only, another was treated with post-stent ballooning only, and a third had the combination technique involving balloon use both before and after stent placement.

While the overall risk of stroke and death was relatively low -- 2.4 percent of patients had a stroke within 30 days of treatment and less than 1 percent died -- those treated with combination pre and post-stent ballooning were twice as likely to suffer a stroke or die. Those who had post-stent ballooning alone also had an elevated risk but in the final analysis, the difference did not reach statistical significance.

The researchers believe that repeat ballooning after stent placement causes stroke by driving the stent deeper into the fragile vessel walls and disturbing the fatty plaque that is built up atop the walls. This, they say, can cause splinters of plaque to chip off and make their way to the brain.

"The main goal of carotid stenting is not so much to restore blood flow as to contain and stabilize preexisting plaque," Malas says. "Our message is clear: Once inside the artery, leave the stent alone."

Unlike the more common heart stenting where the main goal is to open the heart's arteries and restore blood flow to the cardiac muscle, stenting the carotid arteries is done with the brain in mind.

"Carotid stenting is unique," says study author Tammam Obeid, M.B.B.S., a surgery fellow at the Johns Hopkins University School of Medicine. "It is the only stenting procedure where the end target is not muscle but the far more delicate tissue of the brain."

Other investigators involved in the study were Dean Arnaoutakis, Isibor Arhuidese, Umair Qazi, Christopher Abularrage, James Black and Bruce Perler, all of Johns Hopkins.

http://www.bbc.com/news/science-environment-32976352

Cooking skills may have emerged millions of years ago

New research suggests that chimps have most of the mental capabilities needed to cook food.

By Pallab Ghosh Science correspondent, BBC News

This suggests that the ability to cook food is deep seated and may have arisen in human ancestors millions of years ago.

The conclusions also indicate that humans may have developed the ability to cook very soon after they learned how to control fire.

The study has been published in one of the journals of the Royal Society.

Surprising as it may seem, even boiling an egg requires advanced mental skills. Whereas other animals tend to start eating whatever food they find or hunt straight away, humans can store and cook their food, even if we are fairly hungry, because we know that if we wait what we eventually eat will taste better.

It seems that our ability to smack our lips at the prospect of a delicious, well prepared meal requires a similar inspired leap of the imagination as producing art, developing language and creating the technologies that make us uniquely human.

So when your mind wanders and thinks of a nice meal when you should really be paying attention to something else, be assured that it is this foodie forethought that makes us human.

Masterchef

So when did we first develop this ability? To find out, according to Dr Felix Warneken of Harvard University conducted a simian MasterChef contest in which he conducted a series of experiments on chimpanzees to see whether they had what it took to be cooks. Clearly chimps can't cook and so there was no point in giving them a bag of shopping and letting them loose in a kitchen with assorted pots and pans, amusing though the spectacle might have been.

Instead, Dr Warneken carried out a series of experiments to test the individual cognitive skills the chimps needed to be able to cook. He looked to see if they preferred cooked rather than raw food, whether they could wait until raw food could be cooked and if they would put raw food into a box that scientists switched for cooked food. He found that they passed all these tests and more.

So why don't chimps cook? Not being able to control fire is one reason and another, according to Dr Warneken, is that cooking requires what he describes as "social skills" that chimps don't possess.

By social skills he is not alluding to their unremarkable table manners nor their lack of witty dinner party conversation. Rather, it is their inability to trust others in their social groups not to steal their food while they are preparing to cook it that he is referring and it is this he believes is one of the key factors holding them back from being able to cook. Gulping something down as soon as you have foraged it is the surest way of keeping it safe.

According to Dr Warneken, his experiments show that that most of the mental skills needed to cook were there in human ancestors between 5 to 7 million years ago and so all it took for the first emergence of the culinary arts was the controlled use of fire and the ability to trust other people not to pinch our food while our back was turned. "Trust is another component for cooking to become a practice in a social group," he said. "This is required in addition to the individual psychological capacities that we targeted in our experiments."

The motivation for the study was to investigate a controversial theory that cooking was necessary for human brains to become larger. The idea by the primatologist Prof Richard Wrangham, also at Harvard, is that cooking enabled our ancestors to eat more protein, which helped our ancestors develop their brains.

The results indicate that early humans had everything in place once they had learned to control fire and so, according to Dr Warneken, supports Prof Wrangham's ideas. "For this hypothesis to work humans must have adopted cooking fairly early in their evolution," he said.

Experts in human evolution say that they find it "interesting" that chimpanzees and humans share several of the essential psychological capacities needed, but believe that the chimp study does not add much new information to the human story.

Digestible

Prof Chris Stringer of the Natural History Museum in London said: "Cooking was an important milestone for humans in terms of making meat more digestible and neutralising pathogens and toxins, also for its social role, but best evidence for the ability to make fire at will only shows in the last 400,000 years".

Fred Spoor, a professor at University College in London who studies human evolution, said: "Cooking did not happen until 300,000 to 400,000 years ago. That is late in 7 million years of human evolution, so to put it bluntly, who cares that early humans may have liked the idea of cooked food? Perhaps they would have liked eating naturally roasted carcasses of animals occasionally trapped in savannah fires, but that is not cooking."

And as for the idea of cooking driving the transition to bigger brains?

"Substantially larger brains initially emerge around 1.5 million years ago and a major leap was around 500,000 years ago," said Prof Spoor.

"Hence, meat eating probably made this possible but whether roasting played a role at 1.5 million years ago is an open question, because there is poor or no evidence (for it at this time). Cooking at 500,000 years ago is more likely."

http://www.eurekalert.org/pub_releases/2015-06/cu-sps060215.php

Scientists produce strongest evidence yet of schizophrenia's causes

Researchers discover that risk mutations disrupt a delicate chemical balance in the brain, responsible for brain development and function

An international team of scientists led by Cardiff University researchers has provided the strongest evidence yet of what causes schizophrenia - a condition that affects around 1% of the global population.

Published today (17:00BST, 03/06/2015) in the journal Neuron, their work presents strong evidence that disruption of a delicate chemical balance in the brain is heavily implicated in the disorder.

In the largest ever study of its kind, the team found that disease-linked mutations disrupt specific sets of genes contributing to excitatory and inhibitory signalling, the balance of which plays a crucial role in healthy brain development and function. The breakthrough builds on two landmark studies led by members of the Cardiff University team, published last year in the journal Nature.

"We're finally starting to understand what goes wrong in schizophrenia," says lead author Dr Andrew Pocklington from Cardiff University's MRC Centre for Neuropsychiatric Genetics and Genomics. "Our study marks a significant step towards understanding the biology underpinning schizophrenia, which is an

incredibly complex condition and has up until very recently kept scientists largely mystified as to its origins.

"We now have what we hope is a pretty sizeable piece of the jigsaw puzzle that will help us develop a coherent model of the disease, while helping us to rule out some of the alternatives. "A reliable model of disease is urgently needed to direct future efforts in developing new treatments, which haven't really improved a great deal since the 1970s."

Professor Hugh Perry, who chairs the Medical Research Council Neuroscience and Mental Health Board said: "This work builds on our understanding of the genetic causes of schizophrenia - unravelling how a combination of genetic faults can disrupt the chemical balance of the brain.

"Scientists in the UK, as part of an international consortium, are uncovering the genetic causes of a range of mental health issues, such as schizophrenia.

"In the future, this work could lead to new ways of predicting an individual's risk of developing schizophrenia and form the basis of new targeted treatments that are based on an individual's genetic makeup."

A healthy brain is able to function properly thanks to a precise balance between chemical signals that excite and inhibit nerve cell activity. Researchers studying psychiatric disorders have previously suspected that disruption of this balance contributes to schizophrenia.

The first evidence that schizophrenia mutations interfere with excitatory signalling was uncovered in 2011 by the same team, based at Cardiff University's MRC Centre for Neuropsychiatric Genetics and Genomics. This paper not only confirms their previous findings, but also provides the first strong genetic evidence that disruption of inhibitory signalling contributes to the disorder.

To reach their conclusions scientists compared the genetic data of 11,355 patients with schizophrenia against a control group of 16,416 people without the condition. They looked for types of mutation known as copy number variants (CNVs), mutations in which large stretches of DNA are either deleted or duplicated.

Comparing the CNVs found in people with schizophrenia to those found in unaffected people, the team was able to show that the mutations in individuals with the disorder tended to disrupt genes involved in specific aspects of brain function. The disease-causing effects of CNVs are also suspected to be involved in other neurodevelopmental disorders such as intellectual disability, Autism Spectrum Disorder and ADHD.

Around 635,000 people in the UK will at some stage in their lives be affected by schizophrenia. The estimated cost of schizophrenia and psychosis to society is around £11.8 billion a year. The symptoms of schizophrenia can be extremely disruptive, and have a large impact on a person's ability to carry out everyday tasks, such as going to work, maintaining relationships and caring for themselves or others.

The research in Cardiff was funded by the Medical Research Council (MRC) and the European Community's Seventh Framework Programme.

Work carried out by other members of the team based at The Broad Institute of MIT and Harvard was funded by a philanthropic gift to the Stanley Center for Psychiatric Research.

http://www.eurekalert.org/pub_releases/2015-06/uom-pms060215.php

Pluto's moons seen in highest detail yet

New study provides an exciting preview in advance of New Horizons flyby

Much ink has been spilled over Pluto's reclassification as a dwarf planet. And yet, such discussions have not diminished scientific interest in Earth's most distant cousin. A new study is the first to reveal fascinating details about the orbital and rotational patterns of Pluto and its five known moons.

The study, published in the June 4 issue of the journal Nature, describes a system dominated by Pluto and its largest moon, Charon, which together form a 'binary planet.' Four smaller moons orbit this pair. The paper reports the techniques used to discover the two smallest moons, Kerberos and Styx, and also provides a detailed description of the strange and unpredictable rotational states of the two slightly larger moons, Nix and Hydra.

Later this summer, NASA's New Horizons spacecraft will pass by Pluto and its five known moons, providing the most detailed look at this planetary system to date. Kerberos and Styx were discovered in 2011 and 2012, respectively, while Nix and Hydra were first discovered in 2005.

'Like good children, our moon and most others keep one face focused attentively on their parent planet,' said Douglas Hamilton, professor of astronomy at the University of Maryland and a co-author of the Nature study. 'What we've learned is that Pluto's moons are more like ornery teenagers who refuse to follow the rules.'

The imbalanced and dynamically shifting gravitational field created by Pluto and Charon sends the smaller moons tumbling in unpredictable ways. The effect is amplified by the fact that the moons are roughly football shaped, rather than rounded spheres. The findings are the result of a comprehensive analysis of Hubble Space Telescope data regarding the orbits and properties of the four smaller moons.

In contrast to these seemingly random rotational motions, the moons follow a surprisingly predictable pattern as they orbit the binary planet formed by Pluto and Charon. Three of them -- Nix, Styx and Hydra -- are locked together in resonance, meaning that their orbits follow a clockwork pattern of regularity. The same effect can be seen in three of Jupiter's large moons.

'The resonant relationship between Nix, Styx and Hydra makes their orbits more regular and predictable, which prevents them from crashing into one another,' Hamilton said. 'This is one reason why tiny Pluto is able to have so many moons.'

The study also revealed that Kerberos is as dark as charcoal, while the other moons are as bright as white sand. 'This is a very provocative result,' said lead author Mark Showalter, a senior research scientist at the SETI Institute. Astronomers had predicted that dust created by meteorite impacts should coat all the moons evenly, giving their surfaces a uniform look.

