http://www.bbc.com/news/health-35459054

Scientists get 'gene editing' go-ahead

UK scientists have been given the go-ahead by the fertility regulator to genetically modify human embryos.

By James Gallagher Health editor, BBC News website

It is the first time a country has considered the DNA-altering technique in embryos and approved it. The research will take place at the Francis Crick Institute in London and aims to provide a deeper understanding of the earliest moments of human life. It will be illegal for the scientists to implant the modified embryos into a woman. But the field is attracting controversy over concerns it is opening the door to designer - or GM - babies.

DNA is the blueprint of life - the instructions for building the human body. Gene editing allows the precise manipulation of DNA. In a world-first last year, scientists in China announced they had carried out gene editing in human embryos to correct a gene that causes a blood disorder.

Prof Robin Lovell-Badge, a scientific advisor to the UK's fertility regulator, told the BBC: "China has guidelines, but it is often unclear exactly what they are until you've done it and stepped over an unclear boundary. "This is the first time it has gone through a properly regulatory system and been approved."

Groundbreaking

The experiments will take place in the first seven days after fertilisation. During this time we go from a fertilised egg to a structure called a blastocyst, containing 200-300 cells. The work will be led by Dr Kathy Niakan, who has spent a decade researching human development.

Earlier this year, she explained why she had applied to edit human embryos: "We would really like to understand the genes needed for a human embryo to develop successfully into a healthy baby. "The reason why it is so important is because miscarriages and infertility are extremely common, but they're not very well understood."

Out of every 100 fertilised eggs, fewer than 50 reach the early blastocyst stage, 25 implant into the womb and only 13 develop beyond three months. And at the blastocyst stage, some cells have been organised to perform specific roles - some go on to form the placenta, others the yolk sac and others ultimately us.

How and why this takes place is unknown - but some parts of our DNA are highly active at this stage. It is likely these genes are guiding our early development, but it is unclear exactly what they are doing or what goes wrong in miscarriage. The researchers will alter these genes in donated embryos, which will be destroyed after seven days.

The regulator, the Human Fertilisation and Embryology Authority (HFEA), has given its approval and the experiments could start in the next few months.

Arguments

Paul Nurse, the director of the Crick, said: "I am delighted that the HFEA has approved Dr Niakan's application. "Dr Niakan's proposed research is important for understanding how a healthy human embryo develops and will enhance our understanding of IVF success rates, by looking at the very earliest stage of human development."

Dr David King, the director of Human Genetics Alert, said: "This research will allow the scientists to refine the techniques for creating GM babies, and many of the government's scientific advisers have already decided that they are in favour of allowing that. "So this is the first step in a well mapped-out process leading to GM babies, and a future of consumer eugenics."

Dr Sarah Chan, from the University of Edinburgh, said: "The use of genome editing technologies in embryo research touches on some sensitive issues, therefore it is appropriate that this research and its ethical implications have been carefully considered by the HFEA before being given approval to proceed.

"We should feel confident that our regulatory system in this area is functioning well to keep science aligned with social interests."

http://www.eurekalert.org/pub_releases/2016-02/smh-bsd020116.php

Basic science disappearing from medical journals, study finds

Decline could affect physicians' understanding of and interest in the basic mechanisms of disease and treatments

TORONTO - A new study has found a steep decline in the number of scholarly papers about basic science published in leading medical journals in the last 20 years.

"This rapid decline in basic science publications is likely to affect physicians' understanding of and interest in the basic mechanisms of disease and treatments," warned Dr. Warren Lee, lead author of the study published in the February issue of the FASEB Journal, one of the world's most-cited biology journals.

"If the decline continues, could basic science actually disappear from the pages of specialty medical journals?" asked Dr. Lee, a critical care physician at St. Michael's Hospital and a scientist in its Keenan Research Centre for Biomedical Science.

Basic science is research that examines cells and molecules to better understand the causes and mechanisms of disease. It differs from clinical research, which includes clinical trials of drugs and epidemiological studies that review information from charts and health databases.

Dr. Lee and his team did a search on Pubmed, the main database of medical research, to identify articles on basic science published from 1994 to 2013 in the highest-impact journals in cardiology, endocrinology, gastroenterology, infectious diseases, nephrology, neurology, oncology and pulmonology.

While there was no decline in two of the journals, Diabetes Care and the Journal of the American Society of Nephrology, in the remaining six journals, the amount of basic science fell by 40 to 60 percent. In contrast, there was no decline in the number of basic science articles published in three well-known, non-clinical journals dealing with biological sciences, which Dr. Lee also surveyed-- the Journal of Biological Chemistry, the Journal of Clinical Investigation and Cell.

Dr. Lee said the reasons for the decline in the coverage of basic science articles by medical journals are unclear, but it may be due in part to the fact that papers about clinical research are cited by other researchers more often. The number of times a paper is cited contributes to a journal's "impact factor," which indicates its relative importance.

He said the fading of basic science from medical journals also parallels the rise of other forms of research by clinicians, such as epidemiology and more recently medical education, quality of care, and ethics.

"The decline of basic science in these journals worries me, because if medical residents and clinicians are never exposed to basic science, they are going to think that it's unimportant or irrelevant," Dr. Lee said. "And it has become a bit of a vicious cycle. If residents think that basic science research is irrelevant, they won't consider pursuing it as part of their training or their career. Ironically, scientific advances mean that we are on the threshold of what has been called "precision" or "personalized medicine", where doctors will be able to understand exactly what is wrong with each patient and tailor the therapy accordingly. But all of that depends on understanding the underlying science behind the disease. Scientific discovery forms the underpinning of medical advances, and clinicians and medical students need to be part of that."

http://www.eurekalert.org/pub_releases/2016-02/uops-vge012816.php

Viral gene editing system corrects genetic liver disease in newborn mice

Penn study has implications for developing safe therapies for an array of rare diseases via new gene cut-and-paste methods

PHILADELPHIA - For the first time, researchers have treated an animal model of a genetic disorder using a viral vector to deliver genome-editing components in which the disease- causing mutation has been corrected. Delivery of the vector to newborn mice improved their survival while treatment of adult animals, unexpectedly, made them worse, according to a new study by investigators from the Perelman School of Medicine at the University of Pennsylvania The team published their findings this week in Nature Biotechnology.

"Correcting a disease-causing mutation following birth in this animal model brings us one step closer to realizing the potential of personalized medicine," said senior author James Wilson, MD, PhD, a professor of Medicine and director of the Orphan Disease Center at Penn. "Nevertheless, my 35-year career in gene therapy has taught me how difficult translating mouse studies to successful human treatments can be. From this study, we are now adjusting the gene-editing system in the next phases of our investigation to address the unforeseen complications seen in adult animals." Wilson is also director of the Penn Gene Therapy Program.

The Wilson lab focused on liver as a target for gene editing since they had solved the problem of gene delivery in this organ in previous work using traditional gene therapy using vectors based on adeno-associated virus (AAV). However, gene replacement therapy with AAV is not ideal for treating genetic diseases of the liver that manifest as newborns since the non-integrating genome is lost as developing liver cells proliferate.

Because of this Wilson, co-first author Lili Wang, PhD, a research associate professor of Pathology and Laboratory Medicine, and collaborators, thought that the newborn liver might be an ideal organ for AAV-mediated gene correction using CRISPR-Cas9, an RNA-guided genome-editing technology that uses the bacteria protein Cas9. With CRISPR-Cas9 the corrected mutation will persist as the vector genome is lost.

This hypothesis was tested in a mouse model of a rare metabolic urea-cycle disorder caused by a deficiency in an enzyme called ornithine transcarbamylase (OTC). The urea cycle is a series of six liver enzymes that help rid the body of ammonia, a breakdown product of protein metabolism. When one of these enzymes is missing or deficient, ammonia accumulates in the blood and travels to the brain, causing a multitude of problems, including brain damage and death.

OTC deficiency is the most common of the urea-cycle disorders, occurring in one out of every 40,000 births. A mutated OTC gene can cause an enzyme that is shorter than normal, the wrong shape, or may not be produced at all. The genetic mutation responsible for OTC occurs on the X chromosome, so women are typically carriers, while their sons with the mutated gene suffer the disease.

Cut-and-Paste

The team injected two AAVs (specifically an AAV8 serotype discovered in the Wilson lab that has an affinity for liver cells), one expressing Cas9 and the other expressing a guide RNA and a donor DNA, into newborn mice with OTC deficiency.

One AAV ferried the Cas9 enzyme via a liver-specific promoter to ensure it only expresses in liver cells when injected into the blood. The other AAV in the dual system ferried a guide RNA - a 20-base string of genetic building blocks followed by another sequence to lead the Cas9 enzyme to the correct spot within the DNA in the nucleus of the liver cell. The second AAV also contained a donor DNA template to correct the mutation so that the normal OTC protein can be made by the cell. The addition of this donor DNA to actually correct a mutation distinguishes this study from other recent genome-editing research findings that circumvent a mutation by deleting a portion of the normal gene.

This whole correction system is basically a "Cut-and-Paste" function, with the last part of the "Paste" phase relying on the cells' own DNA repair mechanism to properly join the OTC gene back together again.

In the newborn mice, injection of the AAV system reverted the mutation in 10 percent of liver cells, on average, as measured by the presence of the OTC enzyme in liver cells. They also saw an increased survival in young mice challenged with a high-protein diet, which makes OTC-deficient symptoms worse in the mice.

In contrast, more than 30 percent of the untreated OTC-deficient mice died after a week and their ammonia levels were significantly higher than the OTC mice whose genes were corrected. Deep sequencing of DNA isolated from liver cells in the treated mice also showed that correction to the mutation was consistent with the survival results.

On the other hand, gene correction in adult, eight-to-ten-week-old OTC-deficient mice was lower using the same dual-AAV system. The adults also showed diminished protein tolerance and lethal hyperammonia on a normal chow diet. After three weeks, the adult mice on a low dose of the gene correction started to die, and counterintuitively, mice given a high dose started to die nine days after injection.

"We were surprised by these results, but after some further investigation we deciphered the mechanism by which gene editing worsened the condition of the adult animals," Wang said. Looking at the DNA sequence in liver cells in adult mice, they found that the frequency of cells that had a corrected Paste function was only about one percent. "This was certainly not enough to help these adult mice," Wang noted. What was more problematic, and completely unexpected, is that many of the uncorrected genes contained large deletions that eliminated the residual activity of the mutant OTC gene.

The first step in correcting the gene is the creation of a break in the DNA by Cas9 in proximity to the mutation (the Cut) which, in the presence of the donor DNA, sets the stage for correction of the mutation in what is termed homology directed repair (HDR or the Paste). "It appears that HDR is more efficient in newborn liver cells than in adult liver cells." Wilson said.

In the absence of HDR the cell will repair the cut using another process called non-homologous end joining (NHEJ) that leaves in its wake small insertions or deletions. The team directed the cut to a part of the OTC gene that, if perturbed by a small insertion or deletion, would not interfere with the residual function of the mutant OTC gene. This was the case in newborn mice.

The team learned, however, that NHEJ in adult liver cells resulted in much larger deletions, some of which eliminated any residual function of the OTC gene. The net result of low rates of the Paste with responses to the Cut that destroyed the remaining gene function in many cells resulted in lower tolerance to protein in adult mice.

"The ontoward consequences of gene editing observed in adult OTC mice is limited to treating genetic diseases in which the mutation diminishes but does not eliminate function," Wilson explained. In an attempt to avoid this problem in certain adult patients with liver diseases, the team is exploring methods to create the Cut without inciting the large deletions while at the same time, driving higher frequencies of the Paste.

Yang Yang, PhD, a visiting scientist in the Wilson lab, is also a co-first author.

This research was supported by the National Institute of Child Health and Human Development (P01-HD05727) and the Kettering Family Foundation.

http://www.eurekalert.org/pub_releases/2016-02/uoo-rfo012916.php

Rapid formation of bubbles in magma may trigger sudden volcanic eruptions

It has long been observed that some volcanoes erupt with little prior warning. Now, scientists have come up with an explanation behind these sudden eruptions that could change the way observers monitor active or dormant volcanoes.

Previously, it was thought eruptions were triggered by a build-up of pressure caused by the slow accumulation of bubbly, gas-saturated magma beneath volcanoes over tens to hundreds of years. But new research has shown that some eruptions may be triggered within days to months by the rapid formation of gas bubbles in magma chambers very late in their lifetime.

Using the Campi Flegrei volcano near Naples, southern Italy, as a case study, the team of scientists, from the universities of Oxford and Durham in the UK, and the Vesuvius Volcano Observatory in Italy, demonstrate this phenomenon for the first time and provide a mechanism to explain the increasing number of reported eruptions that occur with little or no warning.

The study is published in the journal Nature Geoscience.

Lead author Mike Stock, from the Department of Earth Sciences at the University of Oxford, said: 'We have shown for the first time that processes that occur very late in magma chamber development can trigger explosive eruptions, perhaps in only a few days to months. This has significant implications for the way we monitor active and dormant volcanoes, suggesting that the signals we previously thought indicative of pre-eruptive activity - such as seismic activity or ground deformation - may in fact show the extension of a dormant period between eruptions.

'Our findings suggest that, rather than seismic activity and ground deformation, a better sign of an impending eruption might be a change in the composition of gases emitted at the Earth's surface. When the magma forms bubbles, the composition of gas at the surface should change, potentially providing an early warning sign.'

The researchers analysed tiny crystals of a mineral called apatite thrown out during an ancient explosive eruption of Campi Flegrei. This volcano last erupted in 1538 but has recently shown signs of unrest.

By looking at the composition of crystals trapped at different times during the evolution of the magma body - and with the apatite crystals in effect acting as 'time capsules' - the team was able to show that the magma that eventually erupted had spent most of its lifetime in a bubble-free state, becoming gas-saturated only very shortly before eruption. Under these conditions of slow magma chamber growth, earthquakes and ground deformation observed at the surface may not be signs of impending eruption, instead simply tracking the arrival of new batches of magma at depth.

Professor David Pyle from the Department of Earth Sciences at the University of Oxford, a co-author of the paper, said: 'Now that we have demonstrated that this approach can work on a particular volcano, and given apatite is a mineral found in many volcanic systems, it is likely to stimulate interest in other volcanoes to see whether there is a similar pattern. 'This research will also help us refine our ideas of what we want to measure in our volcanoes and how we interpret the long-term monitoring signals traditionally used by observers.'