'Prior to the Hubble observations, nobody appreciated the intricate dynamics of the Pluto system,' Showalter said. The New Horizons flyby in July may help solve the mystery of Kerberos' dark surface, and will refine scientists' understanding of the odd rotational and orbital patterns uncovered by Hubble. The New Horizons team is using Showalter and Hamilton's discoveries to help guide science planning efforts.

Among other expected insights, a more detailed study of the chaotic Pluto-Charon system could reveal how planets orbiting a distant binary star might behave. Although many exoplanets have been found to orbit binary stars, these star systems are too far away to figure out their rotational patterns using existing technology.

'We are learning that chaos may be a common trait of binary systems,' Hamilton said. 'It might even have consequences for life on planets orbiting binary stars.'

This research was supported by NASA (Award Nos. NNX12AQ11G, NNX14AO40G. NAS5-26555, and NNX12AI80G.). The content of this article does not necessarily reflect the views of this organization.

The research paper, 'Resonant interactions and chaotic rotation of Pluto's small moons,' Mark Showalter and Douglas Hamilton, was published on June 4, in the journal Nature.

http://www.eurekalert.org/pub_releases/2015-06/jhm-ddi060215.php

Developing delirium in the ICU linked to fatal outcomes

Third of patients admitted to an intensive care unit will develop delirium, which may increase one's risk of dying in the hospital

About one-third of patients admitted to an intensive care unit (ICU) will develop delirium, a condition that lengthens hospital stays and substantially increases one's risk of dying in the hospital, according to a new study led by Johns Hopkins Medicine researchers appearing in the British Medical Journal.

"Every patient who develops delirium will on average remain in the hospital at least one day longer," says one of the study's authors, Robert Stevens, M.D., a specialist in critical care and an associate professor at the Johns Hopkins University School of Medicine. Worse, "if you're admitted to the intensive care unit and you develop brain dysfunction, your risk of not surviving your hospital stay is doubled."

Physicians and nurses working in intensive care have long been aware that a significant percentage of their patients develop delirium, a type of brain dysfunction characterized by a sudden onset, fluctuating symptoms, inattention and confusion. However, this study establishes the most definitive link between delirium in the ICU and poor outcomes.

Stevens led an interdisciplinary team of colleagues who sifted through 10,000 published reports before selecting 42 studies that met their specific criteria. For instance, they weeded out any studies that included patients with head injuries, strokes or other neurological disorders to obtain a more precise estimate of delirium in ICU patients.

That left Stevens and his team with 16,595 patients, of which 5,280 -- or 32 percent -- had confirmed cases of delirium as measured by established screening protocols. They conducted a meta-analysis, which found that delirium was associated with a twofold increase in hospital mortality even after adjusting for severity of illness.

One of the best known causes of delirium is medications given to ICU patients, such as sedatives. For instance, benzodiazepine, which is commonly administered to patients to help them calm down and sleep, may paradoxically lead to disorientation and confusion.

The goal moving forward, Stevens says, should be to reduce or eliminate the use of such potentially harmful medications, particularly among higher risk populations, such as the elderly and individuals with dementia. Nighttime interruptions should also be kept to a minimum to ensure that patients get a good night's rest without sedatives.

Other causes of delirium, however, might be harder to address. According to the inflammatory hypothesis, illnesses occurring outside the brain, such as severe pneumonia, can lead secondarily to inflammation in the brain. Another theory posits that delirium is linked to changes in the flow of blood to the brain, sometimes resulting in strokes that are not recognized as such. Intriguingly, Stevens' review also showed that among patients who develop delirium, the risk of long-term cognitive decline increases by 20 to 30 percent.

"We're seeing that even though you may have a very severe illness or injury and you're lucky enough to survive, you're still not quite out of the woods," Stevens says. "We need to think about the measures we can put into place to decrease these long-term burdens."

Additional Johns Hopkins researchers include Han Wang, Eric B. Schneider, Neeraja Nagaraja and Gayane Yenokyan.

http://www.bbc.com/news/health-33007566

Plan for 'global army' of medics

Plans for a global taskforce of 10,000 medics and scientists to tackle major disease outbreaks will be presented at the G7 summit, the BBC understands.

By James Gallagher Health editor, BBC News website

It is a direct response to the biggest ever Ebola outbreak which has infected more than 27,000 people in West Africa. There are also plans to improve disease surveillance and invest more money in drug development. Experts said such measures would have prevented the Ebola outbreak reaching an unprecedented scale.

German Chancellor Angela Merkel holds the presidency of the G7 group of leading nations. Leaders will meet at a summit in Germany on Sunday.

In a newspaper column this week, she said: "We will be discussing how we can be better prepared for such epidemics, how we can prevent them, or at least respond better and faster if they do break out. "The establishment of a worldwide taskforce with a sensible overall concept and adequate funding is undoubtedly a goal for the medium term, but we should be looking at it even now." She has taken advice from Bill Gates, pharmaceutical companies and global health experts.

Documents seen by BBC News include proposals for a global taskforce of 10,000 medics and scientists termed "White Coats" . It would work like an army reserve with people doing their normal jobs, but being ready to be deployed at short notice. It also calls for an autonomous group within the World Health Organization to take responsibility for all outbreaks. There are also proposals to dramatically increase disease surveillance in poor and middle-income countries to prevent outbreaks going unnoticed.

Three disease testing centres would be set up in each target country, mostly in sub-Saharan Africa, with an annual cost of up to £9.7m ($15m). There are further plans to invest up to £65m ($100m) each year to research drugs, tests and vaccines for other threats. This is expected to focus on up to 10 diseases including, Mers-coronavirus, Lassa fever and new strains of flu.

Dr Jeremy Farrar, the director of the Wellcome Trust and one of Ms Merkel's advisors, told the BBC: "We shouldn't underestimate the costs of these events.

"Ebola will be somewhere between five and ten billion dollars, Sars ten years ago will have cost similar amounts. "These are significant costs, the amount of money we would have to spend in order to do the research, to have the surveillance systems in place, and the capacity to respond, would be a fraction of that."

'Massive impact'

Jonathan Ball, prof of virology at the University of Nottingham, commented: "Where the current Ebola epidemic is concerned the global response was inexcusably tardy and the delayed response undoubtedly fuelled the explosive increases in cases towards the end of last year. "Disease surveillance and diagnosis are crucial in identifying outbreaks as soon as they start, and can have a massive impact on controlling infection outbreaks. "These would have prevented the unprecedented spread of Ebola witnessed in West Africa.

"It is difficult to predict where the next virus outbreak will come from, nor what it will be, but preparedness will enable the global community to respond in a timely way and hopefully stamp anything out before it takes a hold - so these are sensible measures."

http://www.eurekalert.org/pub_releases/2015-06/asoa-rbp060115.php

Resuming blood pressure medicine promptly after surgery reduces risk of death

It may be better for patients to resume taking their blood pressure medication sooner after surgery than previously thought.

Chicago - A new study published in the Online First edition of Anesthesiology, the official medical journal of the American Society of Anesthesiologists® (ASA®), found resuming angiotensin receptor blockers (ARBs), common medications used to treat high blood pressure, within two days after surgery decreased death rates in the first month following surgery.

"Sometimes doctors briefly stop ARB medications around the time of surgery because they are known to cause low blood pressure while under general anesthesia, which can be dangerous for the patient," said Susan Lee, M.D., lead author of the study and clinical instructor, department of anesthesia and perioperative care, University of California, San Francisco. "Our study highlights the importance of resuming medications that patients were previously taking at home as soon as it is feasible after surgery."

In the study, researchers examined more than 30,000 patients who were regularly taking ARB medication prior to surgery and were admitted to the Veterans Affairs Healthcare system for non-cardiac surgery between 1999 and 2011.

Nearly one third (10,205) of the patients studied did not have their usual ARB medication resumed within two days of their operation. The delay in restarting ARBs was associated with an increase in death rate within 30-days of surgery, when compared to those whose medication had been promptly resumed. The effect was greater in patients under 60 years old. Researchers also found reduced rates of infection, pneumonia, heart failure and kidney failure in patients whose ARB medications were resumed soon after surgery, suggesting that early resumption may also reduce complications after surgery.

Until now and despite their widespread use, there has been little information to guide physicians in the optimal timing for restarting ARBs after surgery. Doctors may continue to withhold ARB medication after surgery because they are concerned the medication may cause dangerously low blood pressure or disrupt kidney function. However, even after accounting for these complications in the first two days after surgery, resuming ARB medication was associated with a 50 percent lower mortality rate in the first month after surgery.

Immediately following surgery, patients are often transferred to different units within the hospital. Previous research has found that some regularly prescribed medications may not get resumed during these "transitions of care." Last year, the ASA introduced the Perioperative Surgical Home (PSH), a physician-led, patient-centered, multidisciplinary team-based model of coordinated care. With physician anesthesiologists at the helm of the PSH model helping to ensure patient safety and quality of care throughout the entire surgical process - from admission to recovery and post-discharge - the PSH model stands to improve and standardize patient processes like "medication reconciliation," ensuring a patient's medications are resumed appropriately during the surgical process.

http://www.eurekalert.org/pub_releases/2015-06/bu-sd060315.php

Shh! Don't wake the sleeping virus!

Model mimics how dormant infections caused by childhood chicken pox can -- decades later -- trigger the 'rude awakening' of shingles

The red, itchy rash caused by varicella-zoster - the virus that causes chickenpox - usually disappears within a week or two. But once infection occurs, the varicella-zoster virus, or VZV, remains dormant in the nervous system, awaiting a signal that causes this "sleeper" virus to be re-activated in the form of an extremely unpleasant but common disease: herpes zoster, or shingles.

In a study recently published in PLOS Pathogens, scientists at Bar-Ilan University report on a novel experimental model that, for the first time, successfully mimics the "sleeping" and "waking" of the varicella-zoster virus. Based on neurons generated from human embryonic stem cells, and not requiring the use of experimental animals, the model allows scientists to test drugs and develop therapies to prevent shingles. It may also contribute to the fight against other viruses - such as herpes and polio - that target the human nervous system.

A Painful Awakening

"Most adults harbor latent VZV in their nervous system - a 'souvenir' from a bout with childhood chickenpox," says Prof. Ronald Goldstein, a member of BIU's Mina and Everard Goodman Faculty of Life Sciences. "In one-third of people over 50, or in those with weakened immune systems, VZV re-activation triggers the localized rash, itchiness and pain of shingles. In one-third of these cases, however, shingles symptoms are far more serious, causing debilitating pain that can last for months or even years." Goldstein explains that, while an anti-shingles vaccine exists, it provides effective protection in only 50% of cases, and cannot be given to immune-compromised patients - such as transplant recipients - who are at particularly high risk for shingles onset.

The new model - which makes it possible to establish stable, latent-state VZV in neurons derived from human embryonic stem cells, or hESCs - was created by Amos Markus, a PhD student in Goldstein's lab. A major contributor to the model is Prof. Paul "Kip" Kinchington of the Departments of Ophthalmology and of Microbiology and Molecular Genetics at the University of Pittsburgh, with whom Goldstein has been collaborating closely for the past five years. An authority on the genetic modification of VZV, Kinchington made key discoveries about proteins involved in VZV activity.