The Campi Flegrei volcano system has had a colourful history. The Romans thought an area called Solfatara (where gas is emitted from the ground) was the home of Vulcan, the god of fire. Meanwhile, one of the craters in the system, Lake Avernus, was referred to as the entrance to Hades in ancient mythology.

Additionally, Campi Flegrei has long been a site of geological interest. In Charles Lyell's 1830 Principles of Geology, he identified the burrows of marine fossils at the top of the Macellum of Pozzuoli (an ancient Roman market building), concluding that the ground around Naples rises and falls over geological time.

http://www.eurekalert.org/pub_releases/2016-02/bumc-rdc020116.php

Researchers develop concept for new sunscreen that allows body to produce vitamin D

Development of a process for altering sunscreen ingredients that does not impact its SPF, but allows the body to produce vitamin D.

Boston - For the first time researchers have developed a process for altering the ingredients in a sunscreen that does not impact its sun protection factor (SPF), but does allow the body to produce vitamin D. The findings, published in the peer reviewed journal PLOS ONE, has led to the production of a new sunscreen called Solar D.

Sun exposure is the major source of vitamin D for most children and adults worldwide. It is also recognized that vitamin D deficiency and insufficiency is a major health problem that afflicts approximately 40 percent of children and 60 percent of adults. However, because of concern for increased risk for skin cancer, widespread sunscreen use has been implemented. As a result, an SPF of 30 when properly applied, reduces the capacity of the skin to produce vitamin D by almost 98 percent

According to the researchers there are several chemical compounds that are typically used in a sunscreen that efficiently absorbed varying wavelengths of UVB radiation. After removing certain ingredients the researchers compared Solar D, which has an SPF of 30, to a popular commercial sunscreen with the same SPF, and found Solar D allowed for up to 50 percent more production of vitamin D in-vitro.

"Solar D was designed with compounds with differing filter compositions to maximize vitamin D production while maintaining its sun protection for reducing erythema or burning of the skin," explained corresponding author Michael F. Holick, PhD, MD, professor of medicine, physiology and biophysics at Boston University School of Medicine and an endocrinologist at Boston Medical Center.

Solar D is currently available in Australia and will be available in the U.S. summer 2016.

Funding for this study was provided by Exposure Scientific, LLC/Nexidus, Ltd, Pty.

http://www.eurekalert.org/pub_releases/2016-02/ohs-utf020116.php

Uncovering the financial ties of advocates for cancer drug approval

Speakers who nominally represent cancer patients at advisory meetings on new drugs often have financial ties with the company seeking marketing approval.

And those ties aren't always disclosed, according to an analysis appearing in JAMA Internal Medicine. "The industry has hijacked that microphone - they're

using it as their second presentation at advisory committee meetings," says senior author Vinay Prasad, M.D., M.P.H., a specialist in blood cancers for the OHSU Knight Cancer Institute, an assistant professor of medicine (hematology and medical oncology) in the OHSU School of Medicine, and senior scholar in the Center for Ethics in Health Care.

Prasad and co-author Matthew Abola, a medical student at Case Western Reserve University School of Medicine, scrutinized the speakers at all 49 meetings of the Food and Drug Administration's Oncologic Drugs Advisory Committee from 2009 to 2014. FDA advisory committees provide independent expertise and technical guidance on new drugs. Their recommendations are not binding but often predict FDA marketing approvals. At meetings, members often open the floor to public comment.

The researchers tallied how many public speakers at the advisory committee meetings were cancer patients and how many had taken the drug under consideration. They also counted how many speakers represented an organization, and how many had a financial association with the maker of the drug, personally or through an organization. They classified each speaker's comments as favorable, neutral or negative toward FDA approval.

More than 90 percent of the speakers - 95 out of 103 - supported marketing approval. And 31 of the 103, or roughly 30 percent, reported financial ties to the maker of the drug, such as financial support for travel to the meeting or representing an organization that received funds from the drug company. Two speakers reported serving as principal investigators of pivotal trials.

In two instances, Prasad and Abola found financial ties that speakers failed to disclose. They discovered through online searches that in those two cases, the speakers represented organizations that had received money from the drug company prior to the meeting.

Close to half of the speakers were patients with the cancer in question, and 31 percent had used the drug in question (32 out of 103). As such, public speakers at meetings of the Oncologic Drugs Advisory Committee bring unique and valuable perspectives not represented by the sponsor, the FDA or expert panel members, Prasad and Abola say.

But they assert that the testimonies should be weighed carefully, considering the extent of drug company funding and influence in determining which patients appear at the hearings.

"Some of the stories are really compelling, but it's a mistake to assume that people who speak at these hearings represent the average patient or express what the average patient wants," Prasad says. "We're likely hearing more of the upsides. Patients who suffered real side effects, they are not the ones able to travel to these meetings."

Only six speakers presented negative opinions. They generally called for better data on the safety and efficacy of the drugs. None of the six speakers reported financial ties.

http://www.eurekalert.org/pub_releases/2016-02/yu-soa020116.php

Sparing ovaries and removing fallopian tubes may cut cancer risk, but few have procedure

Removing both fallopian tubes while keeping the ovaries during hysterectomies may help protect against ovarian cancer and preserve hormonal levels

During hysterectomies for non-cancerous conditions, removing both fallopian tubes while keeping the ovaries may help protect against ovarian cancer and preserve hormonal levels, but few women receive this surgical option, according to a new study by Yale School of Medicine researchers.

Published in the February issue of the journal Obstetrics & Gynecology, the study was led by Xiao Xu, assistant professor in the Department of Obstetrics, Gynecology & Reproductive Sciences at Yale School of Medicine.

In hysterectomies to treat benign conditions, removing both of the ovaries in addition to the fallopian tubes has been used as a way to reduce ovarian cancer risk. But this practice can induce surgical menopause, which adversely affects cardiovascular, bone, cognitive, and sexual health. New evidence suggests that ovarian cancer often originates from the fallopian tube, rather than from the ovaries. This led the American College of Obstetricians and Gynecologists (ACOG) to issue a statement in 2015 suggesting that the practice of bilateral salpingectomy with ovarian conservation -- surgical removal of both fallopian tubes while retaining the ovaries -- may be a better option for ovarian cancer prevention in women at low risk for ovarian cancer.

Xu and her co-author, Vrunda Bhavsar Desai, M.D., conducted the study using data from the 2012 National Inpatient Sample. The team studied 20,635 adult women undergoing hysterectomy for benign conditions who were at low risk for ovarian cancer or future ovarian surgery.

"We found that among women undergoing inpatient hysterectomies in 2012 who were at low risk for ovarian cancer, very few of them received bilateral salpingectomy with preservation of the ovaries," said Xu. "The rate of bilateral salpingectomy with ovarian conservation was 5.9% in this population. This study provides important baseline information on national practice patterns prior to the ACOG recommendation."

Xu added that the rate of bilateral salpingectomy with ovarian conservation varied widely among 744 hospitals across the country, ranging from 0% to 72.2%.

"The wide variation in hospital practice may result in differential access to prophylactic procedures depending on where patients access care," said Xu. "This can have longer-term health implications given the benefits of ovarian conservation." *Citation: doi: 10.1097/AOG.0000000000001203*

http://www.eurekalert.org/pub_releases/2016-02/bumc-rwu020116.php

Researcher warns US could see substantial impact of Zika virus

Virus believed to cause microcephaly in newborns; mild flu-like symptoms in adults, children

BOSTON - A researcher at Boston Medical Center (BMC) and the Boston University School of Public Health (BUSPH) warns that Zika virus could spread quickly to and potentially within the US. The mosquito-borne virus, which is believed to cause microcephaly in infants who are exposed in utero, causes rash and flu-like symptoms in adults and children who have been infected. Zika virus dates back to 1947; however, the first well-documented outbreak in humans was not reported until 2007. An outbreak in French Polynesia in 2013 was responsible for 19,000 suspected cases, and since October 2015, nearly 4,000 cases of Zika virus-related microcephaly have been reported in Brazil. Microcephaly is abnormal smallness of the head, a congenital condition associated with incomplete brain development and a range of neurological complications. The findings are published online in advance of print in the Annals of Internal Medicine.

Zika virus has been rapidly emerging in the Western Hemisphere in the last few months, and as of Jan. 22, 2016, there were 20 countries and territories in the Americas with Zika virus in circulation. Currently, it can be found in Central America, the Caribbean and Mexico, and transmission has occurred in travelers to these areas returning to non-endemic countries including the US, Canada, Japan, Western Europe, and Israel.

"At this time, we believe that Zika virus is primarily transmitted via infected mosquitoes, and therefore people living in or traveling to impacted areas are strongly encouraged to protect themselves against mosquitoes by using an effective insect repellent (containing DEET or picaridin)," said senior author Davidson Hamer, MD, director of the Travel Clinic at BMC, and professor of global health and medicine at the Boston University School of Public Health and School of Medicine. "However, there is some evidence to suggest that Zika virus could be transmitted via blood transfusion and sexual activity, so researchers are trying to determine if these are meaningful pathways to transmission."

There is also evidence of mother-to-child transmission, which appears to be responsible for the surge in cases of microcephaly being seen in Brazil.

Hamer and his co-author, Lin Chen, MD, of the Mt. Auburn Hospital Travel Clinic, say there is substantial risk of introduction of the Zika virus in the US given the presence of the mosquito species that carry the virus, Aedes aegypti and Ae. albopictus, in many states. While people in the US shouldn't panic, he said they should be aware and vigilant.

"If you are pregnant, put off travel to the endemic areas," Hamer said. "If you absolutely must go, be sure to protect yourself against mosquitoes. For those who are not pregnant, it's still a good idea to delay travel so that you don't risk getting infected and transferring the virus back home - there are many unknowns about its transmission, so there is still a risk."

In 2007, the first case was detected in a human, leading to an outbreak on an island in Micronesia. An estimated 73 percent of the island residents age 3 or older became infected, however, about 80 percent of these cases did not present significant symptoms.

Zika virus is generally mild and typically resolves itself within a week. Symptoms can include rash, conjunctivitis, muscle and joint pain, headache, joint swelling, dizziness and vomiting. However, neurological and autoimmune complications have been linked to the French Polynesia outbreak, particularly development of Guillain-Barre syndrome, a neurological illness that may result in temporary paralysis. Microcephaly has been reported in thousands of cases in Brazil, and recently in a newborn in Hawaii.

There currently is no vaccine or cure for the Zika virus.

http://www.eurekalert.org/pub_releases/2016-02/w-dcm020116.php

Drinking coffee may reduce the risk of liver cirrhosis

Regular consumption of coffee was linked with a reduced risk of liver cirrhosis in a review of relevant studies published before July 2015.

In patients with cirrhosis, the liver becomes scarred often as a result of long-term and persistent injury from toxins like alcohol and viruses like hepatitis C. It can be fatal because it increases the risk of liver failure and cancer.

The analysis found that an extra 2 cups of coffee per day may reduce the risk of cirrhosis by 44%, and it may nearly halve the risk of dying from cirrhosis.

"Coffee appeared to protect against cirrhosis. This could be an important finding for patients at risk of cirrhosis to help to improve their health outcomes," said Dr. O. J. Kennedy, lead author of the Alimentary Pharmacology and Therapeutics analysis. "However, we now need robust clinical trials to investigate the wider benefits and harms of coffee so that doctors can make specific recommendations to patients."

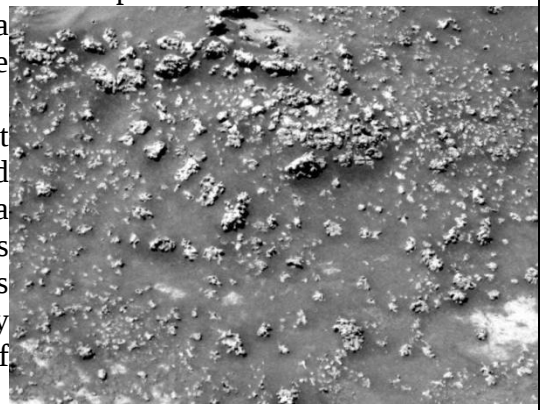
http://bit.ly/1PEqsdf

Mysterious Martian "Cauliflower" May Be the Latest Hint of Alien Life

Unusual silica formations spotted by a NASA rover look a lot like structures formed by microbes around geysers on Earth

By Sarah Scoles

The hunt for signs of life on Mars has been on for decades, and so far scientists have found only barren dirt and rocks. Now a pair of astronomers thinks that strangely shaped minerals inside a Martian crater could be the clue everyone has been waiting for.



In 2008, scientists announced that NASA's Spirit rover had discovered deposits of a mineral called opaline silica inside Mars's Gusev crater. That on its own is not as noteworthy as the silica's shape: Its outer layers are covered in tiny nodules that look like heads of cauliflower sprouting from the red dirt.

A picture snapped by Spirit near Home Plate shows silica formations poking out of the soil, which may have been formed by microbial life. **(NASA/JPL-Caltech)**

No one knows for sure how those shapes—affectionately called "micro-digitate silica protrusions"—formed. But based on recent discoveries in a Chilean desert, Steven Ruff and Jack Farmer, both of Arizona State University in Tempe, think the silica might have been sculpted by microbes. At a meeting of the American Geophysical Union in December, they made the case that these weird minerals might be our best targets for identifying evidence of past life on Mars.

If the logic holds, the silica cauliflower could go down in history as arguably the biggest discovery ever in astronomy. But biology is hard to prove, especially from millions of miles away, and Ruff and Farmer aren't claiming victory yet. All they're saying is that maybe these enigmatic growths are mineral greetings from ancient aliens, and someone should investigate.

Spirit found the silica protrusions near the "Home Plate" region of Gusev crater, where geologists think hot springs or geysers once scorched the red planet's surface. To understand what that long-dormant landscape used to be like, we have to look closer to home: hydrothermal regions of modern Earth that resemble Mars in its ancient past.

To that end, Ruff has twice in the past year trekked to Chile's Atacama Desert, a high plateau west of the Andes cited as the driest non-polar place on Earth. Scientists often compare this desert to Mars, and not just poetically. It's actually *like* Mars. The soil is similar, as is the extreme desert climate.

In this part of the Atacama, it rains less than 100 millimeters per year, and temperatures swing from -13°F to 113°F. With an average elevation of 13,000 feet above sea level, lots of ultraviolet radiation makes it through the thin atmosphere to the ground, akin to the punishing radiation that reaches the surface of Mars.

Just as we interpret others' behavior and emotions by peering into our own psychology, scientists look around our planet to help them interpret Mars, find its most habitable spots and look for signs of life. While the Atacama does have breathable oxygen and evolutionarily clever foxes (which Mars does not), its environment mimics Mars's pretty well and makes a good standin for what the red planet may have been like when it was warmer and wetter.

So when geologists see something in the Atacama or another Mars analog that matches a feature on the red planet, they reasonably conclude that the two could have formed the same way. It's not a perfect method, but it's the best we've got.

"I don't think there is any way around using modern Earth analogs to test where Martian microbes may be found," says Kurt Konhauser of the University of Alberta, who is the editor-in-chief of the journal *Geobiology*.

To understand Home Plate, it makes sense that Ruff turned to El Tatio, a region in the Atacama that is home to more than 80 geysers. While most other earthly animals wouldn't last long here, many microbes do just fine, and fossil evidence suggests they also thrived in the distant past. By inference, Mars's Home Plate might have once made a nice microbial home.

But the comparison goes further: When Ruff peered closely at El Tatio's silica formations, he saw shapes remarkably similar to those that Spirit had seen on Mars. Fraternal cauliflower twins also exist in Yellowstone National Park in Wyoming and the Taupo Volcanic Zone in New Zealand. In both of those places, the silica bears the fossilized fingerprints of microbial life.

Since microbes sculpted the silica features in Wyoming and New Zealand, it's possible they also helped make the formations at El Tatio. And if microbes were involved with the cauliflower at El Tatio, maybe they made it grow on Mars, too.

http://www.eurekalert.org/pub_releases/2016-02/cu-fts020116.php

Flu tackles Super Bowl fans

If you're a fan of the Panthers or Broncos, be sure to wash your hands on Super Bowl Sunday before you give a friend a celebratory fist bump.

ITHACA, N.Y. - A Cornell University economist and his colleagues have found the geographical areas that have an NFL team advance to the Super Bowl had an 18 percent spike in flu-related deaths among people above the age of 65.

"The mechanism that's driving this is the increased socialization that happens as a result of the team being successful," says Nicholas Sanders, assistant professor of economics in the Department of Policy Analysis and Management at Cornell University.

"You have friends over for a Super Bowl party. You all go out to a bar to watch the game. A bunch of people are cramped in a small space, and they're all touching the same napkins and grabbing the same chips. If your team wins, you're all out in the street celebrating. It's that kind of disease transmission that we think might be a driving factor," he said.

The results were most pronounced in years when the dominant influenza strain is more virulent, or when the Super Bowl occurs closer to the peak of influenza season, he added.

The Super Bowl offered a perfect natural experiment to test the researchers' central question: how does a change in people's daily interactions - such as increased travel and social gatherings - affect the way a disease spreads?

The researchers analyzed county-level data from 1974 to 2009, comparing the rates of influenza-related death in areas that had an NFL team in the Super Bowl to the rates in places that also had football teams but did not reach the big game that year. The researchers focused on mortality for those over the age of 65, historically the most vulnerable population.

Although older adults may not change their habits much if their local team makes it to the Super Bowl, their chances of coming into contact with someone who has the flu increase as the infection rate climbs in the overall population, Sanders said.

"It needn't be a direct leap, where an older person is at a bar watching the team. It could be that individual's relative is at a bar and then he visits his parents. Or a worker at a retirement home goes out to get a drink and celebrate her team's win, and then returns to work the next day. Those are all possible disease transmissions," Sanders said.

But the researchers found no change in influenza mortality in cities that hosted the Super Bowl. That could be for several reasons, Sanders said, including because the influx of travelers may prompt locals to avoid going out on the town. Another factor could be the host city's location; the Super Bowl is frequently held in southern cities, where flu transmission rates are generally lower.

Sanders and his colleagues point out that flu prevention techniques apply whether it's Super Bowl Sunday or not: wash your hands, don't share food or drinks. If you are sick, avoid social gatherings.

"Simply being aware of the situation can make people take common-sense precautions, and say, 'Well, I'm not going to shove my hand in that bowl of nuts over there.' I think that's just good advice in general," Sanders said.

The study, "Success Is Something to Sneeze At: Influenza Mortality in Cities that Participate in the Super Bowl," appears in the winter 2016 issue of American Journal of Health Economics. Sanders' co-authors are Charles Stoecker and Alan Barreca, both economists at Tulane University.

http://www.bbc.com/news/uk-35471624

People aged 65 to 79 'happiest of all', study suggests

Sixty-five to 79 is the happiest age group for adults, according to Office for National Statistics research.

The survey of more than 300,000 adults across the UK found life satisfaction, happiness and feeling life was worthwhile all peaked in that age bracket, but declined in the over-80s. Those aged 45 to 59 reported the lowest levels of life satisfaction, with men on average less satisfied than women. That age group also reported the highest levels of anxiety.

Researchers said one possible reason for the lower happiness and well-being scores among this age group might be the burden of having to care for children and elderly parents at the same time. The struggle to balance work and family commitments might also be a factor, they said. Meanwhile, those who were younger or retired had more free time to spend on activities which promoted their well-being, the researchers suggested.

Happiness and well-being dropped off again in those over 80, however, with researchers suggesting this could be down to personal circumstances such as poor health, living alone and feelings of loneliness.

The survey asked people to rate out of 10 how happy and how anxious they had felt the day before, how satisfied they were with their life generally, and how much they felt what they did in life was worthwhile. The published results have been broken down by age, ethnicity, religion, marital status, employment status, religion, and where in the country people live.

They suggested:

- ***Married people had the highest levels of happiness, averaging 7.67 out of 10, higher than co-habiting, single, widowed or divorced people***
- ***People with jobs were happier than unemployed people, with part-time workers the happiest. Of those are were not working, retirees had the highest levels of happiness, followed by students***
- ***Of those who followed a religion, Hindus were marginally the happiest on average, followed by Christians and Sikhs, while those who followed no religion were the least happy***
- ***Women on average reported higher levels of anxiety than men, but were more likely to report better well being and feel their life was worthwhile***
- ***People of Arab ethnicity were found to be the most anxious ethnic group, with people of Chinese ethnicity the least anxious***

- ***Northern Ireland held on to the crown for happiest of the UK's nations, with people there also most satisfied and most likely to say their life was worthwhile - but also the most anxious; the least happy people were in England, with the North East the unhappiest region***

Take the test: Where in Britain would you be happiest?

Research shows the better you fit into the personality of your area, the happier you are. Take the test to find the best place in Britain for you

Researchers found a strong link between health and well-being. People who said their health was very good reported an average life satisfaction rating of 8.01 out of 10, compared with people who said they were in very bad health, whose average rating was just 4.91.

Ageing population

The over-90 age group reported by far the lowest levels of feeling their life was worthwhile, even though their reported levels of happiness and life satisfaction were comparable to those in their 20s and 30s.

Understanding how people of different ages rated their personal well-being could help policy makers target issues to improve lives, the study added.

"We know that the UK population is ageing. There were more than half a million people aged 90 and over living in the UK in 2014 - almost triple the number 30 years ago," it said.

"This shift towards an older population will impact on important policies and services including the labour market, pension provision, and health and social care demand.

"Understanding more about how the oldest age groups rate their personal well-being will help focus on issues that are fundamental to a good later life."

Happiness around the world

The "U-shaped" pattern of happiness, which sees people's happiness dip in middle age, has been observed globally.

- ***It has been documented in more than 70 countries, in surveys of more than 500,000 people in developing and developed countries, although the age at which happiness is lowest differs between countries.***
- ***Previous studies found happiness hits rock bottom at 35.8 years in UK; the low point in the US comes a decade later; in Italy, happiness is lowest at 64.2 years***
- ***US citizens have become less happy with each passing decade since 1900; in Europe, happiness declined until 1950 and has been increasing steadily ever since***
- ***Women are at their least happy at 38.6 years on average; males hit low point at 52.9 years***
- ***Apes, like humans, may also suffer from midlife melancholy - that's according to a study of 508 apes in which their human care-givers assessed their well-being***

Neuroscientist Tali Sharot explains how happiness changes with age

http://www.eurekalert.org/pub_releases/2016-02/b-dn020216.php

'Schizophrenia' does not exist, argues expert

Disease classifications should drop this unhelpful description of symptoms

The term "schizophrenia," with its connotation of hopeless chronic brain disease, should be dropped and replaced with something like "psychosis spectrum syndrome," argues a professor of psychiatry in The BMJ today.

Professor Jim van Os at Maastricht University Medical Centre says several others have called for updated psychiatric classifications, particularly regarding the term "schizophrenia." Japan and South Korea have already abandoned this term.

The official list of mental disorders that doctors use to diagnose patients is found in ICD-10 (International Classification of Diseases, 10th revision) and DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, fifth edition).

But Professor van Os argues that the classification is complicated, particularly for psychotic illness. Currently, psychotic illness is classified among many categories, including schizophrenia, schizoaffective disorder, delusional disorder, depression or bipolar disorder with psychotic features, and others, he explains.

But categories such as these "do not represent diagnoses of discrete diseases, because these remain unknown; rather, they describe how symptoms can cluster, to allow grouping of patients."

This allows clinicians to say, for example, "You have symptoms of psychosis and mania, and we classify that as schizoaffective disorder." If your psychotic symptoms disappear we may reclassify it as bipolar disorder. If, on the other hand, your mania symptoms disappear and your psychosis becomes chronic, we may re-diagnose it as schizophrenia.

"That is how our classification system works. We don't know enough to diagnose real diseases, so we use a system of symptom based classification."

If everybody agreed to use the terminology in ICD-10 and DSM-5 in this fashion, there would be no problem, he says. However, this is not what is generally communicated, particularly regarding the most important category of psychotic illness: schizophrenia.

For example, the American Psychiatric Association, which publishes the DSM, on its website describes schizophrenia as "a chronic brain disorder," and academic journals describe it as a "debilitating neurological disorder," a "devastating, highly heritable brain disorder," or a "brain disorder with predominantly genetic risk factors." This language is highly suggestive of a distinct, genetic brain disease, writes van Os. Yet strangely, no such language is used for other categories of psychotic illness, even though they constitute 70% of psychotic illness.

Scientific evidence indicates that the different psychotic categories can be viewed as part of the same spectrum syndrome, he adds. However, people with this

psychosis spectrum syndrome display extreme diversity (heterogeneity), both between and within people, in psychopathology, treatment response, and outcome. He believes that the best way to inform the public and provide patients with diagnoses, is to forget about "devastating" schizophrenia as the only category that matters "and start doing justice to the broad and heterogeneous psychosis spectrum syndrome that really exists."

And he argues that ICD-11 should remove the term "schizophrenia."

http://www.eurekalert.org/pub_releases/2016-02/hzm--eoh020216.php

Effects on HIV and Ebola

Cell culture experiments reveal potent antiviral activity of Cistus incanus

Neuherberg - Scientists at the Helmholtz Zentrum München discover that extracts of the medicinal plant Cistus incanus (Ci) *ゴジアオイ属* prevent human immunodeficiency viruses from infecting cells. Active antiviral ingredients in the extracts inhibit docking of viral proteins to cells. Antiviral activity of Cistus extracts also targets Ebola- and Marburg viruses. The results were published in Scientific Reports*.

Virus infections are among the ten leading causes of death worldwide and represent a major global health challenge. Their control requires the continuous development of new and potent antiviral drugs/therapeutic options. Despite the availability of numerous drugs for chronic treatment of HIV/AIDS, new drugs are needed to prevent the emergence of drug resistant viral variants. Furthermore, new antiviral drugs are required for rapid treatment of acute infections by viruses like Marburg and Ebola viruses during acute viral outbreaks. A recent study by the team of Professor Ruth Brack-Werner and Dr. Stephanie Rebensburg from the Institute for Virology (VIRO) of the Helmholtz Zentrum München demonstrates that extracts of the medicinal plant attack HIV and Ebola virus particles and prevent them from multiplying in cultured cells.

HIV: broad activity, no resistance

The Brack-Werner team found potent activity of Ci extracts acted against a broad spectrum of clinical HIV-1 and HIV-2 isolates. This also included a virus isolate resistant against most available drugs. „Antiviral ingredients of Ci extracts target viral envelope proteins on infectious particles and prevent them from contacting host cells", Brack-Werner explains. No resistant viruses were detected during long-term treatment (24 weeks) with Ci extract, indicating that Ci extract attacks viruses without causing resistance. The Brack-Werner study suggests that commercial herbal extracts from plants like Cistus incanus*or other plants like Pelargonium sidoides *テンジクアオイ属*** are promising material for the development of scientifically validated antiviral phytotherapeutics. „Since antiviral activity of Ci extracts differs from all clinically approved drugs, Ci-derived products could be an important complementation to current established drug regimens", says Brack-Werner.

Antiviral activity of Cistus extracts also targets Ebola and Marburg proteins in virus particles

Ci extracts not only blocked different HIV isolates, but also virus particles carrying Marburg and Ebola viral envelope proteins. Analysis of the antiviral components of the extract revealed the presence of multiple antiviral ingredients that may act in combination. These results firmly establish broad antiviral activity of Ci extracts against various major human viral pathogens, including previously reported activity against influenza viruses.

Potential applications of Ci extract for global control of lethal virus infections

Further development of these plant extracts may advance global treatment and control of virus infections in various ways. Thus these plant extracts may be useful starting material for the development of potent herbal agents against selected virus infections. Another application could be their development into crèmes or gels (i.e. microbicides) that prevent transmission of viruses like HIV during sexual intercourse. Finally, these plant extracts represent promising collections of natural antiviral agents for the discovery of new antiviral molecules. Future work in the Brack-Werner lab will focus on investigating the antiviral potential of these plant-derived products for applications in humans and detailed analysis of their active antiviral ingredients.