The significance of this advance is in its potential impact on biomedical research; the model makes it possible to experimentally trigger re-activation of the dormant virus, to characterize the molecular processes involved, and to identify potential targets for shingles-prevention therapies.

"We have now demonstrated hESC-derived neurons can host VZV in its dormant state in a petri dish for a period of up to seven weeks," Goldstein says, adding that dormant infections were achieved using two different methods.

In the first, neurons were exposed to small amounts of viral material together with anti-viral drugs. In the second method, a drug-free micro-fluidic set-up allowed the controlled infection of neural axons, something that more closely mimics the uptake of VZV by the human nervous system in chicken pox.

"Once the infection took place, fluorescent markers allowed us to differentiate between those neurons with an active viral infection, and those in which the virus was present, but was not actively spreading," Goldstein continues. "The green-glowing cells, which were infected with dormant VZV, became our target. Our goal was to break down the cellular defenses that keep VZV quiescent - essentially, to wake up the virus as a way of modeling what happens when latent VZV wakes up, and attacks the body in the form of shingles."

Shingles and Cellular "Shock"

According to Goldstein, shingles is frequently associated with the some immune-compromising, system-shocking event - a linkage he has incorporated into his cell-based, experimental platform.

"Shock causes our bodies' natural defenses to falter - whether the shock is a physical event like surgery, a ski accident, or even an emotional event, like divorce," Goldstein says. "We therefore 'shocked' the dormant virus into action by introducing events that caused the sleeping virus to wake up and become active.

For VZV, this is the first time that such re-activation has been achieved in a laboratory environment."

Some Like it Cool

A key factor in VZV re-activation revealed by the study was the critical role of temperature.

"At first, we had difficulty obtaining a robust re-activation in tissue culture," Goldstein says. "We then remembered that - in both chicken pox and shingles - viral replication takes place in blisters on the surface of the skin, not in internal organs.

To more accurately mimic the re-activation process as it occurs in the body, we cooled our dormantly-infected human neurons down to 34 degrees centigrade - three degrees lower than normal internal body temperature. We found that, under these cooler conditions, VZV re-activation proceeded at a much more rapid pace, with many more neurons affected.

Hitting the Snooze Button

The current model builds on previous work in which Goldstein - a former President of the Israel Stem Cell Society who was the first researcher to successfully coax human embryonic stem cells (hESCs) into generating human peripheral sensory neurons - showed that hESC-derived neurons can host active, infectious VZV.

In these earlier studies, Goldstein also produced microscopic movies showing - for the first time - how VZV rapidly takes up residence within living human neurons in culture, just as it does in children with chicken pox.

Now, by creating an experimental model that mimics the transition between latency and active infection, Goldstein and his colleagues have taken another important step forward.

"We hope to use this model to develop a therapeutic method based on gene editing, which would prevent the virus from waking up and causing shingles," he says. "Such a method could be used in the treatment of patients with elevated shingles risk, such as people whose immunity has been compromised due to trauma, disease, or immunosuppressant therapies."

Goldstein points out that for the past 20 years, chicken pox vaccinations used in the West contain a live virus that can, and has, re-activated to cause shingles. The new model, in addition to providing an experimental platform for the development of a safer VZV vaccine and genetic therapies to prevent shingles, may also be useful for testing drugs and genetic engineering strategies designed to combat any virus that attacks the human nervous system.

The above-described research is supported by the US National Institutes of Health, the Israel Academy of Sciences, the US-Israel Binational Science Foundation.

http://www.medscape.com/viewarticle/845698

Should You Volunteer in a Disaster? Advice for Physicians

Answering the Call

Ingrid G. Hein

This is good for your soul. It's good for your fellow human beings. And nothing is comparable to saving another life.

Peter N. Bretan, Jr, MD, California urologist and transplant surgeon; member of Team Orleans during the Hurricane Katrina disaster in 2002; and founder of LifePlant International, a medical relief group providing education and aid to the Philippines

As the earthquake shook Nepal last month, it also sent a tremor to the core of the North American doctor's altruistic bones. When you have the skills to save lives and there is such an incredible need, it's hard to stand by and watch.

Most nongovernmental organizations (NGOs) are busy with existing teams when new volunteers call in the heat of a crisis. It's easy for a doctor to become frustrated and disappointed. Instincts take over. Why not just go solo and lend a hand?

"Want to throw some penicillin in a backpack and head into the sunset? Think again," says Eileen D. Barrett, MD, governor of the New Mexico chapter of the American College of Physicians and internal chair of the volunteerism committee. Specializing in internal medicine, Dr Barrett has practiced in Thailand; Burma; and most recently, Sierra Leone.

She says that no matter how well intended, medical experts who show up in disaster areas are almost always more of a burden than a help. Physicians need more than first-class medical skills to be of use in a crisis zone.

Dr Barrett has always traveled abroad with an organization, to ensure that she can focus on using her medical skills, with training and a support system to back her up. To care for Ebola patients in West Africa, she attended a training session in Boston, then another given by the World Health Organization in Freetown, Sierra Leone.

Especially for that type of work, "it is only safe to work with established NGOs or governmental organizations," emphasizes Dr Barrett. "Untrained or unaffiliated doctors who arrive spontaneously are usually more of a hindrance than a help because nobody knows who they are, or what they are capable of. They end up needing care themselves, taking valuable trained aid workers away from their work."

Being impulsive about volunteering abroad doesn't help anyone. Dr Barrett compares it with choosing a school, a city, or a specialty. "It's important to first really know who you are and what you want, then do some research to figure out what organization might suit the experience you are looking for."

Start with some simple questions, she suggests, such as:

- ***How much discomfort can you live with?***
- ***Will you be okay taking bucket baths for 2 months?***
- ***If there is no electricity, will you be okay not reaching your family?***
- ***Will you be able to keep your religion to yourself in a community with a different religion?***
- ***Can you work with other people's tools?***
- ***Can you sleep on a very thin mattress for weeks at a time?***

Next, it's about being honest about your skills as a doctor. If your specialty is radiology, you don't want to risk being placed with women who are having babies or with children who have malaria. "These people are excited to finally see a doctor...if you can't deliver, everyone ends up disappointed," said Dr Barrett.

Think seriously about who you are, what you have to offer, and what type of experience you are looking for. Are you looking for an adventure? A vacation? Or are you just medically curious? Do you just want to observe or be of service—ready to pick up a broom? "There is no reason to feel bad if you want to be a tourist, but don't pretend otherwise, even to yourself," she recommends.

Supporting Existing Health Systems

A disaster zone with unaffiliated volunteers is awful, Fahim Rahim, MD confirms, after returning from Nepal. Managing partner of the Idaho Kidney Institute, host of a local radio show called "House Call," and licensed to practice medicine in Nepal, Dr Rahim rallied volunteers and donations, mostly through social media, to help in the aftermath of the earthquake with what he believes to be one of the biggest privately funded relief teams.

By the end of the first week on the ground, Dr Rahim had assembled a unified team of doctors, surgeons, paramedics, and friends from the United States, plus a group of nurses from Canada. Still, he says, the area around the airport was chaotic.

"People didn't know where to start. They were scared to be outside. There was nowhere to pitch a tent, and everything was covered in rubble.

"Fear was a big problem. We experienced at least half a dozen earthquake aftershocks every day, with about a 3.3 magnitude." Dr Fahim had connections in Sindhupalchowk, one of 75 Nepalese districts. His group set up camp by a local hospital, and became their support team.

"It was like a war zone. The 320-bed hospital had more than 1300 patients. There were people recovering on the floor, in the alley, and camping. There were fractures from head to toe, casualties, and sick babies

" The two surgeons were welcomed into the operating room to relieve the doctors who were fatigued from working around the clock.

"The best way to help is being part of the existing system," says Dr Fahim. "Flying solo is disaster tourism. You're better off going home if you're just drinking the water supplies and using the hotels."

Going Where the Needs Are

Gillian Burkhardt, MD, has been globetrotting with relief organizations since finishing her residency. She studied in Cameroon and worked in the Congo while she was a resident. She has worked as an obstetrician/gynecologist in Sierra Leone, Ivory Coast, and Liberia, and was a Peace Corps volunteer in Madagascar. Today, she works with Médecins Sans Frontières (MSF) (also known as Doctors Without Borders) and was recently in Sierra Leone caring for pregnant women with Ebola.

"It's incredibly rewarding," says Dr Burkhardt. "I love obstetric work, delivering babies and dealing with complications. Working in Africa for women's health, we see things that shouldn't happen. Women are dying of things that are unheard of in this country. Providing simple medicine and safe surgery is lifesaving."

The most important part of doing disaster and relief work is to have an open mind, she says.

"You have to be ready to go where the needs are, not just where you want to go. Make sure you find an organization that fits you. Be vigilant; make sure you agree with their mission," she advises. "It's not as daunting as people make it out to be. If you're interested, you can make it happen."

Dr Burkhardt has a true passion for the multicultural work environment. "I like working with people from other countries and cultures, whether it's the host team or the MSF team. Often, I'm one of the only Americans. It's fascinating to see how other doctors approach clinical medicine."

Balancing Altruism With Realistic Expectations

An altruistic sentiment is a good start, but it's not enough. Even the most enthusiastic doctors don't always thrive in disaster zones, or in places where the needs are very different from those in North America.

Roger van Helmond has worked with MSF since 1995 and has been on eight missions. "Each one requires a cultural adaptation," he says. He now works as a recruiter from MSF's New York office, where they recruited 46 American doctors last year. In total, MSF deployed 445 people in 2014.

Their process is rigorous because it has to be. Although 63% of doctors want a second assignment after their first, others don't make it past the first month.

"We had a young physician—a great guy—who went to medical school with his heart set on working for MSF. He did everything to prepare for working with us. He volunteered in Nepal (before the earthquake). He did fundraising for us; he was ready to go."

First-time MSF deployments typically last 9-12 months, and are rarely to disaster zones.

South Sudan is one of MSF's "benchmark" countries, where the organization is well established. New recruits are sent to benchmark countries to experience a conflict zone—epidemics, malnutrition, and refugee camps. "We have many very basic programs there," van Helmond explains.

The young doctor was deployed for his first assignment to South Sudan, and was back after 2 weeks. He explained that he had put MSF on a pedestal; his expectations had been too high. "He was crushed," van Helmond says.

But that has become the exception for MSF. The organization's recruiters try to discover the "soft skills" offered by physicians who are interested in joining the team.

Have you been abroad? Do you have travel experience in developing countries? Are you flexible—can you "go with the flow"? Do you have management and coaching experience? Van Helmond says it's important to be flexible and open to the unknown. "We deal with 109 nationalities. Are you okay in an emergency department, not knowing what will be there? If you are a pediatrician, will you be okay working with adults?"

A 3-day training course is part of the recruitment process. Since establishing the course, says van Helmond, the success rate with candidates has improved. "We get to know each other better, and are able to assess how well the person will fit in a multicultural environment. Sometimes people back out; they find it's not what they expected."

"You have to be level-headed," he explains. "If you have a dispute with the nurse in the operating room, you have to know that you will be sitting down to dinner together that evening and be okay."

Van Helmond says he advises people to stay for at least 2 months before calling it quits. It takes time to get used to the environment and teammates. "Even after my eight assignments, the cultural adaptation is never the same," he says.

Disaster Response Organizations

Hundreds, if not thousands, of organizations provide disaster response, relief, and aid abroad. What follows is a very short list of groups that are looking for physicians to work in medical relief. For a more comprehensive list, check the JAMA Career Center: Volunteer Opportunities or the American Medical Association's list, International Organizations.