Original Publications:

**Rebensburg, S. et al. (2016) Potent in vitro antiviral activity of Cistus incanus extract against HIV and Filoviruses targets viral envelope proteins. Scientific Reports, doi: 10.1038/srep20394*

***Helfer, M. et al. (2014) The root extract of the medicinal plant Pelargonium sidoides is a potent HIV-1 attachment inhibitor. PLOS ONE, doi: 10.1371/journal.pone.0087487*

http://www.eurekalert.org/pub_releases/2016-02/uosc-cdc020216.php

Carbon dioxide captured from air converted directly to methanol fuel for the first time

Research could one day create a sustainable fuel source from greenhouse gas emissions

They're making fuel from thin air at the USC Loker Hydrocarbon Research Institute.

For the first time, researchers there have directly converted carbon dioxide from the air into methanol at relatively low temperatures.

The work, led by G.K. Surya Prakash and George Olah of the USC Dornsife College of Letters, Arts and Sciences, is part of a broader effort to stabilize the

amount of carbon dioxide in the atmosphere by using renewable energy to transform the greenhouse gas into its combustible cousin - attacking global warming from two angles simultaneously. Methanol is a clean-burning fuel for internal combustion engines, a fuel for fuel cells and a raw material used to produce many petrochemical products.

"We need to learn to manage carbon. That is the future," said Prakash, professor of chemistry and director of the USC Loker Hydrocarbon Research Institute.

The researchers bubbled air through an aqueous solution of pentaethylenehexamine (or PEHA), adding a catalyst to encourage hydrogen to latch onto the CO2 under pressure. They then heated the solution, converting 79 percent of the CO2 into methanol. Though mixed with water, the resulting methanol can be easily distilled, Prakash said.

The new process was published in the Journal of the American Chemical Society on Dec. 29. Prakash and Olah hope to refine the process to the point that it could be scaled up for industrial use, though that may be five to 10 years away.

"Of course it won't compete with oil today, at around $30 per barrel," Prakash said. "But right now we burn fossilized sunshine. We will run out of oil and gas, but the sun will be there for another five billion years. So we need to be better at taking advantage of it as a resource."

Despite its outsized impact on the environment, the actual concentration of CO2 in the atmosphere is relatively small - roughly 400 parts per million, or 0.04 percent of the total volume, according to the National Oceanographic and Atmospheric Administration. (For a comparison, there's more than 23 times as much the noble gas Argon in the atmosphere - which still makes up less than 1 percent of the total volume.)

Previous efforts have required a slower multistage process with the use of high temperatures and high concentrations of CO2, meaning that renewable energy sources would not be able to efficiently power the process, as Olah and Prakash hope.

The new system operates at around 125 to 165 degrees Celsius (257 to 359 degrees Fahrenheit), minimizing the decomposition of the catalyst - which occurs at 155 degrees Celsius (311 degrees Fahrenheit). It also uses a homogeneous catalyst, making it a quicker "one-pot" process. In a lab, the researchers demonstrated that they were able to run the process five times with only minimal loss of the effectiveness of the catalyst.

Olah and Prakash collaborated with graduate student Jotheeswari Kothandaraman and senior research associates Alain Goeppert and Miklos Czaun of USC Dornsife. Their research was supported by the USC Loker Hydrocarbon Research Institute, and their paper can be found online here: http://pubs.acs.org/doi/abs/10.1021/jacs.5b12354

http://www.eurekalert.org/pub_releases/2016-02/wuis-nnm020216.php

Novel nanoparticle made of common mineral may help keep tumor growth at bay

Engineers at Washington University in St. Louis found a way to keep a cancerous tumor from growing by using nanoparticles of the main ingredient in common antacid tablets.

The research team, led by Avik Som, an MD/PhD student, and Samuel Achilefu, PhD, professor of radiology and of biochemistry & molecular biophysics in the School of Medicine and of biomedical engineering in the School of Engineering & Applied Science, in collaboration with two labs in the School of Engineering & Applied Science, used two novel methods to create nanoparticles from calcium carbonate that were injected intravenously into a mouse model to treat solid tumors. The compound changed the pH of the tumor environment, from acidic to more alkaline, and kept the cancer from growing.

With this work, researchers showed for the first time that they can modulate pH in solid tumors using intentionally designed nanoparticles. Results of the research were recently published online in Nanoscale.

"Cancer kills because of metastasis," said Som, who is working on a doctorate in biomedical engineering in addition to a medical degree. "The pH of a tumor has been heavily correlated with metastasis. For a cancer cell to get out of the extracellular matrix, or the cells around it, one of the methods it uses is a decreased pH." The researchers set out to find new approaches to raise the pH of the tumor and do so only in the tumor environment. In water, the pH in calcium carbonate increases as high as 9. But when injected into the body, the team discovered that calcium carbonate only raises the pH to 7.4, the normal pH in the human body. However, working with calcium carbonate presented some challenges.

"Calcium carbonate doesn't like to be small," Som said. "Calcium carbonate crystals are normally 10 to 1,000 times bigger than an ideal nanoparticle for cancer therapy. On top of that, calcium carbonate in water will constantly try to grow, like stalactites and stalagmites in a cave."

To solve this issue, Som worked with other researchers in the School of Engineering & Applied Science to create two unique solutions. Teaming up with researchers in the lab of Pratim Biswas, PhD, the Lucy & Stanley Lopata Professor and chair of the Department of Energy, Environmental & Chemical Engineering, they developed a method using polyethyleneglycol-based diffusion to synthesize 20- and 300-nanometer-sized calcium carbonate.

Working with Srikanth Singamaneni, PhD, assistant professor of materials science, they developed another method to create 100-nanometer-sized calcium carbonate by building on a method known as ethanol-assisted diffusion. By harnessing the complementary expertise of the different labs, the researchers developed a solvent made of albumin to keep the calcium carbonate nanoparticles from growing, allowing them to be injected into the body intravenously.

Commonly, nanoparticles have been made with gold and silver. However, neither are present in the human body, and there are concerns about them accumulating in the body.

"Calcium and carbonate are both found heavily in the body, and they are generally non-toxic," Som said. "When calcium carbonate dissolves, the carbonate becomes carbon dioxide and is released through the lungs, and calcium is often incorporated into the bones."

Som and the team injected the calcium carbonate nanoparticles into the mouse fibrosarcoma model daily, which kept the tumor from growing. However, when they stopped injecting the nanoparticles, it started growing again.

Going forward, the researchers plan to determine the optimal dose to prevent metastasis, improve targeting to tumors and determine if it could be used with chemotherapy drugs.

http://www.eurekalert.org/pub_releases/2016-02/aiop-egr012816.php

Exploring gambles reveals foundational difficulty behind economic theory (and a solution!)

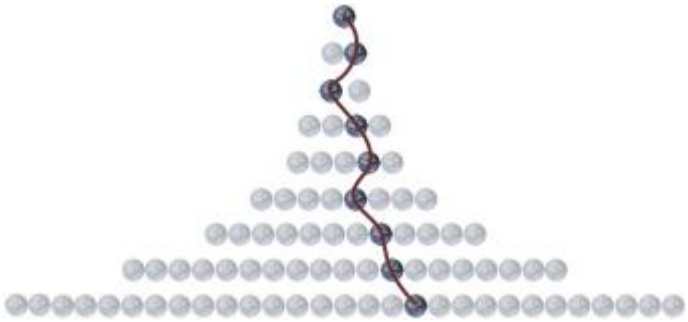
By evaluating gambles via dynamics, Ole Peters and Murray Gell-Mann discovered a foundational difficulty behind current economic theory. They propose an alternative perspective that provides an elegant -- simple -- solution to many of the open key problem

WASHINGTON, D.C. - In the wake of the financial crisis, many started questioning different aspects of the economic formalism.

This included Ole Peters, a Fellow at the London Mathematical Laboratory in the U.K., as well as an external professor at the Santa Fe Institute in New Mexico, and Murray Gell-Mann, a physicist who was awarded the 1969 Nobel Prize in physics for his contributions to the theory of elementary particles by introducing quarks, and is now a Distinguished Fellow at the Santa Fe Institute. They found it particularly curious that a field so central to how we live together as a society seems so unsure about so many of its key questions.

So they asked: Might there be a foundational difficulty underlying our current economic theory? Is there some hidden assumption, possibly hundreds of years old, behind not one but many of the current scientific problems in economic theory? Such a foundational problem could have far-reaching practical consequences because economic theory informs economic policy.

As they report in the journal *Chaos*, from AIP Publishing, the story that emerged is a fascinating example of scientific history, of how human understanding evolves, gets stuck, gets unstuck, branches, and so on.



This image depicts parallel worlds branching into the future, with reality selecting one trajectory through the space of possibilities. **Peters and Gell-Mann**

"We found, for instance, that Daniel Bernoulli made an inconspicuous but consequential error in 1738 that was corrected by Laplace in 1814, but reintroduced by Menger in 1934," said Peters. "This is one factor that held back the development of our perspective."

The key concepts of time and randomness are at the heart of their work. "Questions of an economic nature stood at the beginning of formal thinking about randomness in the 17th century," he explained. "These are all relatively young concepts -- there's nothing in Euclid about probability theory." Think of it simply in terms of: Should I bet money in a game of dice? How much should I pay for an insurance contract? What would be a fair price for a life annuity?

"All of these questions have something to do with randomness, and the way to deal with them in the 17th century was to imagine parallel worlds representing everything that could happen," Gell-Mann said. "To assess the value of some uncertain venture, an average is taken across those parallel worlds."

This concept was only challenged in the mid-19th century when randomness was used formally in a different context -- physics. "Here, the following perspective arose: to assess some uncertain venture, ask yourself how it will affect you in one world only -- namely the one in which you live -- across time," Gell-Mann continued.

"The first perspective -- considering all parallel worlds -- is the one adopted by mainstream economics," explained Gell-Mann. "The second perspective -- what happens in our world across time -- is the one we explore and that hasn't been fully appreciated in economics so far."

The real impact of this second perspective comes from acknowledging the omission of the key concept of time from previous treatments. "We have some

350 years of economic theory involving randomness in one way only -- by considering parallel worlds," said Peters. "What happens when we switch perspectives is astonishing. Many of the open key problems in economic theory have an elegant solution within our framework."

In terms of applications for their work, its key concept can be used "to derive an entire economic formalism," said Peters. In their article, Peters and Gell-Mann explore the evaluation of a gamble. For example, is this gamble better than that gamble? This is the fundamental problem in economics. And from a conceptually different solution there follows a complete new formalism.

They put it to the test after their friend Ken Arrow -- an economist who was the joint winner of the Nobel Memorial Prize in Economic Sciences with John Hicks in 1972 -- suggested applying the technique to insurance contracts. "Does our perspective predict or explain the existence of a large insurance market? It does -- unlike general competitive equilibrium theory, which is the current dominant formalism," Peters said.

And so a different meaning of risk emerges -- taking too much risk is not only psychologically uncomfortable but also leads to real dollar losses. "Good risk management really drives performance over time," Peters added. "This is important in the current rethinking of risk controls and financial market infrastructure."

This concept reaches far beyond this realm and into all major branches of economics. "It turns out that the difference between how individual wealth behaves across parallel worlds and how it behaves over time quantifies how wealth inequality changes," explained Peters. "It also enables refining the notion of efficient markets and solving the equity premium puzzle."

One historically important application is the solution of the 303-year-old St. Petersburg paradox, which involves a gamble played by flipping a coin until it comes up tails and the total number of flips, n, determines the prize, which equals $2 to the nth power. "The expected prize diverges -- it doesn't exist," Peters elaborated. "This gamble, suggested by Nicholas Bernoulli, can be viewed as the first rebellion against the dominance of the expectation value -- that average across parallel worlds -- that was established in the second half of the 17th century."

What's the next step for their work? "We're very keen to develop fully the implications for welfare economics and questions of economic inequality. This is a sensitive subject that needs to be dealt with carefully, including empirical work," noted Peters. "Much is being done behind the scenes -- since this is a conceptually different way of doing things, communication is a challenge, and our work has been difficult to publish in mainstream economics journals."

Their results described in *Chaos* are easily generalized, which is necessary to reinterpret the full formalism. But it "may not add very much in practical terms, and it gets a little technical." So that's a future "to-do item" for Peters and Gell-Mann.

"Our *Chaos* paper is a recipe for approaching a wide range of problems," said Peters. "So we're now going through the entire formalism with our collaborators to see where else our perspective is useful."

"Evaluating gambles using dynamics," by Ole Peters and Murray Gell-Mann. (DOI: 10.1063/1.4940236). may be accessed at http://scitation.aip.org/content/aip/journal/chaos/26/2/10.1063/1.4940236.

http://bit.ly/1UQpRoN

A Chin-Stroking Mystery: Why Are Humans the Only Animals With Chins?

It's an evolutionary conundrum and scientists are still divided over the answer

By Danny Lewis

Many scientists have stroked their chins in puzzlement over...well, the human chin. The bony nub that juts out from the bottom of the lower jaw is unique in the animal kingdom, and although researchers have proposed several theories over the years as to why, the chin remains a mystery.

The chin isn't just the lower part of your face: It's a specific term for that little piece of bone extending from the jaw. While it may seem odd, humans are in fact the only animals that have one. Even chimpanzees and gorillas, our closest genetic cousins, lack chins. Instead of poking forward, their lower jaws slope down and back from their front teeth. Even other ancient hominids, like the Neanderthals, didn't have chins —their faces simply ended in a flat plane, Ed Yong writes for *The Atlantic*.

"If you're looking across all of the hominids, which is the family tree after the split with chimpanzees, there [are] not really that many traits that we can point to that we can say are exclusively human," Duke University's James Pampush tells Robert Siegel for NPR. "[T]hose animals all walked on two legs. The one thing that really sticks out is the chin."

Over the last century, scientists have proposed many ideas to explain why humans evolved chins, from helping us chew food to speaking. Pampush argues that many of these theories don't hold up under further scrutiny. He published this idea recently in the journal *Evolutionary Anthropology*. "The chin is one of these rare phenomena in evolutionary biology that really exposes the deep philosophical differences between researchers in the field," Pampush tells Yong.

One of the most popular ideas is that our ancestors evolved chins to strengthen our lower jaws to withstand the stresses of chewing. But according to Pampush, the

chin is in the wrong place to reinforce the jaw. As for helping us speak, he doubts that the tongue generates enough force to make this necessary. A third idea is that the chin could help people choose mates, but sexually selective features like this typically only develop in one gender, Pampush tells Siegel.