Medical Reserve Corps

In the United States, physician volunteers can sign up with the Medical Reserve Corps, a national network of local volunteers who participate in public health initiatives, emergency response, and programs to help build resilience in local communities.

If you are in medical school, take advantage of international residency programs (too numerous to list here). These offer a good introduction to working abroad and in third-world environments, and will help you learn what experiences best suit you.

MSF

MSF was started in 1971 by two doctors who had been volunteering with the Red Cross. They saw a need to band together with other doctors, especially in establishing a knowledge base of war surgery, triage medicine, and education.

Today the organization has programs in about 70 countries. Their motto is, "Go where the patients are."

Although that seems like an obvious mission, conflict situations around the world can make that a difficult task—thus, the name "Doctors Without Borders." MSF "volunteers" actually receive $1731 per month and are provided with everything they need while on a mission, including medical insurance and holidays.

See the MSF website to start the application process, which takes 3-4 months. Applicants must be willing to work anywhere they are needed, and go where assigned. Doctors are assigned to disaster zones only after completing a first mission.

Speaking French is a big plus, but is not mandatory; however, many of the organization's missions are in French-speaking countries. At the time of this writing, an American recruiter said that he had 15 doctors ready to go on their first mission, but only one spoke French. She would be first to get an assignment.

Health Volunteers Overseas

Health Volunteers Overseas is an organization that puts training, education and professional development of a health workforce in resource-scarce countries at the forefront of its mission. Guided by the principle of sensitivity and respect for cultural and social beliefs of the host country, Health Volunteers Overseas focuses on local diseases and health conditions, teaching, prevention, and promotion of lifelong learning.

The placement process has multiple steps, and often it takes several months to be assigned. Programs take place around the globe and range from physical therapy, pediatrics, and oral health to dermatology, hematology, internal medicine, and oncology.

International Medical Corps

Working hard to be the first responders in a an emergency, the International Medical Corps (IMC) works with the community, hires local staff, and develops partnerships at all levels. Their motto is "from relief to self-reliance." Of their

staff of 7800 worldwide, 96% are recruited locally so that the skills to deal with adversity are passed into local hands.

A humanitarian nonprofit organization, IMC was established in 1984 by volunteer doctors and nurses. The organization looks for emergency response, nonmedical, and domestic volunteers. The organization also has a graduate internship program. IMC's emergency response volunteers must be available within 72 hours of being called, for a duration of 2-8 weeks. Preference is given to those who can deploy longer.

Partners in Health

Partners in Health (PIH) works toward making longer-term commitments to disaster zones and rural areas in need, especially to provide an option for the poor. The group builds movements to fight poverty, social injustice, and health inequities.

The organization was founded by Dr Paul Farmer, a physician and anthropologist, and Ophelia Dahl (daughter of novelist Roald Dahl), a strong advocate for rights of the poor, after they met in Haiti in 1983.

When it launched in 1987, PIH delivered healthcare to Haiti's Central Plateau region before the earthquake.

Today they have projects all over the world, including Rwanda, Lesotho, Malawi, Mexico, Russia, and Peru, and they have partnered with the Community Outreach and Patient Empowerment (COPE) project to serve Native Americans in the Navajo Nation in the United States.

Global Disaster Immediate Response Team

Global Disaster Immediate Response Team (DIRT) works in partnership with host countries and the United Nations on disaster response missions.

The organization is well equipped with high-tech equipment for rapid response, and it deploys its medical team with all the support needed to be effective, including a reconnaissance team, communications team, and urban search and rescue team.

Using the Special Force's small unit leadership model, response is provided within 24-48 hours of a disaster.

This NGO has deployed teams to Haiti, Pakistan, New Zealand, Japan, and recently to Nepal. They have ongoing operations in Japan and Haiti, including an emergency medical services project.

DIRT maintains a database of volunteers to contact in case of a disaster. Most of the time, these are short-term deployments, and DIRT calls on those who are highly qualified and skilled for the region needing help.

http://www.eurekalert.org/pub_releases/2015-06/hhmi-yvi060115.php

Your viral infection history in a single drop of blood

Possible to test for all infections with any known human virus by analyzing a single drop of a person's blood

New technology developed by Howard Hughes Medical Institute (HHMI) researchers makes it possible to test for current and past infections with any known human virus by analyzing a single drop of a person's blood. The method, called VirScan, is an efficient alternative to existing diagnostics that test for specific viruses one at a time.

With VirScan, scientists can run a single test to determine which viruses have infected an individual, rather than limiting their analysis to particular viruses. That unbiased approach could uncover unexpected factors affecting individual patients' health, and also expands opportunities to analyze and compare viral infections in large populations. The comprehensive analysis can be performed for about $25 per blood sample.

Stephen Elledge, an HHMI investigator at Brigham and Women's Hospital, led the development of VirScan. "We've developed a screening methodology to basically look back in time in people's [blood] sera and see what viruses they have experienced," he says. "Instead of testing for one individual virus at a time, which is labor intensive, we can assay all of these at once. It's one-stop shopping."

Elledge and his colleagues have already used VirScan to screen the blood of 569 people in the United States, South Africa, Thailand, and Peru. The scientists described the new technology and reported their findings in the June 5, 2015, issue of the journal Science.

VirScan works by screening the blood for antibodies against any of the 206 species of viruses known to infect humans. The immune system ramps up production of pathogen-specific antibodies when it encounters a virus for the first time, and it can continue to produce those antibodies for years or decades after it clears an infection. That means VirScan not only identifies viral infections that the immune system is actively fighting, but also provides a history of an individual's past infections.

To develop the new test, Elledge and his colleagues synthesized more than 93,000 short pieces of DNA encoding different segments of viral proteins. They introduced those pieces of DNA into bacteria-infecting viruses called bacteriophage. Each bacteriophage manufactured one of the protein segments - known as a peptide - and displayed the peptide on its surface. As a group, the bacteriophage displayed all of the protein sequences found in the more than 1,000 known strains of human viruses.

Antibodies in the blood find their viral targets by recognizing unique features known as epitopes that are embedded in proteins on the virus surface. To perform the VirScan analysis, all of the peptide-displaying bacteriophage are allowed to mingle with a blood sample.

Antiviral antibodies in the blood find and bind to their target epitopes within the displayed peptides. The scientists then retrieve the antibodies and wash away everything except for the few bacteriophage that cling to them. By sequencing the DNA of those bacteriophage, they can identify which viral protein pieces were grabbed onto by antibodies in the blood sample. That tells the scientists which viruses a person's immune system has previously encountered, either through infection or through vaccination.

Elledge estimates it would take about 2-3 days to process 100 samples, assuming sequencing is working optimally. He is optimistic the speed of the assay will increase with further development.

To test the method, the team used it to analyze blood samples from patients known to be infected with particular viruses, including HIV and hepatitis C. "It turns out that it works really well," Elledge says. "We were in the sensitivity range of 95 to 100 percent for those, and the specificity was good--we didn't falsely identify people who were negative. That gave us confidence that we could detect other viruses, and when we did see them we would know they were real."

Elledge and his colleagues used VirScan to analyze the antibodies in 569 people from four countries, examining about 100 million potential antibody/epitope interactions.

They found that on average, each person had antibodies to ten different species of viruses. As expected, antibodies against certain viruses were common among adults but not in children, suggesting that children had not yet been exposed to those viruses. Individuals residing South Africa, Peru, and Thailand, tended to have antibodies against more viruses than people in the United States. The researchers also found that people infected with HIV had antibodies against many more viruses than did people without HIV.

Elledge says the team was surprised to find that antibody responses against specific viruses were surprisingly similar between individuals, with different people's antibodies recognizing identical amino acids in the viral peptides. "In this paper alone we identified more antibody/peptide interactions to viral proteins than had been identified in the previous history of all viral exploration," he says. The surprising reproducibility of those interactions allowed the team to refine their analysis and improve the sensitivity of VirScan, and Elledge says the method will continue to improve as his team analyzes more samples. Their findings on viral epitopes may also have important implications for vaccine design.

Elledge says the approach his team has developed is not limited to antiviral antibodies. His own lab is also using it to look for antibodies that attack a body's own tissue in certain autoimmune diseases that are associated with cancer. A similar approach could also be used to screen for antibodies against other types of pathogens.

http://www.eurekalert.org/pub_releases/2015-06/nu-etp060215.php

Eating the placenta: trendy but no proven health benefits and unknown risks

No scientific evidence that it protects against depression, pain or other benefits

Placenta doesn't prevent postpartum depression, ease pain, boost energy or aid lactation

Celebrities spike trend, but no studies show human benefits

Unknown risks to women and babies

CHICAGO -- Celebrities such as Kourtney Kardashian blogged and raved about the benefits of their personal placenta 'vitamins' and spiked women's interest in the practice of consuming their placentas after childbirth.

But a new Northwestern Medicine review of 10 current published research studies on placentophagy did not turn up any human or animal data to support the common claims that eating the placenta -- either raw, cooked or encapsulated -- offers protection against postpartum depression, reduces post-delivery pain, boosts energy, helps with lactation, promotes skin elasticity, enhances maternal bonding or replenishes iron in the body.

More concerning, there are no studies examining the risk of ingesting the placenta, called placentophagy, which acts as a filter to absorb and protect the developing fetus from toxins and pollutants, scientists said.

The study will be published June 4 in Archives of Women's Mental Health.

'There are a lot of subjective reports from women who perceived benefits, but there hasn't been any systematic research investigating the benefits or the risk of placenta ingestion,' said corresponding study author Dr. Crystal Clark. 'The studies on mice aren't translatable into human benefits.'

Clark is assistant professor of psychiatry and behavioral sciences at Northwestern University Feinberg School of Medicine and a psychiatrist specializing in reproduction-related mood disorders at Northwestern's Asher Center for the Study and Treatment of Depressive Disorders.

Placentophagy is an unknown risk for the women who eat it and for their infants, if they are breastfeeding.

'Our sense is that women choosing placentophagy, who may otherwise be very careful about what they are putting into their bodies during pregnancy and nursing, are willing to ingest something without evidence of its benefits and, more

importantly, of its potential risks to themselves and their nursing infants,' said lead author Cynthia Coyle, a Feinberg faculty member and a psychologist.

'There are no regulations as to how the placenta is stored and prepared, and the dosing is inconsistent,' Coyle said. 'Women really don't know what they are ingesting.'

Research is needed to provide the answers, Coyle said. She also hopes the study sparks conversations between women and their physicians about their post-birth plans, so doctors can inform their patients about the science or lack thereof and support patients in their decision-making process.

Clark became interested in placentophagy after some of her pregnant patients asked if eating their placentas would interfere with their antidepressant medications. She was unfamiliar with the practice and began to ask her other patients about it.

'I was surprised that it was more widespread than I anticipated,' Clark said.

Although almost all non-human placental mammals ingest their placenta after giving birth, the first documented accounts of postpartum women practicing placentophagy were in North America in the 1970s, the study reports. In recent years, advocates and the media have popularized health benefits of the practice, and more women are considering it as an option for postpartum recovery.

'The popularity has spiked in the last few years,' Clark said. 'Our sense is that people aren't making this decision based on science or talking with physicians. Some women are making this based on media reports, blogs and websites.'