When it comes down to it, the chin may have no real purpose. According to Pampush, it could just be something called a "spandrel," or an evolutionary byproduct left from another feature changing. In the chin's case, it could be the result of the human face shrinking over time as our posture changed and our faces shortened, or a remnant from a period of longer jaws.

"It seems that the appearance of the chin itself is probably related to patterns of facial reduction in humans during the Pleistocene," Nathan Holton, who studies facial evolution at the University of Iowa, tells Yong. "In this sense, understanding why faces became smaller is important to explaining why we have chins."

The spandrel hypothesis is as good a theory as any, but it too has its problems. It's hard to find evidence to test if something is an evolutionary byproduct, especially if it doesn't serve an obvious function. But if researchers one day do manage to figure out where the chin came from, it could put together another piece of the puzzle of what makes us different from our primate and Neanderthal cousins, Yong writes. "Perhaps it will tell us really what gave us that last little step into becoming anatomically modern," Pampush tells Siegel.

http://www.eurekalert.org/pub_releases/2016-02/agu-gis020216.php

Greenland ice sheet releasing 'Mississippi River' worth of phosphorus

Greenland's melting ice sheet unleashing 400,000 metric tons of phosphorus every year

WASHINGTON, DC -- Not only is Greenland's melting ice sheet adding huge amounts of water to the oceans, it could also be unleashing 400,000 metric tons of phosphorus every year - as much as the mighty Mississippi River releases into the Gulf of Mexico, according to a new study. Phosphorus is a key nutrient that could, if it reaches the open ocean, enrich waters of the Arctic Ocean, potentially stimulating growth of the marine food chain, the study's authors said.

Phosphorus is an essential nutrient that feeds plankton at the base of the ocean food web. Glacial meltwater has long been known to contain phosphorus, but now new research shows that as the Greenland ice sheet melts it could be releasing far more of the nutrient than previously thought, reports Jon Hawkings, a Cabot Institute researcher at the Bristol Glaciology Centre at the University of Bristol in the United Kingdom.

Hawkings and his collaborators spent three months in 2012 and 2013 gathering water samples and measuring the flow of water from the 600-square-kilometer (230-square-mile) Leverett Glacier and the smaller, 36-square-kilometer (14-square-mile) Kiattuut Sermiat Glacier in Greenland as part of a Natural Environment Research Council-funded project to understand how much phosphorus, in various forms, was escaping from the ice sheet over time and draining into the sea. They then used that data to extrapolate how much phosphorus was likely being released from the entire Greenland ice sheet.

They found greater amounts of phosphorus in the waters of the Leverett Glacier than had been detected at previous study sites, which have looked mostly at smaller glaciers. The large Leverett Glacier, however, is more representative of the glaciers that contribute the bulk of meltwater coming from the Greenland ice sheet, said Hawkings.

"We find annual phosphorus input (for all of Greenland's outlet glaciers) are at least equal to some of the world's largest rivers, such as the Mississippi and the Amazon," Hawkings and his colleagues report in a new study accepted for publication in Global Biogeochemical Cycles, a journal of the American Geophysical Union. That amount could increase as the climate warms and more ice melts, according to the study's authors.

It is not clear yet how much of the phosphorus being released from the ice sheet is reaching the open ocean, but if a large amount of phosphorus coming off the glacier makes it to the sea, the nutrient could rev up biological activity of Arctic waters, according to the study's authors. The nutrient could stimulate growth of plankton at the base of the ocean food web that could impact birds, fish and marine mammals higher up the food chain. The research also suggests ice sheet-derived phosphorus could eventually reach the northern Pacific and Atlantic oceans, which are connected to the Arctic Ocean.

Unleashing nutrients

Oceanographers have historically thought of glaciers and ice sheets as frozen systems that don't add nutrients or water to the oceans, Hawkings explained. Research over the past couple of decades has shown there is flowing water at the base of glaciers. As climate change warms Greenland and more ice melts and makes its way into the sea, the ice sheet is potentially becoming a more important source of nutrients, he said.

Glacial meltwater gains phosphorus when it travels in moulins, or "pipes" through the ice - through the guts of the glacier and down to the where the ice meets the bedrock. Where the ice meets the bedrock at the very bottom of the glacier, the meltwater is exposed to phosphorus-rich rocks that are pulverized by the moving glacier. "Glaciers are very, very good at crushing up rock," said Hawkings.

The concentrations of dissolved phosphate the researchers found in the Leverett Glacier meltwater - which is just one form of phosphorus found in the meltwater - were similar to concentrations found in Arctic rivers, and among the highest levels recorded in glacial meltwaters worldwide. The total phosphorus concentrations found in the meltwater of the Leverett Glacier - which includes phosphorus-rich particles - was 10 times greater than concentrations found in Arctic river waters.

If the majority of the phosphorus found in meltwater from all of Greenland's glaciers reaches the sea, it would be equal to about 400,000 metric tons (440,000 U.S. tons) per year of phosphorus, more than Arctic rivers are estimated to contribute to the Arctic Ocean, according to the new study. However, how much phosphorus makes it from the meltwater into the open oceans is not yet known. The largest portion of phosphorus, which is in the form of powdered rock minerals, could be settling out of the meltwater and end up buried in Greenland's fjords before it has time to dissolve, Hawkings said.

"This is an important finding because it highlights the role that the rapidly changing Greenland ice sheet plays in supplying nutrients to the Arctic Ocean," observed Eran Hood of the University of Alaska Southeast in Juneau, who studies the meltwater from coastal glaciers in Alaska, and was not involved in the new study.

"Now we need to understand how much of this phosphorus, especially in the particulate, ends up being utilized in high-latitude marine ecosystems form," said Hood. "I think that's an important open question."

http://www.eurekalert.org/pub_releases/2016-02/nioe-pri020216.php

Pharmaceutical residues increasingly disrupt aquatic life: A hidden global change

Let's forget about the climate for a minute. Largely hidden from public view, another global change is causing increasing disruption.

Residues of medicines in water can kill aquatic animals and play havoc with their food web and reproductive cycle. An international team of researchers led by the Netherlands Institute of Ecology (NIOO-KNAW) makes an urgent case for better wastewater treatment and biodegradable pharmaceuticals.

Algae that are becoming far less edible for water fleas and fish, leaving them to starve. Aquatic animals undergoing unwanted sex changes. And fish on their annual run struggling to locate their spawning ground. These are some of the disruptive effects of pharmaceutical residues on the aquatic environment.

"Chemical substances from pharmaceuticals wreak havoc on underwater chemical communication", says the head of the NIOO's department of Aquatic Ecology, Ellen van Donk. She's been heading a team of Dutch, German and US researchers, who take stock of the problem in the next issue of Reviews of Environmental Contamination and Toxicology. "The effects are becoming more and more visible in lakes and ponds worldwide, if you know what to look for."

Smellscape

Unnoticeable to most people, there's an endless stream of 'chatter' going on below the water's surface. Most of that chatter takes the form of infochemicals: chemical substances released by aquatic plants and animals that travel through the water. Just think of the scent of a water flea that is picked up by a fish.

Aquatic organisms depend on this intricate 'smellscape' of information to locate mates and food, and to steer clear of natural enemies. But even low concentrations of pharmaceutical compounds can have a huge impact. "Some of these substances may closely resemble natural infochemicals", explains Van Donk, "so they may unintentionally trigger a similar reaction. Or they could do just the opposite and block vital communications."

Increase

Ever larger amounts of pharmaceutical residues, such as female hormones from the contraceptive pill, have been finding their way into the water - through urination by humans and livestock. These residues are not easily biodegradable, and sewage treatment plants are not equipped to remove them.

"There are no hard figures", says Van Donk, "but surface water measurements suggest that concentrations of residues from hormones, anti-depressants and painkillers have in fact increased substantially."

Solutions

The best way to fight this insidious global change, according to Van Donk and her team, is to find more effective wastewater treatment methods. "In addition, we should look at how pharmaceuticals can be processed better by the human body." It may, for instance, be possible to make pharmaceuticals biodegradable or to absorb them completely.

"At the NIOO, we have been experimenting with an alternative wastewater treatment method that makes use of micro-organisms and algae", adds Van Donk. "This method is allowing us to recycle valuable so-called 'waste' materials, and we're now investigating if it could also help us break down pharmaceutical residues more successfully."

With more than 300 staff and students, the NIOO is one of the largest research institutes of the Royal Netherlands Academy of Arts and Sciences (KNAW). It specialises in terrestrial and aquatic ecology. As of 2011, it is located in an innovative and sustainable research building in Wageningen.

http://www.eurekalert.org/pub_releases/2016-02/d-nrs012916.php

New research shows each hour of sedentary time is associated with a 22 percent increased risk of developing type 2 diabetes

22% increased risk of developing type 2 diabetes per hour of daily sedentary time

Each extra hour of daily sedentary time (for example spent sitting at a computer) is associated with a 22% increased risk of developing type 2 diabetes, concludes new research published in Diabetologia (the journal of the European Association for the Study of Diabetes). The study is by Julianne van der Berg, Maastricht University, the Netherlands, and colleagues.

The study investigated cross-sectional associations of total duration and patterns of sedentary behaviour with glucose metabolism status and the metabolic syndrome. The study participants used the thigh-worn activPAL3 accelerometer, which classifies sedentary behaviour using data on posture, as this has shown to be an accurate means of assessing sedentary behaviour.

In this study, data were taken from the Maastricht Study, an observational, prospective, population-based cohort study in the Netherlands. The authors included 2,497 participants (mean age 60 years, 52% men) from this study who were asked to wear their accelerometer 24 hours per day for 8 consecutive days. The authors calculated the daily amount of sedentary time, daily number of sedentary breaks, number of prolonged sedentary periods (of 30 minutes or more), and the average duration of these sedentary periods. To determine diabetes status, participants underwent an oral glucose tolerance test.

Overall, 1,395 (56%) participants had a normal glucose metabolism, 388 (15%) had an impaired glucose metabolism and 714 (29%) had type 2 diabetes. Participants with type 2 diabetes spent the most time sedentary, up to 26 more minutes per day in comparison with participants with an impaired or normal glucose metabolism.

The increased risk of diabetes per additional hour of sedentary time was 22%. No significant associations were seen for the number of sedentary breaks, the number of prolonged sedentary periods or average duration of these sedentary periods with diabetes status.

The authors say their study is the largest in which this type of posture-identifying accelerometry has been used to objectively measure total duration and patterns of sedentary behaviour in a cohort of people with type 2 diabetes, impaired glucose tolerance, and normal glucose metabolism. The authors say: "An extra hour of sedentary time was associated with a 22% increased odds for type 2 diabetes."

They conclude : "Future studies in participants with type 2 diabetes should be conducted to confirm our results...nevertheless, our findings could have important implications for public health as they suggest that sedentary behaviour may play a significant role in the development and prevention of type 2 diabetes, independent of high-intensity physical activity. Consideration should be given to including strategies to reduce the amount of sedentary time in diabetes prevention programmes."

http://nyti.ms/20q86Ri

Zica Car Will Be Renamed, Tata Motors of India Says

When the Indian automaker Tata Motors unveiled the name of its curvaceous new hatchback late last year, it declared that the car would be the "the next big thing." The name? Zica.

By MIKE McPHATE FEB. 2, 2016

That was before a like-named viral outbreak linked to thousands of birth defects became an international public health emergency and a daily topic in news reports.

On Tuesday, Tata sought to defuse the unfortunate association with the mosquito-borne Zika virus, saying it would rename its car, just as it was to be unveiled this week at an automobile show outside New Delhi.

Tata said it would rename its new hatchback, which was to be unveiled this week at an automobile show outside New Delhi. **Tata Motors**

Tata, based in Mumbai, said in a statement, "Empathizing with the hardships being caused by the recent 'Zika' virus outbreak across many countries, Tata Motors, as a socially responsible company, has decided to rebrand the car."

The lead-up to the car's unveiling has coincided with news coverage of the growing Zika crisis across South and Central America.

Tata appeared to place big hopes on the Zica, even enlisting one of the world's biggest soccer stars, Lionel Messi, as a pitchman. The automaker said a new name would be announced in a few weeks.

A similar branding problem has vexed companies and individuals named Isis, which was the name of an Egyptian goddess long before it was connected to the Islamic State terrorist organization.

Some, after holding out for months, eventually gave in to pressure to go by something else.

http://www.eurekalert.org/pub_releases/2016-02/uota-efc020316.php

Energy from cellphone towers amplify pain in amputees, UT Dallas study finds

For years, retired Maj. David Underwood has noticed that whenever he drove under power lines and around other electromagnetic fields, he would feel a buzz in what remained of his arm.

When traveling by car through Texas' open spaces, the buzz often became more powerful. "When roaming on a cellphone in the car kicked in, the pain almost felt like having my arm blown off again," said Underwood, an Iraq War veteran who was injured by an improvised explosive device (IED). His injuries have resulted in 35 surgeries and the amputation of his left arm. Shrapnel from the IED also tore part of his leg and left him with more than 100 smaller wounds. "I didn't notice the power lines, cellphones on roam or other electromagnetic fields until I first felt them in my arm."

Until a recent study led by researchers at The University of Texas at Dallas was published online last month in PLOS ONE, there was no scientific evidence to back up the anecdotal stories of people, such as Underwood, who reported aberrant sensations and neuropathic pain around cellphone towers and other technology that produce radio-frequency electromagnetic fields.

"Our study provides evidence, for the first time, that subjects exposed to cellphone towers at low, regular levels can actually perceive pain," said Dr. Mario Romero-Ortega, senior author of the study and an associate professor of bioengineering in the University's Erik Jonsson School of Engineering and Computer Science. "Our study also points to a specific nerve pathway that may contribute to our main finding."

Most of the research into the possible effects of cellphone towers on humans has been conducted on individuals with no diagnosed, pre-existing conditions. This is one of the first studies to look at the effects of electromagnetic fields (EMFs) in a nerve-injury model, said Romero-Ortega, who researches nerve regeneration and builds neural interfaces -- technology that connects bionic or robotic devices to the peripheral nerve. There are nearly 2 million amputees in the United States, according to the Centers for Disease Control and Prevention, and many suffer from chronic pain.