The authors of this paper are currently gathering data on the perceptions, beliefs and placental practices of health care providers internationally and nationally, as well as patients locally, and whether providers are recommending placentophagy to patients.

Dr. Clark's research is supported in part by grant K12 HD055884 from the Eunice Kennedy Shriver National Institute of Child Health & Human Development of the National Institutes of Health.

http://www.eurekalert.org/pub_releases/2015-06/uoc-rod060115.php

Reprogramming of DNA observed in human germ cells for first time

Some genes that escape reprogramming may contribute to human diseases in subsequent generations

A team of researchers led by the University of Cambridge has described for the first time in humans how the epigenome - the suite of molecules attached to our DNA that switch our genes on and off - is comprehensively erased in early primordial germ cells prior to the generation of egg and sperm. However, the study, published today in the journal Cell, shows some regions of our DNA - including those associated with conditions such as obesity and schizophrenia - resist complete reprogramming.

Although our genetic information - the 'code of life' - is written in our DNA, our genes are turned on and off by epigenetic 'switches'. For example, small methyl molecules attach to our DNA in a process known as methylation and contribute to the regulation of gene activity, which is important for normal development. Methylation may also occur spontaneously or through our interaction with the environment - for example, periods of famine can lead to methylation of certain genes - and some methylation patterns can be potentially damaging to our health. Almost all of this epigenetic information is, however, erased in germ cells prior to transmission to the next generation

Professor Azim Surani from the Wellcome Trust/Cancer Research UK Gurdon Institute at the University of Cambridge, explains: "Epigenetic information is important for regulating our genes, but any abnormal methylation, if passed down from generation to generation, may accumulate and be detrimental to offspring. For this reason, the information needs to be reset in every generation before further information is added to regulate development of a newly fertilised egg. It's like erasing a computer disk before you add new data."

When an egg cell is fertilised by a sperm, it begins to divide into a cluster of cells known as a blastocyst, the early stage of the embryo. Within the blastocyst, some cells are reset to their master state, becoming stem cells, which have the potential to develop into any type of cell within the body. A small number of these cells become primordial germ cells with the potential to become sperm or egg cells.

In a study funded primarily by the Wellcome Trust, Professor Surani and colleagues showed that a process of reprogramming the epigenetic information contained in these primordial germ cells is initiated around two weeks into the embryo's development and continues through to around week nine. During this period, a genetic network acts to inhibit the enzymes that maintain or programme the epigenome until the DNA is almost clear of its methylation patterns.

Crucially, however, the researchers found that this process does not clear the entire epigenome: around 5% of our DNA appears resistant to reprogramming. These 'escapee' regions of the genome contain some genes that are particularly active in neuronal cells, which may serve important functions during development. However, data analysis of human diseases suggests that such genes are associated with conditions such as schizophrenia, metabolic disorders and obesity.

Walfred Tang, a PhD student who is the first author on the study, adds: "Our study has given us a good resource of potential candidates of regions of the genome where epigenetic information is passed down not just to the next generation but potentially to future generations, too. We know that some of these

regions are the same in mice, too, which may provide us with the opportunity to study their function in greater detail."

Epigenetic reprogramming also has potential consequences for the so-called 'dark matter' within our genome. As much as half of human DNA is estimated to be comprised of 'retroelements', regions of DNA that have entered our genome from foreign invaders including bacteria and plant DNA. Some of these regions can be beneficial and even drive evolution - for example, some of the genes important to the development of the human placenta started life as invaders. However, others can have a potentially detrimental effect - particularly if they jump about within our DNA, potentially interfering with our genes. For this reason, our bodies employ methylation as a defence mechanism to suppress the activity of these retroelements.

"Methlyation is effective at controlling potentially harmful retroelements that might harm us, but if, as we've seen, methylation patterns are erased in our germ cells, we could potentially lose the first line of our defence," says Professor Surani.

In fact, the researchers found that a notable fraction of the retroelements in our genome are 'escapees' and retain their methylation patterns - particularly those retroelements that have entered our genome in our more recent evolutionary history. This suggests that our body's defence mechanism may be keeping some epigenetic information intact to protect us from potentially detrimental effects.

http://www.eurekalert.org/pub_releases/2015-06/cp-nso052815.php

New species of horned dinosaur with 'bizarre' features revealed

Nearly intact skull of a very unusual horned dinosaur--a close relative of the Triceratops

About 10 years ago, Peter Hews stumbled across some bones sticking out of a cliff along the Oldman River in southeastern Alberta, Canada. Now, scientists describe in the Cell Press journal Current Biology on June 4 that those bones belonged to a nearly intact skull of a very unusual horned dinosaur--a close relative of the familiar Triceratops that had been unknown to science until now.

"The specimen comes from a geographic region of Alberta where we have not found horned dinosaurs before, so from the onset we knew it was important," says Dr. Caleb Brown of the Royal Tyrrell Museum of Palaeontology in Alberta, Canada. "However, it was not until the specimen was being slowly prepared from the rocks in the laboratory that the full anatomy was uncovered, and the bizarre suite of characters revealed. Once it was prepared it was obviously a new species, and an unexpected one at that. Many horned-dinosaur researchers who visited the museum did a double take when they first saw it in the laboratory."

Brown likes to say, only partly in jest, that the uniqueness of this specimen was so obvious that you could tell it was a new species from 100 meters away.

What made this new horned dinosaur distinctive was the size and shape of its facial horns and the shield-like frill at the back of the skull. This new species is similar in many respects to Triceratops, except that its nose horn is taller and the two horns over its eyes are "almost comically small." But the new dinosaur's most distinctive feature is that frill, including what Brown describes as a halo of large, pentagonal plates radiating outward, as well as a central spike. "The combined result looks like a crown," he says.

This is an artistic life reconstruction of the new horned dinosaur Regaliceratops peterhewsi in the palaeoenvironment of the Late Cretaceous of Alberta, Canada. **Art by Julius T. Csotonyi. Courtesy of Royal Tyrrell Museum, Drumheller, Alberta.**

Brown and study co-author Donald Henderson named the new dinosaur Regaliceratops peterhewsi, a reference to its crown-like frill and to the man who first found and reported it to the museum. Despite the formal name, the scientists say they've taken to calling this dinosaur by the nickname "Hellboy."

While this new dinosaur is intriguing in its own right, Brown and Henderson say what's most significant are the implications for the evolution of dinosaurs' horned ornamentation. It's long been known that horned dinosaurs fall into one of two groups: the Chasmosaurines, with a small horn over the nose, larger horns over the eyes, and a long frill, and the Centrosaurines, characterized by a large horn over the nose, small horns over the eyes, and a short frill.

"This new species is a Chasmosaurine, but it has ornamentation more similar to Centrosaurines," Brown says. "It also comes from a time period following the extinction of the Centrosaurines." Taken together, he says, that makes this the first example of evolutionary convergence in horned dinosaurs, meaning that these two groups independently evolved similar features.

The researchers say they hope to uncover more Regaliceratops peterhewsi specimens. They'll also be working on digital reconstructions of the skull, noting that, though intact, the fossil has been deformed after 70 million years in the Rocky Mountain foothills. "This discovery also suggests that there are likely more horned dinosaurs out there that we just have not found yet, so we will also be looking for other new species," Brown says.

Current Biology, Brown et al.: "A New Horned Dinosaur Reveals Convergent Evolution in Cranial Ornamentation in Ceratopsidae" http://dx.doi.org/10.1016/j.cub.2015.04.041

http://www.eurekalert.org/pub_releases/2015-06/pu-sts060515.php

Study: Top salads with eggs to better absorb vegetables' carotenoids

adding eggs to a salad mixed with a variety of raw vegetables is an effective method to improve the absorption of carotenoids

WEST LAFAYETTE, Ind. -- Adding eggs to a salad with a variety of raw vegetables is an effective method to improve the absorption of carotenoids, which are fat-soluble nutrients that help reduce inflammation and oxidative stress, according to research from Purdue University.

"Eating a salad with a variety of colorful vegetables provides several unique types of carotenoids, including beta-carotene, lutein, zeaxanthin and lycopene," said Wayne Campbell, a professor of nutrition science. "The lipid contained in whole eggs enhances the absorption of all these carotenoids."

This research is published online in the American Journal of Clinical Nutrition and is funded by the American Egg Board-Egg Nutrition Center, National Institutes of Health and Purdue Ingestive Behavior Research Center.

"Most people do not eat enough vegetables in their diets, and at the same time, people are consuming salad dressings that have less fat or are fat-free," said Jung Eun Kim, a postdoctoral researcher in Purdue's Department of Nutrition Science. "Our research findings support that people obtained more of the health-promoting carotenoids from raw vegetables when cooked whole eggs were also consumed. Eggs, a nutrient-rich food containing essential amino acids, unsaturated fatty acids and B vitamins, may be used to increase the nutritive value of vegetables, which are under consumed by the majority of people living in the United States."

In the study, 16 participants consumed a raw mixed-vegetable salad with no eggs, a salad with one and a half eggs, and a salad with three eggs at different times. All salads were served with three grams of canola oil. The second salad had 75 grams of scrambled whole eggs and the third 150 grams of scrambled whole eggs. The absorption of carotenoids was 3.8-fold higher when the salad included three eggs compared to no eggs.

The study used scrambled eggs to make sure the participants consumed both the yolk and egg whites.

"While other egg forms were not tested, we believe the results would be comparable as long as the egg yolk is consumed," said Campbell, whose research also has looked at salads with different amounts of soybean oil, canola oil and butter. "The lipids in salad dressings also increase the absorption of carotenoids but it is easy to overuse salad dressings and consume excess calories. Many salad dressings contain about 140-160 calories per serving, about two tablespoons. One large whole egg is about 70 calories and provides 6 grams of protein. People are at a greater risk of putting too many calories on a salad because they don't always know proper portion sizes for salad dressings, but you do know the portion size of an egg."

American Journal of Clinical Nutrition article "Effects of egg consumption on carotenoid absorption from co-consumed, raw vegetables" can contact Amy Patterson Neubert, Purdue News Service, at 765-494-9723, apatterson@purdue.edu

The study also included Susannah L. Gordon, a graduate student in the Department of Nutrition Science, and Mario G. Ferruzzi, a professor of food science and nutrition science.

http://www.eurekalert.org/pub_releases/2015-06/smh-rth060215.php

Researchers targeting host rather than flu virus have success with new treatment in mice

Study tested drug that acts on the endothelial cells that line the blood vessels

TORONTO - The flu kills hundreds of thousands of people around the world every year, yet there is essentially only one class of drugs to fight the ever-changing virus. Cases of flu resistant to this class of drugs have already been reported and researchers worry a completely new strain of flu could evolve, leading to a pandemic like the one in 1918 that killed approximately 50 million people.

Many researchers are trying to develop new drugs to defeat the flu virus. But researchers at St. Michael's Hospital had a completely different idea.

People who die from the flu actually die from respiratory failure, when the lung's tiny blood vessels start leaking fluid into the lung's air sacs. Dr. Warren Lee, a researcher with the hospital's Keenan Research Centre for Biomedical Sciences, wondered what would happen if someone developed a treatment that would prevent those blood vessels from leaking?

Working with mice, Dr. Lee tested a new drug developed by researchers at Sunnybrook Hospital that acts on the endothelial cells that line the blood vessels.

Their work, published today in the journal Scientific Reports, found that:

The drug, Vasculotide, was effective against multiple strains of influenza, including the 2009 swine flu pandemic strain. Without the drug, 100 per cent of the mice died within one week. With the drug, more than 80 per cent survived. In addition:

The drug worked even if it was administered days after the infection began. Traditional antiviral drugs such as Tamiflu must be started immediately.

The drug worked alone and in combination with antivirals.

It worked without compromising the body's ability to mount an immune response to the virus.

Dr. Lee, a critical care physician and cell biologist, said that while this research was conducted in mice, he found the results exciting since the drug was effective in two different strains of mice and three different strains of flu. He said that since the mechanism of blood vessels leaking into lungs is common throughout animals, he was optimistic the drug could be effective in animals other than mice, including humans.

St. Michael's and Sunnybrook have jointly applied for a U.S. patent for the drug.

This study received funding from the Canadian Institutes of Health Research, the Physicians' Services Incorporated Foundation and a Government of Ontario Early Researcher Award.

http://www.eurekalert.org/pub_releases/2015-06/uof-rvi060515.php

Rabbit virus improves bone marrow transplants, kills some cancer cells

University of Florida Health researchers have discovered that a rabbit virus can deliver a one-two punch, killing some kinds of cancer cells while eliminating a common and dangerous complication of bone marrow transplants.

For patients with blood cancers such as leukemia and multiple myeloma, a bone marrow transplant can be both curative and perilous. It replenishes marrow lost to disease or chemotherapy but raises the risk that newly transplanted white blood cells will attack the recipient's body.

Now researchers say the myxoma virus, found in rabbits, can do double duty, quelling the unwanted side effects of a bone marrow transplant and destroying cancer cells.

The virus could be especially helpful to patients who have recurring cancer but cannot find a suitable bone marrow donor, said Christopher R. Cogle, M.D., the study's lead investigator and an associate professor in the UF College of Medicine's division of hematology and oncology. Bone marrow transplants from partially matched donors carry about an 80 percent risk of graft-versus-host disease, and the myxoma treatment would address that, Cogle said.

The myxoma virus also could improve bone marrow transplant options among African-Americans and the elderly. Those patients are less likely to find fully matched bone marrow donors, which raises the risk of graft-versus-host disease, according to Cogle.

"Myxoma is one of the best strategies because it is effective but doesn't affect normal stem cells," he said.

During laboratory testing on human cells, the process worked this way: The myxoma virus is attached to a type of white blood cell known as a T-cell. The virus-laden white blood cells can then be delivered as part of a bone marrow transplant from a donor. That's when the virus gets activated and goes to work. It blocks graft-versus-host disease, a complication of bone marrow transplants that can cause problems including skin rash, shortness of breath, abdominal pain, jaundice and muscle weakness. In severe cases, these complications can be fatal. The white blood cells then deliver the myxoma virus to cancer cells, which are killed off by the virus.

The findings were published in the April 22 edition of the journal Blood. After successfully testing the process with human cells, researchers are now studying its effectiveness in a mouse model.

The dual action of the myxoma virus is particularly encouraging, said Grant McFadden, Ph.D., a professor in the UF College of Medicine department of molecular genetics and microbiology. It's the first time that a virus has been shown to simultaneously prevent graft-versus-host disease and kill cancer cells in the laboratory, McFadden said.

The process is known to work on blood-related disorders such as multiple myeloma and acute myeloid leukemia but could someday have broader application for other kinds of cancer, he said. The myxoma virus originates among rabbits in Australia and parts of Europe and is benign to humans.

The discovery might never have happened if not for a chance meeting at a coffee kiosk on the health campus. Cogle and McFadden introduced themselves to each other, which led to a collaboration that has lasted seven years.

"It's one of the benefits of a health research campus like UF," Cogle said. "His virus killed the cancer cells that I grew in my lab and spared normal blood stem cells."

Another crucial part of the research team's work was done by Nancy Villa, Ph.D., a research scientist in the division of hematology and oncology. Villa's findings were crucial to understanding and explaining how myxoma prevents graft-versus-host disease, Cogle said.

McFadden credits Villa for finding a way to explain to other scientists how the virus-laden white blood cells can prevent graft-versus-host disease and still be an effective killer of cancer cells. That knowledge will be crucial as the team presses on with its research, McFadden said.

After the initial success with human cells, McFadden is cautiously optimistic that a clinical trial could begin within a year. Before that, researchers need to develop a clinical-grade virus, do safety testing and raise about $1 million for clinical trials.

The UF-owned patent on the myxoma process has been licensed to a Houston-based company, which will seek to raise money for clinical trials, Cogle said.

The research was supported by grants of $1.5 million each from the Florida Bankhead-Coley Cancer Research Program and the National Institutes of Health/National Cancer Institute. Additional financial support came from the Gatorade Trust, which is administered by the department of medicine and the UF Research Foundation.

http://bit.ly/1HdjnfR

‘Female Viagra' to treat low libido gets go-ahead from FDA panel

The first drug for treating low sexual desire in women looks set to go on sale in the US next year

12:12 05 June 2015 by Clare Wilson

Flibanserin, sometimes called the female Viagra, was approved by 18 votes to 6 by a US Food and Drug Administration advisory panel yesterday, although some of the committee members had doubts about the drug's risks and benefits.

They required that certain "risk-management options" be put in place, on top of the usual list of side effects listed in the medicine's patient information leaflet.

We have yet to hear what this means, but options include doctors having to verbally warn women not to drink alcohol or use various other medicines when taking the drug.

The FDA's final say is due by August, but it usually follows the decision of its advisory panel.

Assuming it gets the go-ahead, manufacturer Sprout Pharmaceuticals of Raleigh, North Carolina, plans to give the drug the brand-name Addyi, and has promised not to advertise the product directly to patients – which is normally allowed in the US – for the first 18 months it goes on sale.

Addyi is no Viagra though – women would have to take it every day, whether or not they want sex.

And, while the famous little blue pill works by increasing blood flow to the genitals, this new drug instead alters brain chemistry, affecting receptors for various signalling chemicals including serotonin and dopamine.

Limited effects

Yesterday, the panel members expressed concerns about the drug's side effects: it can cause sleepiness, sudden drops in blood pressure and fainting, especially in combination with alcohol. Yet its effects on sexual desire are limited.

In tests it led to couples having sex – or other "sexually satisfying encounters" – an average of once a month extra, from a baseline of two to three times a month.

The medicine has been controversial because it has been rejected by the FDA twice before, with the agency requesting further trials and safety data.

Sprout has claimed it is sexist that there are several medicines available for treating male impotence, yet none for women with low desire.

That premise is rejected by those such as Cindy Pearson of the National Women's Health Network who urged the FDA panel to reject the drug yesterday.

"The problem is not gender inequity, the problem is the drug," she says.

http://bit.ly/1Ilso59

Stop Calling Flibanserin "Female Viagra"

As the FDA weighs the merits of a new drug to boost female libido, it's clear this is not a little blue pill for women

By Helen Thompson

A drug to treat low libido in women is on track for approval by the U.S. Food and Drug Administration. Yesterday afternoon, an advisory panel voted to recommend approval of the drug in question, flibanserin, Rob Stein reports for *NPR*. While the FDA doesn't have to follow their advice, most of the time they do.

Though some have questioned the drug’s effectiveness, flibanserin has been nicknamed by many media outlets as "Viagra for women." But, there's one key problem with that characterization: The two drugs work in completely different ways.

Viagra works by increasing blood flow to the genitals. In contrast, flibanserin's target lies in the brain, as Clare Wilson reports for *New Scientist*. In animal studies, it increases levels of two neurotransmitter molecules in the brain: dopamine, which controls the brain's reward pathways, and norepinephrine, the hormone that helps the brain focus in stressful situations. At the same time, the drug causes a decrease in levels of serotonin, the so-called "happiness" hormone. Researchers aren't entirely sure why this combination of chemical levels results in an increased libido, but studies suggest that it does.

In fact, the German drug firm Boehringer Ingelheim initially developed the compound as an anti-depressant, but it flopped in that department. When women reported feeling an increase in libido, though, the company refocused their efforts. But, they had trouble showing that the drug had a significant effect, as Amanda Holpuch explains for *The Guardian*. In 2010, the FDA rejected Boehringer Ingelheim's proposal to market flibanserin in the United States, so they dropped the drug. Sprout Pharmaceuticals picked it up and again submitted it for approval in 2013. The FDA nixed it, again.

In clinical trials, women taking the drug reported having one more "satisfying sexual encounter" per month than normal. Given that the drug also comes with side effects like sudden drops in blood pressure, sleepiness and fainting spells, it's unclear whether the benefits outweigh risks, Wilson writes. That's what has kept flibanserin off the market in the past. With the support of women’s groups and politicians, this time around Sprout hopes to change that by recasting the drug as the feminist counterpart to the infamous little blue pill. (Not all women's health groups are on board, though.)

Not only do the two drugs work on entirely different mechanisms, they also treat totally different sexual issues. Viagra and similar drugs for men treat impotency,

often caused by a drop in blood flow to the genital area. It's an easy enough thing to fix. Thus, the drug corrects the issue by simply increasing circulation to the region. Viagra does nothing to increase desire in the brain.

Both men and women can suffer from a loss of libido for due to things like trouble in the relationship, stress, depression, aging and even genes. While roughly 40 percent of women are dissatisfied with their sex lives, the causes might be slightly different in each case. Rather than treating a clear-cut medical problem, the drug aims to correct a hodgepodge of personal and societal issues by tweaking brain chemistry. For that reason, some psychologists argue that chemistry is the wrong approach to treating low sex drives. Instead, therapy might be a better option.

Pharmaceutical companies have long searched for a pill to make women with low libido more sexually excited. But, many times, the search is misguidedly framed by Viagra's success. In fact, Sprout's campaign seems to be built on the idea that women need a libido drug because so many exist on the market for men.

Flibanserin isn't the first "female Viagra" on the block, though. Actually, that was plain, old Viagra. In 2008, research suggested it could help women on antidepressants orgasm. While Viagra does increase circulation in women's' genitals it doesn't actually boost their desire to have sex, as Wilson points out. Studies suggested that a lack of testosterone in the brain might cause low libido in some women, so they tested testosterone patches to increase female arousal. The FDA did not approve.

Back in 2013, Smart News' Colin Schultz wrote about other desire drugs that still being assessed in women experiencing low sexual desire: Lybrido and Lybridos. Lybrido is a combination of testosterone and the main ingredient in Viagra, while Lybridos combines testosterone with an anxiety medication called busiprone.

The FDA advisory committee approved flibanserin by a vote of 18 to 6, with the caveat that Sprout must devise a plan to address safety concerns. Regardless of the drug's fate there's one key thing to take away from its time in the spotlight: Calling it "Viagra for women" suggests a basic misunderstanding of women and of Viagra.

http://bit.ly/1dVaqN7

With This Self-Healing Concrete, Buildings Repair Themselves

A concrete developed by Dutch scientists and embedded with limestone-producing bacteria is ready to hit the market

By Emily Matchar

When you break your leg, it eventually knits itself back together. Osteoblast cells produce minerals that create the structure of new bone, turning fragments back into a whole. Why, thought microbiologist Henk Jonkers, can't buildings do the same thing?

Inspired by the human body, Jonkers, who works at the Delft University of Technology in the Netherlands, created self-healing concrete. He embeds the concrete with capsules of limestone-producing bacteria, either Bacillus pseudofirmus or Sporosarcina pasteurii, along with calcium lactate. When the concrete cracks, air and moisture trigger the bacteria to begin munching on the calcium lactate. They convert the calcium lactate to calcite, an ingredient in limestone, thus sealing off the cracks.