After interacting with Underwood, Romero-Ortega decided to study the phenomena that Underwood described. The team hypothesized that the formation of neuromas -- inflamed peripheral nerve bundles that often form due to injury -- created an environment that may be sensitive to EMF-tissue interactions. To test this, the team randomly assigned 20 rats into two groups -- one receiving a nerve injury that simulated amputation, and the other group receiving a sham treatment. Researchers then exposed the subjects to a radiofrequency electromagnetic antenna for 10 minutes, once per week for eight weeks. The antenna delivered a power density equal to that measured at 39 meters from a local cellphone tower -- a power density that a person might encounter outside of occupational settings.

Researchers found that by the fourth week, 88 percent of subjects in the nerve-injured group demonstrated a behavioral pain response, while only one subject in the sham group exhibited pain at a single time point, and that was during the first week. After growth of neuroma and resection -- the typical treatment in humans with neuromas who are experiencing pain -- the pain responses persisted.

"Many believe that a neuroma has to be present in order to evoke pain. Our model found that electromagnetic fields evoked pain that is perceived before neuroma formation; subjects felt pain almost immediately," Romero-Ortega said. "My hope is that this study will highlight the importance of developing clinical options to prevent neuromas, instead of the current partially effective surgery alternatives for neuroma resection to treat pain."

Researchers also performed experiments at the cellular level to explain the behavioral response. That led researchers to explore the protein TRPV4, which is known to be a factor in heat sensitivity and the development of allodynia, which some subjects displayed. "It is highly likely that TRPV4 is a mediator in the pain response for these subjects," Romero-Ortega said. "Our calcium imaging experiments were a good indicator that TRPV4 is worth further exploration."

Romero-Ortega said since the research produced pain responses similar to those in anecdotal reports and a specific human case, the results "are very likely" generalizable to humans.

"There are commercially available products to block radio frequency electromagnetic energy. There are people who live in caves because they report to be hypersensitive to radiomagnetism, yet the rest of the world uses cellphones and does not have a problem. The polarization may allow people to disregard the complaints of the few as psychosomatic," he said. "In our study, the subjects with nerve injury were not capable of complex psychosomatic behavior. Their pain was a direct response to man-made radiofrequency electromagnetic energy."

At one point in the study, members of the research group showed Underwood video of subjects in the experiment and their response to radiofrequency electromagnetic fields.

"It was exactly the same type of movements I would have around cellphones on roam, power lines and other electromagnetic fields," said Underwood, who has served on congressional medical committees and been exposed to some of the best

doctors in the world. "It is pretty amazing that a few short conversations with this team led to validation of what I, and many others, experience."

Researchers said that the next step is to develop devices that block neuropathic pain from radiofrequency electromagnetic energy.

Dr. Bryan Black, a research associate in the Department of Bioengineering in the Jonsson School; Dr. Rafael Granja-Vazquez, a postdoctoral fellow at UT Dallas; Dr. Benjamin Johnston of Brown University; and Dr. Erick Jones Sr., a professor of industrial, manufacturing and systems engineering at UT Arlington, also contributed to the work.

http://www.eurekalert.org/pub_releases/2016-02/mc-mcr012916.php

Mayo Clinic researchers extend lifespan by as much as 35 percent in mice

Senescent cells negatively impact health and shorten lifespan by as much as 35 percent in normal mice

ROCHESTER, Minn. - Researchers at Mayo Clinic have shown that senescent cells - cells that no longer divide and accumulate with age - negatively impact health and shorten lifespan by as much as 35 percent in normal mice. The results, which appear today in Nature, demonstrate that clearance of senescent cells delays tumor formation, preserves tissue and organ function, and extends lifespan without observed adverse effects.

"Cellular senescence is a biological mechanism that functions as an 'emergency brake' used by damaged cells to stop dividing," says Jan van Deursen, Ph.D., Chair of Biochemistry and Molecular biology at Mayo Clinic, and senior author of the paper. "While halting cell division of these cells is important for cancer prevention, it has been theorized that once the 'emergency brake' has been pulled, these cells are no longer necessary."

The immune system sweeps out the senescent cells on a regular basis, but over time becomes less effective. Senescent cells produce factors that damage adjacent cells and cause chronic inflammation, which is closely associated with frailty and age-related diseases.

Mayo Clinic researchers used a transgene that allowed for the drug-induced elimination of senescent cells from normal mice. Upon administration of a compound called AP20187, removal of senescent cells delayed the formation of tumors and reduced age-related deterioration of several organs. Median lifespan of treated mice was extended by 17 to 35 percent. They also demonstrated a healthier appearance and a reduced amount of inflammation in fat, muscle and kidney tissue.

"Senescent cells that accumulate with aging are largely bad, do bad things to your organs and tissues, and therefore shorten your life but also the healthy phase of your life," says Dr. van Deursen. "And since you can eliminate the cells without negative side effects, it seems like therapies that will mimic our findings - or our genetic model that we used to eliminate the cells - like drugs or other compounds that can eliminate senescent cells would be useful for therapies against age-related disabilities or diseases or conditions."

Darren Baker, Ph.D., a molecular biologist at Mayo Clinic, and first author on the study is also optimistic about the potential implications of the study for humans.

"The advantage of targeting senescent cells is that clearance of just 60-70 percent can have significant therapeutic effects," says Dr. Baker. "If translatable, because senescent cells do not proliferate rapidly, a drug could efficiently and quickly eliminate enough of them to have profound impacts on healthspan and lifespan."

The research was supported by the National Institutes of Health, the Paul F. Glenn Foundation, the Ellison Medical Foundation, the Noaber Foundation and the Mayo Clinic Robert and Arlene Kogod Center on Aging.

Others on the research team include: Bennett Childs; Matej Durik, Ph.D.; Melinde Wijers, Jian Zhong, Ph.D., Rachel Saltness, Grace Verzosa, M.D., Abdulmohammad Pezeshki, Ph.D., Khashayarsha Khazaie, Ph.D., Jordan D. Miller, Ph.D.; all of Mayo Clinic.

Drs. van Deursen and Baker are inventors on patents licensed to Unity Biotechnology by Mayo Clinic and Dr. van Deursen is a founder of Unity Biotechnology.

http://www.eurekalert.org/pub_releases/2016-02/luhs-npt020316.php

No proof that radiation from X rays and CT scans causes cancer

Radiation fears based on unproven theoretical model, Loyola researcher reports

MAYWOOD, Il. - The widespread belief that radiation from X rays, CT scans and other medical imaging can cause cancer is based on an unproven, decades-old theoretical model, according to a study published in the American Journal of Clinical Oncology.

The model, known as linear no-threshold (LNT), is used to estimate cancer risks from low-dose radiation such as medical imaging. But risk estimates based on this model "are only theoretical and, as yet, have never been conclusively demonstrated by empirical evidence," corresponding author James Welsh, MD and colleagues write. Use of the LNT model drives unfounded fears and "excessive expenditures on putative but unneeded and wasteful safety measures."

Dr. Welsh is a Loyola University Medical Center radiation oncologist and a professor in the Department of Radiation Oncology of Loyola University Chicago Stritch School of Medicine.

The LNT model dissuades many physicians from using appropriate imaging techniques and "discourages many in the public from getting proper and needed imaging, all in the name of avoiding any radiation exposure," Dr. Welsh and colleagues write.

The authors reexamined the original studies, dating back more than 70 years, which led to adoption of the LNT model. This reappraisal found that the data reported in those studies do not actually support the LNT model.

In the LNT model, the well-established cancer-causing effects of high doses of radiation are extended downward in a straight line to very low doses. The LNT model assumes there is no safe dose of radiation, no matter how small. However, the human body has evolved the ability to repair damage from low-dose radiation that naturally occurs in the environment.

The LNT model dates to studies, conducted in the 1940s, of fruit flies exposed to various doses of radiation. The scientists who conducted those studies concluded there is no safe level of radiation, thus giving rise to the LNT model that is used to this day. But their conclusion was unwarranted because their experiments had not been done at truly low doses. A study exposing fruit flies to low-dose radiation wasn't conducted until 2009, and this study did not support the LNT model.

Studies of atomic bomb survivors and other epidemiological studies of human populations have never conclusively demonstrated that low-dose radiation exposure can cause cancer.

Any claim that low-dose radiation from medical imaging procedures is known to cause cancer "should be vigorously challenged, because it serves to alarm and perhaps harm, rather than educate," Dr. Welsh and colleagues write. The authors conclude the LNT model "should finally and decisively be abandoned."

The study is titled "The birth of the illegitimate linear no-threshold model - an invalid paradigm for estimating risk following low-dose radiation exposure."

In addition to Dr. Welsh, co-authors are Jeffry Siegel, PhD, president and CEO of Nuclear Physics Enterprises (first author); Charles Pennington of NAC International and Bill Sacks, MD, PhD, emeritus medical officer in the FDA Center for Devices and Radiological Health.

http://www.eurekalert.org/pub_releases/2016-02/miot-rdh020316.php

Researchers develop hack-proof RFID chips

New technology could secure credit cards, key cards, and pallets of goods in warehouses

Researchers at MIT and Texas Instruments have developed a new type of radio frequency identification (RFID) chip that is virtually impossible to hack.

If such chips were widely adopted, it could mean that an identity thief couldn't steal your credit card number or key card information by sitting next to you at a café, and high-tech burglars couldn't swipe expensive goods from a warehouse and replace them with dummy tags.

Texas Instruments has built several prototypes of the new chip, to the researchers' specifications, and in experiments the chips have behaved as expected. The researchers presented their research this week at the International Solid-State Circuits Conference, in San Francisco.

According to Chiraag Juvekar, a graduate student in electrical engineering at MIT and first author on the new paper, the chip is designed to prevent so-called side-channel attacks. Side-channel attacks analyze patterns of memory access or fluctuations in power usage when a device is performing a cryptographic operation, in order to extract its cryptographic key.

"The idea in a side-channel attack is that a given execution of the cryptographic algorithm only leaks a slight amount of information," Juvekar says. "So you need to execute the cryptographic algorithm with the same secret many, many times to get enough leakage to extract a complete secret."

One way to thwart side-channel attacks is to regularly change secret keys. In that case, the RFID chip would run a random-number generator that would spit out a new secret key after each transaction. A central server would run the same generator, and every time an RFID scanner queried the tag, it would relay the results to the server, to see if the current key was valid.

Blackout

Such a system would still, however, be vulnerable to a "power glitch" attack, in which the RFID chip's power would be repeatedly cut right before it changed its secret key. An attacker could then run the same side-channel attack thousands of times, with the same key. Power-glitch attacks have been used to circumvent limits on the number of incorrect password entries in password-protected devices, but RFID tags are particularly vulnerable to them, since they're charged by tag readers and have no onboard power supplies.

Two design innovations allow the MIT researchers' chip to thwart power-glitch attacks: One is an on-chip power supply whose connection to the chip circuitry would be virtually impossible to cut, and the other is a set of "nonvolatile" memory cells that can store whatever data the chip is working on when it begins to lose power.

For both of these features, the researchers -- Juvekar; Anantha Chandrakasan, who is Juvekar's advisor and the Vannevar Bush Professor of Electrical Engineering and Computer Science; Hyung-Min Lee, who was a postdoc in Chandrakasan's group when the work was done and is now at IBM; and TI's Joyce Kwong, who did her master's degree and PhD with Chandrakasan -- use a special type of material known as a ferroelectric crystals.

As a crystal, a ferroelectric material consists of molecules arranged into a regular three-dimensional lattice. In every cell of the lattice, positive and negative charges naturally separate, producing electrical polarization. The application of an electric

field, however, can align the cells' polarization in either of two directions, which can represent the two possible values of a bit of information.

When the electric field is removed, the cells maintain their polarization. Texas Instruments and other chip manufacturers have been using ferroelectric materials to produce nonvolatile memory, or computer memory that retains data when it's powered off.

Complementary capacitors

A ferroelectric crystal can also be thought of as a capacitor, an electrical component that separates charges and is characterized by the voltage between its negative and positive poles. Texas Instruments' manufacturing process can produce ferroelectric cells with either of two voltages: 1.5 volts or 3.3 volts.

The researchers' new chip uses a bank of 3.3-volt capacitors as an on-chip energy source. But it also features 571 1.5-volt cells that are discretely integrated into the chip's circuitry. When the chip's power source -- the external scanner -- is removed, the chip taps the 3.3-volt capacitors and completes as many operations as it can, then stores the data it's working on in the 1.5-volt cells.

When power returns, before doing anything else the chip recharges the 3.3-volt capacitors, so that if it's interrupted again, it will have enough power to store data. Then it resumes its previous computation. If that computation was an update of the secret key, it will complete the update before responding to a query from the scanner. Power-glitch attacks won't work.

Because the chip has to charge capacitors and complete computations every time it powers on, it's somewhat slower than conventional RFID chips. But in tests, the researchers found that they could get readouts from their chips at a rate of 30 per second, which should be more than fast enough for most RFID applications.

The MIT researchers' work was also funded by the Japanese automotive company Denso.

http://www.eurekalert.org/pub_releases/2016-02/b-sde020416.php

Senior doctors expose 'scandal' of pacemaker battery life

The battery life of implantable heart monitors must be improved to reduce the need for replacement and the risks this carries for patients, argue two senior doctors in The BMJ today.

Cardiologists John Dean and Neil Sulke say over half of patients with pacemakers will need new batteries and many need several replacements. Not only is money wasted replacing batteries before they've expired, this "exposes patients to risk of serious complications, including life threatening infection," they warn.

The situation is worse for patients with an implantable cardioverter defibrillator (ICD), they add, since the risks of infection at the time of implant and device replacement are higher than with pacemakers and the batteries have a shorter life (around four to seven years on average). "The increased risk of infection associated with battery replacement makes it critical that we prolong the life of implantable devices as much as possible," they write.

Yet they point out that the current financial model discourages the development of longer life devices. "With financial disincentives for both manufacturers and purchasers it is hardly surprising that longer life devices do not exist."

Furthermore, patients are often assumed to prefer smaller devices, they say, but when offered the choice, over 90% would opt for a larger, longer lasting device over a smaller one that would require more frequent operations to change the battery.

"We need to review the timing of replacement of implantable devices in all patients," they write. "While early replacement may be reasonable for high risk patients, allowing batteries to deplete for longer before replacement in lower risk patients could help to maximise device longevity."

For ICDs the waste is even more striking, they add. These devices reach their elective replacement indication when they are still capable of delivering at least six full energy shocks. "So for patients who receive no shock therapy we are prematurely discarding a device costing up to £25,000, which could last at least another six months."