This innovation could solve a longstanding problem with concrete, the world's most common construction material. Concrete often develops micro-cracks during the construction process, explains Jonkers. These tiny cracks don't immediately affect the building's structural integrity, but they can lead to leakage problems. Leakage can eventually corrode the concrete's steel reinforcements, which can ultimately cause a collapse. With the self-healing technology, cracks can be sealed immediately, staving off future leakage and pricey damage down the road. The bacteria can lie dormant for as long as 200 years, well beyond the lifespan of most modern buildings.

Jonkers has been road-testing the self-healing concrete on a lifeguard station, which is by nature prone to wind and water damage. The structure has remained watertight since 2011, he says. The invention has also recently earned Jonkers a nomination for a European Inventors Award, with winners being announced at a June 11 ceremony in Paris.

This year, the technology will hit the market for the first time. It will come as three separate products: self-healing concrete, a repair mortar and a liquid repair medium. Unfortunately, the costs of the technology are still quite high, about €30-40 (about $33-44) per square meter. This means it will initially only be viable for projects where leakage and corrosion are particularly problematic, such as underground and underwater structures. The price of the calcium lactate needed for the bacteria to produce calcite is part of the problem, but Jonkers and his team are working to create a cheaper, sugar-based alternative. And as demand for the concrete increases, the price should decrease.

"We are currently in the process of upscaling its production," says Jonkers. "Our expectation is that we can deliver the healing agent in large quantities [by the] middle of 2016."

Other types of self-repairing concrete are under development around the world. In the UK, researchers at the University of Bath, Cardiff University and Cambridge have developed a material similar to Jonkers' that uses bacteria to fill in crevices, which they hope could be used to repair roads and other infrastructure. They estimate it could reduce costs by up to 50 percent. MIT scientists have been working on a concrete healing system that uses sunlight to activate polymer

microcapsules, which would plug cracks. A University of Michigan engineer has come up with a concrete with microfibers that bends instead of breaking; if tiny trs do occur, the material expands and reinforces itself with calcium carbonate.

Victor Li, the University of Michigan engineer, says the advantage of products like his is that they can actually recover the original load-bearing capacity of the concrete rather than simply filling in the gaps with healing products.

"I expect self-healing concrete to be in use within the next few years," he says.

Concrete production accounts for a massive 5 percent of the world's carbon emissions, and global demand for concrete has doubled over the past decade, largely due to increasing urbanization. So any technology that makes concrete structures longer lasting has the potential not just to cut costs but to reduce our carbon footprint. The future of green building, it seems, may be gray.

http://bit.ly/1MeGCFL

Monkeys' cozy alliance with wolves looks like domestication

In the alpine grasslands of eastern Africa, Ethiopian wolves and gelada monkey are giving peace a chance.

- **17:33 05 June 2015 by Bob Holmes**

The geladas – a type of a baboon – tolerate wolves wandering right through the middle of their troops, while the wolves ignore potential meals of baby geladas in favour of rodents, which they can catch more easily when the monkeys are present. The unusual pact echoes the way dogs began to be domesticated by humans (see box, below), and was spotted by primatologist Vivek Venkataraman, at Dartmouth College in New Hampshire, during field work at Guassa plateau in the highlands of north-central Ethiopia.

Feeling right at home **(Image: Jeff Kerby. Project funding: National Geographic)**

Even though the wolves occasionally prey on young sheep and goats, which are as big as young geladas, they do not normally attack the monkeys – and the geladas seem to know that, because they do not run away from the wolves.

"You can have a wolf and a gelada within a metre or two of each other and virtually ignoring each other for up to 2 hours at a time," says Venkataraman. In contrast, the geladas flee immediately to cliffs for safety when they spot feral dogs, which approach aggressively and often prey on them.

When walking through a troop, the wolves seem to take care to behave in a non-threatening way. They move slowly and calmly as they forage for rodents and avoid the zigzag running they use elsewhere, Venkataraman observed.

Deliberate association

This suggested that they were deliberately associating with the geladas. Since the wolves usually entered gelada groups during the middle of the day, when rodents are most active, he wondered whether the geladas made it easier for the wolves to catch the rodents – their primary prey.

Venkataraman and his colleagues followed individual wolves for 17 days, recording each attempted capture of a rodent, and whether it worked. The wolves succeeded in 67 per cent of attempts when within a gelada troop, but only 25 per cent of the time when on their own.

It's not yet clear what makes the wolves more successful when they hunt within gelada groups. It could be that the grazing monkeys flush out the rodents from their burrows or vegetation, Venkataraman suggests.

Mobile hide

Another possibility is that the monkeys, which are about the same size and colour as the wolves, distract the rodents and make it easier for the wolves to approach undetected. "I like to think of it as a mobile hide," says Claudio Sillero, a conservation biologist at the University of Oxford who studies the critically endangered Ethiopian wolves. "The wolves benefit from hiding in the herd."

Whatever the mechanism, the boost to the wolves' foraging appears to be significant enough that the wolves almost never give in to the temptation to grab a quick gelada snack. Only once has Venkataraman seen a wolf seize a young gelada, and other monkeys quickly attacked it and forced it to drop the infant, then drove the offending wolf away and prevented it from returning later.

The wolves may benefit from associating with other species as well. For example, Sillero has noted that they also tend to forage in the vicinity of herds of cattle, which may help them catch rodents. Other predators might also be doing this without anyone noticing, says Colin Chapman, a primatologist at McGill University in Montreal, Canada. "I don't think we've looked at it very much, because the predators are usually scared off by people. I think it could be pretty common," he says.

Taming man's best friend

Wolves and primates hanging around together, gradually becoming tolerant of one another's presence: that sounds a lot like the first steps in the domestication of dogs by humans.

Dogs were domesticated between 40,000 and 11,000 years ago, and although the process remains shrouded in mystery, one hypothesis is that it started when wolves began following roaming human groups to take advantage of the large carcasses they left behind after hunts.

That may have encouraged other carnivores to keep their distance, offering a benefit for the humans, too. Eventually wolves may have even helped humans hunt better and outcompete other hominins, too.

Could something similar now be happening with Ethiopian wolves and geladas on African highlands?

The gelada case is comparable to what early domestication of dogs might have been like, says Claudio Sillero of the University of Oxford.

However, the geladas don't seem to get anything from the relationship, since the wolves are unlikely to deter other predators such as leopards or feral dogs, he says. Without a reciprocal benefit, Sillero doubts that the relationship could progress further down the road to domestication.

Journal reference: *Journal of Mammalogy, DOI: 10.1093/jmammal/gyu013*

http://bit.ly/1T3ZA79

Why Surgeons Make Catastrophic Mistakes

A new analysis codes the reasons behind mistakes like operating on the wrong leg, or leaving a tool behind in a person's body.

Francie Diep

You've probably heard about the woman who had a sponge left in her body from a hysterectomy four years ago, or the man whose surgeon accidentally implanted a kidney on his left side, instead of his right.

One recent estimate found that catastrophic mistakes—including implanting the wrong thing, or performing the wrong procedure—occur in one out of every 12,000 surgeries in the United States. To figure out why, a team of researchers from the Mayo Clinic in New York decided to analyze botched surgeries at their own clinic in the way investigators do military airplane accidents.

The researchers used an aviation accident-investigation tool called the Human Factors Analysis and Classification System, which helped them pinpoint which human errors are most common in surgeries gone wrong. Their work suggests hospitals should look for ways to reduce the mental lifting that surgery team members must do during a procedure, the researchers write in a paper published last week in the journal *Surgery*. That's in addition to the "systems engineering"-type of solutions that hospitals have recently used to reduce errors, such as installing computer systems that automatically track surgical sponges' whereabouts—and alert people when sponges get left behind in the body. (Sponges are the most common item left behind after surgeries.)

To conduct the study, Mayo researchers collected Human Factors Analysis and Classification System data from debriefings that surgery teams held after catastrophic mistakes. The HFACS checklist includes 161 human errors, grouped into specific categories, that can happen during a procedure. The researchers categorized the causes of 69 botched surgeries this way.

Serious surgical accidents tend to involve many human failures, the researchers found. The average surgery saw nine separate missteps. The most common problems they found fell into the category of the mental conditions of the surgeons and nurses, including overconfidence, and focusing too much on a minute detail and consequently losing sight of the big picture. Another common problem category: "decision errors," like failing to understand the risks of a procedure, or mixing up procedures, tests, and medications that perhaps have similar names. Meanwhile, oversight factors, such as a lack of accountability, and organizational factors—say, a lack of funding—were less likely to be cited as reasons for catastrophic surgery mistakes.

The results suggest surgery team members are cognitively overloaded, the researchers write. That's why they're making these mental mistakes. More complicated procedures and technology, and patients with more complicated health problems, all tax surgeons and nurses, other research has found. That can lead people to perform procedures incorrectly, even when they don't intend to.

In their paper, the researchers don't offer many specific suggestions for improvement, but they did note that lightening the mental load might involve scheduling fewer procedures for staffers, or giving surgical team members more independence, so they don't have to check everything with one overworked supervisor. Fixing human mistakes in surgery, it turns out, likely means making things easier for the humans involved.

http://www.bbc.com/news/health-32938075

'Lab on a card' spots poor quality drugs that can kill

At the Moi Teaching and Research Hospital in Eldoret, Kenya, pharmacists have a "drawer of shame". In it, they put drugs which look suspicious, because they are either fake or of poor quality.

By Philippa Roxby Health reporter, BBC News

Rather than making people better, poor quality medicines prolong their sickness, often cause side-effects and increase the risk of drug resistance - leading to more illness and deaths, particularly among children. While counterfeit medicines are deliberately mislabelled and mis-sold by criminals, poor quality pharmaceuticals are a silent killer because they look genuine.

Mini lab

And it is thanks to a cheap, paper-based screening tool that pharmacist Mercy Maina and her colleagues are able to check on the ingredients of the medicines they prescribe to patients in Eldoret. The tool is known as a PAD (Paper Analytical Device) and is essentially a mini lab on a piece of card, Mercy explains.

"It's simple, you apply the tablet on a specific area on the device, dip the card in water and wait for a colour reaction, then compare the results to a standard to interpret the results."

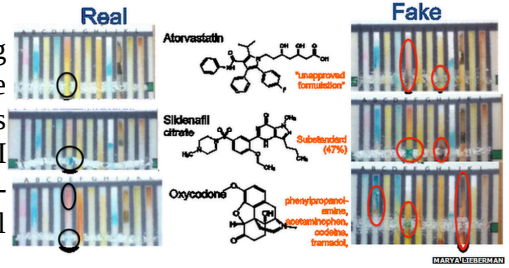
Each of the cards contains 12 separate strips which react with a drug to create a "coloured bar code" that gives information about its chemical content. Using the card, they have been able to determine the quality of a wide variety of antibiotics, anti-malarial drugs and tuberculosis medication, and report any odd results to the Kenya Pharmacy and Poisons Board while filling up their "drawer of shame".

The poor quality drugs may look like the real thing but they don't work properly - usually because of mistakes made during the manufacturing process, poor storage conditions (in hot temperatures) and inadequate transportation.

'Global pandemic'

No-one really knows the scale of the global problem of low-quality medicines because the data is so hard to come by, but experts agree that it is now a serious international public health issue. Research published in the Malaria Journal in 2014 said that poor quality medicines "threaten the lives of millions of patients and are alarmingly common in many parts of the world". Experts writing in the American Journal of Tropical Medicine and Hygiene recently called the proliferation of falsified and sub-standard medicines "a global pandemic". And it is in developing countries, like Kenya, that the problem is most acute. A recent national quality control survey in the country suggested that around 25% of drugs could be sub-standard.

Prof Paul Newton, an expert in drug quality from the Worldwide Antimalarial Resistance Network, says the exact figure doesn't really matter. "I would argue that even if 1% of the anti-malarial supply is poor quality that will be an important public health problem.



The colour strips on the card device allow the ingredients of drugs to be checked for authenticity

"As malaria is so common, many people will be affected with more sickness and death than they should have been if the medicines had been of good quality."

The WHO estimates that 30% of countries have no drug regulation body or one that doesn't function well enough - and that means there is a serious lack of quality control. So out in the field, attention is focused on testing the quality and authenticity of drugs before they are sold to patients.

White powders

Prof Marya Lieberman, from Notre Dame University in the US, who devised the Pad project four years ago, says the device has the ability to test 36 different drugs at the moment. The results have been revealing - they have found drugs diluted with paracetamol, which only relieves symptoms as opposed to fighting off disease. Other drugs have been found to contain hidden amoxicillin (to which people can be dangerously allergic), and tests on fake medicines have unearthed evidence of starch, maize meal and a variety of unidentifiable white powders.

The device can't test all possible fake formulations because some drugs don't contain the right groups of chemicals to test for, but it does have huge potential, Prof Lieberman says. "Fewer than 10% of people are tested for diabetes in developing countries so we are poised to check the quality of these medications when they are prescribed, in a big way."

Because the card is very straightforward to use, anybody can be trained to use it - and that's important in countries with few resources. But testing medicines to find out if they are good quality "is just one component of what needs to be done", says Prof Newton.

'Lack of political will'

So, who is is to blame for the rise in low-quality medicines?

"It is a failure on many fronts," he says, citing the lack of investment in medicine regulation and some pharmaceutical companies not investing in appropriate quality assurance and quality control to ensure that they produce good-quality products. There is also "a lack of political will to ensure that manufactured medicines comply with national and international standards." Manufacturers have also been accused of sloppiness, of sacrificing the quality of the drugs they make to save money.

When pharmaceuticals have such a huge impact on global health and the potential to save millions of lives in developing countries, where malaria and other diseases are rife, low-quality drugs are a hidden killer which scientists are determined to weed out.

http://nyti.ms/1RXAxkE

After Silences and Setbacks, the LightSail Spacecraft Is Revived, Deploying Its Solar Sail

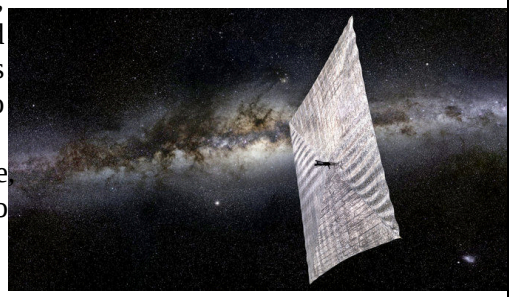
After malfunctions, silences and other unexpected twists, a small experimental spacecraft testing the possibility of harnessing sunlight for propulsion finally did what it was designed to do on Sunday: It unfurled a large, shiny sheet of Mylar.

By KENNETH CHANGJUNE 7, 2015

“It worked,” said William Sanford Nye, chief executive of the Planetary Society, a nonprofit organization promoting space exploration that is operating and

financing the project. Mr. Nye, who is better known as Bill Nye the Science Guy, acknowledged the success did not come easily, calling it an "emotional roller coaster."

Twice since it was launched last month, the LightSail craft fell into unexpected silence, but the team of engineers working on the project managed to revive it.



An artist's rendering of LightSail. Its Mylar sail is currently folded to about the size of a loaf of bread, but it is to be unfurled to a span of almost 345 square feet. Credit Josh Spradling/The Planetary Society

On Sunday, just after 2 p.m. Eastern time, a command was sent to the spacecraft to deploy the sail.

Nothing happened.

For reasons not understood, LightSail ignored the command.

"I was despondent," Mr. Nye said. "I was a little down. This mission has had me down many times."

On the next orbit, about two hours later — the last chance for the day — the team sent the command again. The electric motor that was to extend four 13-foot booms to pull out almost 345 square feet of Mylar started turning.

When the spacecraft passed out of radio range, the tiny motor had turned 67,000 times, halfway to the 134,200 needed to fully deploy the sail. "There was no reason to expect it wouldn't keep going," Mr. Nye said.

On Monday, the spacecraft is to send down photographs to confirm that the sail is spread out.

The technology, using sunlight to traverse the solar system in the same way mariners once crossed oceans in sailing ships, is not a new idea, but it has not been widely used. While particles of light impart only a smidgen of momentum, the force is continuous and provides propulsion without fuel.

LightSail, packed into a box about the size of a loaf of bread, was one of 10 payloads that last month hitchhiked on a rocket that took an unmanned United States Air Force space plane into orbit. LightSail was successfully deployed and worked for two days before its computer crashed because of a software flaw.

Eight days of silence followed until, as engineers expected, a high-speed charged particle zipping through space fortuitously scrambled part of the computer's memory and caused the computer to restart.

With communications re-established, the team began a two-step process of flipping up the solar panels, then extending the sail.

After the solar panels were put in position, the spacecraft appeared to suffer a battery problem, and it fell out of touch for a second time.

David Spencer, a professor at the Georgia Institute of Technology who is the mission manager, said the problem might have been caused by a surge in electrical current when the spacecraft passed from shadow to sunshine.

On Saturday afternoon, the team again made contact with LightSail, and moved quickly to execute the deployment of the sail before anything else could go wrong. The mission should now come to a quick end, probably in two to 10 days.

The orbit of the spacecraft is too low to overcome atmospheric drag and demonstrate actual solar sailing. This flight was intended to wring out issues before a second LightSail is to be launched to a higher orbit next year — and to that extent, it has been successful. The cost of the two LightSail missions is about $5.3 million.

Solar sailing has been a dream for Planetary Society leaders for four decades. Carl Sagan, one of the organization's founders, talked about the idea with Johnny Carson on "The Tonight Show" in 1976.

http://www.eurekalert.org/pub_releases/2015-06/tl-tlo060415.php

The Lancet: Over 95 percent of the world's population has health problems -- with over a third having more than 5 ailments

Just one in 20 people worldwide had no health problems in 2013

Just one in 20 people worldwide (4·3%) had no health problems in 2013, with a third of the world's population (2·3 billion individuals) experiencing more than five ailments, according to a major new analysis from the Global Burden of Disease Study (GBD) 2013, published in The Lancet.

Moreover, the research shows that, worldwide, the proportion of lost years of healthy life (disability-adjusted life years; DALYS [1]) due to illness (rather than death) rose from around a fifth (21%) in 1990 to almost a third (31%) in 2013.

As the world's population grows, and the proportion of elderly people increases, the number of people living in suboptimum health is set to rise rapidly over coming decades, warn the authors.

The findings come from the largest and most detailed analysis to quantify levels, patterns, and trends in ill health and disability around the world between 1990 and 2013.

In the past 23 years, the leading causes of health loss have hardly changed. Low back pain, depression, iron-deficiency anaemia, neck pain, and age-related hearing loss resulted in the largest overall health loss worldwide (measured in terms of YLD--Years Lived with Disability--ie, time spent in less than optimum health [2]) in both 1990 and 2013.

In 2013, musculoskeletal disorders (ie, mainly low back pain, neck pain, and arthritis) and mental and substance abuse disorders (predominantly depression, anxiety, and drug and alcohol use disorders) accounted for almost half of all health loss worldwide.

Importantly, rates of disability are declining much more slowly than death rates. For example, while increases in rates of diabetes have been substantial, rising by around 43% over the past 23 years, death rates from diabetes increased by only 9%.

"The fact that mortality is declining faster than non-fatal disease and injury prevalence is further evidence of the importance of paying attention to the rising health loss from these leading causes of disability, and not simply focusing on reducing mortality," [3] says Theo Vos, lead author and Professor of Global Health at the Institute of Health Metrics and Evaluation, University of Washington, USA.

The GBD 2013 Disease and Injury Incidence and Prevalence Collaborators analysed 35 620 sources of information on disease and injury from 188 countries between 1990 and 2013 to reveal the substantial toll of disabling disorders and the overall burden on health systems from 301 acute and chronic diseases and injuries, as well as 2337 health consequences (sequelae) that result from one or more of these disorders.

Key findings include:

In 2013, low back pain and major depression ranked among the top ten greatest contributors to disability in every country, causing more health loss than diabetes, chronic obstructive pulmonary disease, and asthma combined.

Worldwide, the number of individuals with several illnesses rapidly increased both with age and in absolute terms between 1990 and 2013. In 2013, about a third (36%) of children aged 0-4 years in developed countries had no disorder compared with just 0·03% of adults older than 80 years. Furthermore, the number of individuals with more than ten disorders increased by 52% between 1990 and 2013.

Eight causes of chronic disorders--mostly non-communicable diseases--affected more than 10% of the world population in 2013: cavities in permanent teeth (2·4 billion), tension-type headaches (1·6 billion), iron-deficiency anaemia (1·2 billion), glucose-6-phosphate dehydrogenase deficiency trait (1·18 billion), age-related hearing loss (1·23 billion), genital herpes (1·12 billion), migraine (850 million), and ascariasis (800 million; giant intestinal roundworm).

The number of years lived with disability increased over the last 23 years due to population growth and ageing (537·6 million to 764·8 million), while the rate (age-standardised per 1000 population) barely declined between 1990 and 2013 (115 per 1000 people to 110 per 1000 people).

The main drivers of increases in the number of years lived with disability were musculoskeletal, mental, and substance abuse disorders, neurological disorders, and chronic respiratory conditions. HIV/AIDS was a key driver of rising numbers of years lived with disability in sub-Saharan Africa.

There has also been a startling increase in the health loss associated with diabetes (increase of 136%), Alzheimer's disease (92% increase), medication overuse headache (120% increase), and osteoarthritis (75% increase).

In central Europe, falls cause a disproportionate amount of disability and health burden, ranking as the second leading cause of disability in 11 of 13 countries. In many Caribbean nations anxiety disorders ranked more highly, and diabetes was the third greatest contributor to disability in Mexico, Nicaragua, Panama, and Venezuela. Disability from past war and conflict was the leading contributor to health loss in Cambodia, Nicaragua, Rwanda, and ranked second in Vietnam.

According to Professor Vos, "Large, preventable causes of health loss, particularly serious musculoskeletal disorders and mental and behavioural disorders, have not received the attention that they deserve. Addressing these issues will require a shift in health priorities around the world, not just to keep people alive into old age, but also to keep them healthy." [3]

This study was funded by the Bill & Melinda Gates Foundation.

[1] Years of healthy life lost are measured in terms of disability adjusted life years (DALYS). These are worked out by combining the number of years of life lost as a result of early death and the number of years lived with disability.

[2] Years lived with disability (YLD) calculated by combining prevalence (proportion of the population with the disorder in any given year) and the general public's assessment of the severity of health loss (disability weight).

[3] Quotes direct from author and cannot be found in text of Article.