They suggest that with existing technology, engineers could design and build pacemakers that would last for 25 years or more, while further developments in battery technology might enable smaller or rechargeable devices.

"There is an urgent need to minimise the requirement for replacement of these devices. Doing so will save lives, minimise suffering, and reduce costs," they conclude.

http://www.eurekalert.org/pub_releases/2016-02/du-tsd020416.php

Taser shock disrupts brain function, has implications for police interrogations

What does a 50,000-volt shock do to a person's brain?

More than two million citizens have been Tased by police as Taser stun guns have become one of the preferred less-lethal weapons by police departments across the United States during the past decade. But what does that 50,000-volt shock do to a person's brain?

Despite widespread adoption by law enforcement - stun guns are now used in 17,000 police departments - little is known about exactly how the shocks affect individuals' cognitive functioning, or, more specifically, how receiving an electric shock from a Taser might affect the ability of a suspect to understand and waive their Miranda rights.

New research from a first-of-its-kind human study by Drexel University and Arizona State University reveals that the burst of electricity from a stun gun can impair a person's ability to remember and process information. In a randomized control trial, volunteer participants were subjected to Taser shocks and tested for cognitive impairment. Some showed short-term declines in cognitive functioning comparable to dementia, raising serious questions about the ability of police suspects to understand their rights at the point of arrest.

The study informs public policy in the area of police interrogations, specifically addressing the length of time police departments might wait before interviewing suspects who have been Tased by police officers.

The study, "TASER Exposure and Cognitive Impairment: Implications for Valid Miranda Waivers and the Timing of Police Custodial Interrogations," was published this month in the journal Criminology & Public Policy.

Funded by the U.S. Department of Justice's National Institute of Justice, it marks the first time that the Taser has been submitted to a major randomized clinical trial on a community sample outside the purview of Taser International.

The article was authored by Robert J. Kane, PhD, professor and director of the Criminology and Justice Studies Department in Drexel's College of Arts and Sciences and a senior research fellow at the Center for Violence Prevention and Community Safety at ASU; and Michael D. White, PhD, a professor in ASU's School of Criminology and Criminal Justice, who were co-principal investigators on the study, along with Justin Ready, PhD, an assistant professor in ASU's School of Criminology and Criminal Justice.

"The findings of this study have considerable implications for how the police administer Miranda warnings," said Kane. "If suspects are cognitively impaired after being Tased, when should police begin asking them questions? There are plenty of people in prison who were Tased and then immediately questioned. Were they intellectually capable of giving 'knowing' and 'valid' waivers of their Miranda rights before being subjected to a police interrogation? We felt we had moral imperative to fully understand the Tasers' potential impact on decision-making faculties in order to protect individuals' due process rights."

To examine the effects of the Taser, the researchers recruited 142 participants who were required to undergo intensive screening protocols, including those for drug use and cardiac and psychiatric problems.

According to White, "the study involved an elaborate admission process with significant protections in place to insure participant safety." The randomized control trial was conducted at a hospital, with nurses and a physician on hand in case of emergency.

The participants were randomly divided into four groups. A control group of 37 participants did nothing, 32 people hit a punching bag to simulate the heightened physical state one might expect in a tense police encounter, 35 received five-second shocks and 38 hit a punching bag and received five-second shocks.

Each participant completed a battery of cognitive instruments at a preliminary screening stage, immediately before treatment exposure, immediately after completion of their treatment condition, one hour later and one week later. The research team assessed participants' scores both within and across groups over time to assess change in cognitive functioning.

Participants showed the greatest variability on the Hopkins Verbal Learning Test, which can indicate anything from mild learning impairments to dementia by measuring a person's ability to learn new information (a string of words) and then recall that information after different intervals of time. The results indicate that Taser exposure caused statistically significant reductions in verbal learning and memory. The effects lasted, on average, less than one hour.

While short-term, the severity of the disruption was considerable, according to the researchers. The mean score for each group at pre-test was 26 - just above the national average.

At post-test, one quarter of each Taser group scored below 20 on the HVLT, which represents the mean level cognitive functioning for 79-year-old adults, placing participants within the range of mild cognitive impairment. White said "our test administrators could clearly observe the difficulty many participants had with the HVLT after TASER exposure."

"Tasers are a great alternative to deadly force. When used in lieu of firearms, Tasers can save lives," said Kane. "But using a Taser is not without risk. Although they are considered safe when used on healthy people, people have died from being Tased. They should be treated as a dangerous weapon."

The results also showed that Taser exposure caused significant negative change in several subjective state self-measures, including concentration difficulty, anxiety level and feeling overwhelmed. The significant findings in the subjective state measures raise the possibility that emotional factors after Taser exposure are important and may affect test performance.

"Being shocked had a traumatic effect on some participants," said Kane. "Some were emotionally debilitated by the experience."

The researchers point out that study participants were high-functioning, healthy young people who were accustomed to test taking and were sober and drug free at the time they were Tased, and thus, were functioning at a much higher level of cognition than do the 'typical' suspects in the field who experience Taser exposure at the hands of police officers. "We would expect 'typical' suspects - who may be

high, drunk or mentally ill and in crisis at the time of exposure - to experience even greater impairment to cognitive functioning as the result of Taser exposure," said Kane."

The questions driving this study involve serious issues including constitutionally protected rights of the accused, use of force by police and previously unexamined effects of the Taser on the human body.

"When police take suspects into custody, they read them their Miranda rights, which state that suspects have the right to remain silent, and anything they say can and will be used against them in a court of law," said Kane.

"The findings from this study suggest that people who have been shocked with a Taser may be unable to understand and rationally act upon his or her legal rights, and may be more likely to waive their Miranda rights directly after Taser exposure or to give inaccurate information to investigators. These decisions can have profound impact on an eventual judicial finding of guilt or innocence."

The researchers suggest a public dialogue about how to best integrate the Taser into everyday lawful policing in ways that maintain officer safety while reducing potential social costs incurred by suspects exposed to a Taser discharge. They ask: "What would it cost police to wait 60 minutes after a Taser deployment before engaging suspects in custodial interrogations?"

http://www.eurekalert.org/pub_releases/2016-02/uow-rhi020416.php

Researchers hone in on why female newborns are better protected from brain injury

Why gender difference in hypoxic ischemic encephalopathy exists has remained a mystery

MADISON, Wis. - Each year, thousands of newborn babies suffer complications during pregnancy or birth that deprive their brains of oxygen and nutrient-rich blood and result in brain injury.

This deprivation results in hypoxic ischemic encephalopathy (HIE), which can lead to long-term neurological issues such as learning disabilities, cerebral palsy or even death.

Researchers have known for some time that male infants are more vulnerable to HIE than females, but why this gender difference exists has remained a mystery.

In a study published this week in the journal eNeuro, researchers at the Waisman Center at the University of Wisconsin-Madison, led by Pelin Cengiz, associate professor in the Department of Pediatrics, show that a particular protein found in the brains of both male and female mice is present at higher levels in females, which offers them stronger protection against this type of brain injury.

"People often think that biological sex differences start to arise only after puberty, but they actually start in the womb and persist until the tomb," says Cengiz, paraphrasing a 1999 statement by the Institute of Medicine.

"So, treatment approaches that may work for newborn boys may not work for girls, and vice versa. We need to get it right to develop effective therapies."

The protein is called estrogen receptor α, or ERα for short, and the researchers set out to learn how it confers its gender-specific protective effects.

Their first clue lay with a particular drug known to protect female but not male newborn mice from the effects of brain injury caused by HIE.

The drug works by turning on a cascade of protective effects in the brain in response to oxygen deprivation and reduced blood flow.

The team learned that, like the drug, ERα also causes a similar cascade in infant mice and the protein is actually required for the drug to be effective.

The researchers found that female mice lacking the ERα protein could not activate protective factors following HIE, even when treated with the drug.

When the researchers studied the brains of male and female mice that could make the ERα protein, they learned that levels of this protective protein were significantly higher in female compared to male brains following oxygen deprivation and reduced blood flow.

"Under normal circumstances the brains of male and female mice have similar amounts of ERα," says Cengiz, who is now exploring why ERα levels increase in female but not male brains after HIE.

Understanding the mechanism of how female brains are more resistant to damage from oxygen deprivation and reduced blood flow is a first step toward helping newborns of both sexes recover after suffering from HIE and live functional lives. It could also lead to more effective therapies and treatments for both genders, Cengiz says.

But more work needs to be done. For one thing, Cengiz and her colleagues looked at only the hippocampus region of the brain, which is linked to memory and learning and is involved in other neurological roles.

The hippocampus is also a site where new neurons are continually generated throughout the lifespan.

"We focused on the hippocampus because we see memory and learning disabilities in many of the children affected by HIE," says Cengiz, "and it is also the part of the brain that is most often injured after HIE."

While it could be years before human babies benefit, each molecular mystery researchers unravel provides a potential new road to developing new therapies, Cengiz says, noting: "We are driven by the desire to improve outcomes for all newborns who suffer brain injury from HIE."

http://www.eurekalert.org/pub_releases/2016-02/ttgr-dat020416.php

Dogs accelerate the advance of new cancer treatments for both pets and people

National review shows studying cancer in dogs offers 'a unique opportunity' for helping patients, saving time and decreasing costs

PHOENIX, Ariz. - A Science Translational Medicine review suggests integrating dogs with naturally occurring cancers into studies of new drug therapeutics could result in better treatments for our four-legged friends while helping inform therapeutic development for human cancers.

The review, conducted by the Institute of Medicine (IOM) of the National Academy of Science, including faculty at the Translational Genomics Research Institute (TGen), hopes to close the gap between human and canine cancer research, and accelerate the knowledge developed by studying cancer in both people and pets, a field known as comparative oncology.

"We are hopeful this analysis will be useful in developing and advancing an agenda for the field of comparative oncology," said Dr. Jeffrey Trent, TGen President and Research Director, and one of the authors of the study. "Many canine breeds develop naturally occurring cancers, such as breast cancer and melanoma, that share remarkable genetic similarities with their human equivalent. This allows us a unique opportunity to have what we learn in the human be of help to the dog, and what we learn in the dog to be of direct help to human patients with these cancers."

Dr. William Hendricks, an Assistant Professor at TGen specializing in canine research, agreed: "It has been remarkable to see first hand the similarity in genetic changes, called mutations, between a dog with melanoma and a human patient with the same disease. Looking through the lens of genetics is giving us new targets and offering new hope for improving our treatment of humans and dogs."

This "gap analysis" is the result of a National Academies Institute of Medicine workshop -- The role of Clinical Studies for Pets with Naturally Occurring Tumors in Translational Cancer Research -- held June 8-9, 2015, in Washington, D.C.

"Low cancer drug development success rates and the associated high attrition rates of new drugs, particularly late in human clinical trials, are indicative of a key shortcoming in the preclinical development path," said Dr. Chand Khanna, a former Senior Scientist at NCI's Center for Cancer Research, who holds both a Doctor of Veterinary Medicine and a Ph.D. in Pathobiology, an interdisciplinary field devoted to basic research into the mechanisms of disease.

"Strong similarities between the biology of cancer in dogs and humans have been shown, including patterns of response to therapies and cancer recurrence," said Dr. Khanna, the review's senior author. "Specific types of cancer are functionally identical between dogs and humans, and in some cases the cancers can be considered indistinguishable between the species."

Findings the authors report include:

A limited understanding of the filed of comparative oncology in the cancer drug development community.

The value of comparative oncology can be seen not only in accelerating drug development and eventual FDA approval, but also in saving time, costs and risks to patients by providing early assessments of clinical trials that should be discontinued.

Studying canines to answer questions about drug target biology -- before and after exposure to novel treatments -- should be a priority.

Comparative oncology also should prioritize the development and validation of biomarkers in circulating blood, and guide decisions about optimal drug combination strategies.

There is a need to include veterinarians in clinical practice and in the pharmaceutical industry, physician and veterinary medical associations, and aligned philanthropic groups, in the discussion of opportunities presented by comparative oncology.

Tissue samples of canine cancers stored in tissue banks and bio-specimen repositories "should now be leveraged in order to rapidly accelerate comparative oncology."

Importantly, this review found that the knowledge of genetic alterations that drive human cancers far exceeds knowledge of those same alterations in canine cancers. More than 30,000 human cancers have been genomically profiled, while genomic sequencing data has been published for fewer than 50 canine cancers.

"Our understanding of the genomic landscape of canine cancer is widely considered to be the single largest gap currently present in comparative oncology today," said Dr. Amy LeBlanc, Director of the Comparative Oncology Program at NCI's Center for Cancer Research, and the review's lead author.

Other recommendations included in the review: Veterinary schools are best positioned and prepared to successfully recruit and manage canine patients for comparative oncology studies; the successes in immunotherapy in human cancer treatments should be extended to canine clinical trials; and a centralized registry of canine clinical trials should be created, providing easy access for pet owners and veterinarians.

This "Focus" article, published Feb. 3, 2016, in Science Translational Medicine is titled: Perspectives from man's best friend: National Academy of Medicine's Workshop on Comparative Oncology: http://stm.sciencemag.org/content/8/324/324ps5.

http://bit.ly/1XcPlOS

Tiny doses of opioid could be first fast anti-suicide drug

Could a painkiller turn people away from suicide?

A preliminary trial of an opioid called buprenorphine shows that the drug can reduce suicidal thoughts after just one week. If validated in larger studies, it could become the first fast-acting anti-suicide drug.

Such a drug is sorely needed. The US Centers for Disease Control and Prevention (CDC) estimates that more than 9 million adults in the country reported having suicidal thoughts in 2013. Over a million went on to attempt suicide. "Around 400,000 suicidal people are coming to emergency rooms every year," says Elizabeth Ballard at the National Institute of Mental Health. "Pharmacologically, nothing has been approved for acute treatment of suicidal ideation so anything that can help them is greatly needed."

When people seek help, they may be offered behavioural therapy or drugs such as antidepressants. But neither of these is guaranteed to alleviate feelings, and both can take six weeks or more to kick in. Ketamine, a drug being considered as an immediate treatment, can cause hallucinations and its effects wear off quickly. "Having something you could use on your own outside of a hospital would be beneficial," says Ballard.

Altered perception

Jaak Panksepp at Washington State University and his colleagues decided to see whether an opioid can counter suicidal feelings. Opioids are one of the brain's natural feel-good chemicals. They are released to relieve pain when we hurt ourselves, and are involved when we deal with mental pain, such as that caused by social rejection, a common trigger for suicidal thoughts. Recent studies have shown that the system seems to malfunction in people with depression. Separate work has shown that giving people low doses of opioids decreases their perception of social rejection. "Converging lines of evidence point to a connection between mental pain, depression, suicidal ideation and the body's natural opioids," says Panksepp.

Panksepp's team and collaborators at the University of Haifa in Israel gave very low doses of buprenorphine to 40 people identified as being severely suicidal – almost two-thirds of the group had already attempted to kill themselves. A second group received a placebo. The severity of the participants' thoughts was measured every week for a month by a psychiatrist using a questionnaire. Half the participants were given their drug to take at home, the other half received it in the hospitals where they were staying for treatment.

At the start of the month-long trial, the average score of the participants was about 20. People given buprenorphine dropped an average of six points after one week and nearly 10 points by the end. Participants given a placebo only dropped two points after the full month of treatment. To put this in context, a score of 20 is deemed worrying enough to hospitalise a person for their own safety. This wouldn't be thought necessary for a score of 10.

Twelve members of the study were unable to continue beyond the first week because they were so ill and two people – one from each group – attempted to end their lives during the trial. However, a week after the trial finished, everyone who had completed it reported no worsening of their condition.

"Anything with effects even at the two week to month level would help a lot of people," says Ballard. "I think it's an exciting area of study."

Opioid abuse

Panksepp says he's confident that giving people higher doses of buprenorphine would have seen the effect kick in even earlier. Upping the dose is likely to be controversial, however, especially in the US, where the abuse of prescription opioids is so bad it is being called an epidemic. The latest information, from 2013, shows that across the country, 44 people died from overdoses of drugs such as OxyContin (oxycodone) each day.

The danger with opioids is that taking too much can dampen a person's breathing to lethal levels. Of all the opioids, buprenorphine carries the lowest risk because there's a dose beyond which users get no additional pain-relief – or high. It is even prescribed to people who are addicted to other opioids. What's more, the daily dosages Panksepp's team administered were 30 times lower than the amount needed to create an addiction to the drug. No participants reported going through withdrawal once they stopped taking buprenorphine, suggesting that none became dependent on the drug during the study.

"I think they're onto something. However, buprenorphine acts on a number of different opioid receptors and it's still unclear which one or ones are playing a role in the anti-suicidal effects," says Joan Striebel, a psychiatrist with the California Department of State Hospitals. "I hope this work spurs more interest in what specific molecules could be involved in suicidal thought."

"As a psychiatrist I have spent the last 25 years of my life speaking to people who want to kill themselves on an almost daily basis. Studying and treating the neurochemistry may help us prevent broken lives," says co-author Yoram Yovell of the Institute for the Study of Affective Neuroscience at the University of Haifa.

Journal reference: The American Journal of Psychiatry, *DOI: 10.1176/appi.ajp.2015.15040535*

Speak to your doctor before taking any medication.

Need a listening ear? *US National Suicide Prevention Lifeline: 1 800 273 8255; UK Samaritans: 08457 90 90 90*; hotlines in other countries.

http://www.bbc.com/news/science-environment-35484763

Spread of bee disease 'largely manmade'

The global trade in bees is driving a pandemic that threatens hives and wild bees, UK scientists say.

By Helen Briggs BBC News

A deadly bee disease has spread worldwide through imports of infected honeybees, according to genetic evidence. Stricter controls are needed to protect bees from other emerging diseases, researchers report in Science journal. The virus together with the Varroa mite can kill-off whole hives, putting bee populations at risk.

Lead researcher Dr Lena Bayer-Wilfert of the University of Exeter said European bees are at the heart of the global spread of what she calls a "double blow" for colonies. "This is clearly linked to the human movement of honeybee colonies around the globe," she told BBC News. "It shows a piece of evidence we can't argue with."

'Major threat'

The pattern of the spread shows the movement of the virus around the world is manmade rather than natural, say scientists.

Co-researcher Prof Roger Butlin of the University of Sheffield said Deformed Wing Virus (DWV) was a major threat to honeybee populations across the world with the epidemic "driven by the trade and movement of honeybee colonies".

In the research, scientists at the University of Exeter, Sheffield and Salford tracked the emergence of DWV by analysing genetic samples from honeybees and Varroa mites in 32 locations of 17 countries. They found that the epidemic largely spread from Europe to North America and countries such as New Zealand, with the European honeybee as the main transmitter.

Prof Stephen Martin of the University of Salford said the combination of the virus and the mite were at the heart of the crash in honeybee populations. "It supports the idea that DWV is the main cause for the colony losses associated with Varroa and that this comes from European bees," he said.

Scientists believe the combination is particularly deadly because the parasite feeds on bee larvae, while also injecting the deadly virus into the body of grown bees.

The double threat is thought to have wiped out millions of honeybee colonies over recent decades.

Strict limits

The researchers are calling for tighter controls on importing honeybees, such as mandatory health screenings and more checks on movements across borders. And they say every effort should be made to stop Varroa entering the few areas that are free of the mite to provide a refuge for conservation purposes.

Dr Bayer-Wilfert added: "We must now maintain strict limits on the movement of bees, whether they are known to carry Varroa or not."

Commenting on the study, Prof Mark Brown of Royal Holloway University of London said there were already trade controls in place for honeybees - such as checks by vets - but these were clearly not sufficient. "We need better regulation if we want to stop this happening in the future for other viruses that are likely to emerge," he said.

The European honeybee is used worldwide for commercial pollination of crops such as nuts and fruit as well as for honey production.

http://www.eurekalert.org/pub_releases/2016-02/ats-mir020216.php

Most internet resources for IPF are inaccurate, incomplete and outdated

Evaluation of almost 200 websites found that the information on idiopathic pulmonary fibrosis from these sites was often faulty

After evaluating content on idiopathic pulmonary fibrosis on almost 200 websites, researchers with medical backgrounds found that the information on IPF from these sites was often incomplete, inaccurate and outdated. The study, "Accuracy and Reliability of Internet Resources for Information on Idiopathic Pulmonary Fibrosis" highlights the need for the medical community to continually reassess the accuracy of online information. The research was printed online ahead of print in the American Thoracic Society's American Journal of Respiratory and Critical Care Medicine.

Characterized by a progressive decline in lung function, specifically shortness of breath, IPF is chronic and ultimately fatal. Patients who rely on the Internet for treatment recommendations may be putting themselves at considerable risk. "Nearly half of IPF-related websites suggested a role for at least one medication with no proven benefit, and more than a third of websites recommended medications that are harmful in IPF," said co-author Christopher Ryerson, MD, Assistant Professor at the Centre for Heart Lung Innovation at St. Paul's Hospital and the University of British Columbia.

Using DISCERN, a validated instrument for assessing the quality of written medical information, the researchers evaluated IPF-related content on sites that included foundation/ advocacy organizations, news/media reports, blogs, and scientific resources as well as industry/for profit companies. Each site received a score based on the quality of the content, the quality of the information regarding treatment options, the overall publication, and the reliability of the publication.

The researchers found that foundation/advocacy websites were more likely to recommend non-indicated therapies for treatment. News/media reports were less

likely to provide an overview of IPF, instead focusing on a single item such as newly-approved treatment. The top two websites for both content and quality scores were Wikipedia and Medscape.

"The Internet will remain a common source of health information for patients," said Jolene Fisher, MD, Respirologist at University Health Network, University of Toronto and study co-author. "The medical community, including IPF specialists, needs to take a more active role in ensuring patients have access to accurate and up-to-date online medical information. Patients with IPF should be aware that the information they are accessing may be inaccurate and that harmful recommendations may be made, even on websites from reputable organizations."

http://www.eurekalert.org/pub_releases/2016-02/nhgr-nri020316.php

NIH researchers identify striking genomic signature shared by 5 types of cancer

National Institutes of Health researchers have identified a striking signature in tumor DNA that occurs in five different types of cancer.

They also found evidence that this methylation signature may be present in many more types of cancer. The specific signature results from a chemical modification of DNA called methylation, which can control the expression of genes like a dimmer on a light switch. Higher amounts of DNA methylation (hypermethylation), like that found by the researchers in some tumor DNA, decreases a gene's activity. Based on this advance, the researchers hope to spur development of a blood test that can be used to diagnose a variety of cancers at early stages, when treatments can be most effective. The study appeared February 5, 2016, in The Journal of Molecular Diagnostics.

"Finding a distinctive methylation-based signature is like looking for a spruce tree in a pine forest," said Laura Elnitski, Ph.D., a computational biologist in the Intramural Research Program at NIH's National Human Genome Research Institute (NHGRI). "It's a technical challenge to identify, but we found an elevated methylation signature around the gene known as ZNF154 that is unique to tumors." Dr. Elnitski is head of the Genomic Functional Analysis Section and senior investigator in the Translational and Functional Genomics Branch at NHGRI.

In 2013, her research group discovered a methylation mark (or signature) around ZNF154 in 15 tumor types in 13 different organs and deemed it a possible universal cancer biomarker. Biomarkers are biological molecules that indicate the presence of disease. Dr. Elnitski's group identified the methylation mark using DNA taken from solid tumors. "No one in my group slept the night after that discovery," Dr. Elnitski said. "We were so excited when we found this candidate biomarker. It's the first of its kind to apply to so many types of cancer."

In this new study, they developed a series of steps that uncovered telltale methylation marks in colon, lung, breast, stomach and endometrial cancers. They showed that all the tumor types and subtypes consistently produced the same methylation mark around ZNF154.

"Finding the methylation signature was an incredibly arduous and valuable process," said NHGRI Scientific Director Dan Kastner, M.D., Ph.D. "These findings could be an important step in developing a test to identify early cancers through a blood test."

The NIH Intramural Sequencing Center sequenced the tumor DNA that had been amplified using a technique called polymerase chain reaction (PCR). Dr. Elnitski and her group then analyzed the results, finding elevated levels of methylation at ZNF154 across the different tumor types.

To verify the connection between increased methylation and cancer, Dr. Elnitski's group developed a computer program that looked at the methylation marks in the DNA of people with and without cancer. By feeding this information into the program, they were able to predict a threshold for detecting tumor DNA. Even when they reduced the amount of methylated molecules by 99 percent, the computer could still detect the cancer-related methylation marks in the mixture. Knowing that tumors often shed DNA into the bloodstream, they calculated the proportions of circulating tumor DNA that could be found in the blood.

Next steps

Dr. Elnitski will next begin screening blood samples from patients with bladder, breast, colon, pancreatic and prostate cancers to determine the accuracy of detection at low levels of circulating DNA. Tumor DNA in a person with cancer typically comprises between 1 and 10 percent of all DNA circulating in the bloodstream. The group noted that when 10 percent of the circulating DNA contains the tumor signature, their detection rate is quite good. Because the methylation could be detected at such low levels, it should be adequate to detect advanced cancer as well as some intermediate and early tumors, depending on the type.

Dr. Elnitski's group will also collaborate with Christina Annunziata, M.D., Ph.D., an investigator in the Women's Malignancies Branch and head of the Translational Genomics Section at NIH's National Cancer Institute (NCI). They will test blood samples from women with ovarian cancer to validate the process over the course of treatment and to determine if this type of analysis leads to improved detection of a recurrence and, ultimately, improved outcomes.

"Ovarian cancer is difficult to detect in its early stages, and there are no proven early detection methods," said Dr. Annunziata. "We need a reliable biomarker for detecting the disease when a cure is more likely. We are looking forward to testing Dr. Elnitski's novel approach using DNA methylation signatures."

Current blood tests are specific to a known tumor type. In other words, clinicians must first find the tumor, remove a sample of it and determine its genome sequence. Once the tumor-specific mutations are known, they can be tracked for appearance in the blood. The potential of the new approach is that no prior knowledge of cancer is required, it would be less intrusive than other screening approaches like colonoscopies and mammograms and it could be used to follow individuals at high risk for cancer or to monitor the activity of a tumor during treatment. Once the blood test is developed, the scientific community must conduct studies to ensure that it does not indicate the presence of cancer when it is not there or miss cancer when it is there.

Dr. Elnitski does not yet understand the connection between tumors and elevated DNA methylation. It may represent derailment of normal processes in the cell, or it may have something to do with the fact that tumors consume a lot of energy and circumvent the cellular processes that keep growth in check. Researchers also don't know exactly what the gene ZNF154 does.

"We have laid the groundwork for developing a diagnostic test, which offers the hope of catching cancer earlier and dramatically improving the survival rate of people with many types of cancer," Dr. Elnitski said.

http://www.eurekalert.org/pub_releases/2016-02/uov-dmw020516.php

Discovery: Many white-tailed deer have malaria

Smithsonian and UVM researchers discover first-ever native malaria in the Americas

Two years ago, Ellen Martinsen, was collecting mosquitoes at the Smithsonian's National Zoo, looking for malaria that might infect birds--when she discovered something strange: a DNA profile, from parasites in the mosquitoes, that she couldn't identify.

By chance, she had discovered a malaria parasite, Plasmodium odocoilei--that infects white-tailed deer. It's the first-ever malaria parasite known to live in a deer species and the only native malaria parasite found in any mammal in North or South America. Though white-tailed deer diseases have been heavily studied--scientist hadn't noticed that many have malaria parasites.

Martinsen and her colleagues estimate that the parasite infects up to twenty-five percent of white-tailed deer along the East Coast of the United States. Their results were published February 5 in Science Advances.

In hiding

White-tailed deer in the Smithsonian's National Zoological Park. **Ellen Martinsen**

"You never know what you're going to find when you're out in nature--and you look," says Martinsen, a research associate at the Smithsonian's Conservation Biology Institute and adjunct faculty in the University of Vermont's biology department. "It's a parasite that has been hidden in the most iconic game animal in the United States. I just stumbled across it."

The new study, led by Martinsen, was a collaboration with scientists at the Smithsonian Conservation Biology Institute, the American Museum of Natural History, the National Park Service, the University of Georgia, the University of Wisconsin-Milwaukee--and UVM biologist and malaria expert Joseph Schall.

Though Martinsen and Schall are quick to note that they anticipate little danger to people from this newly discovered deer malaria, it does underline the fact that many human health concerns are connected to wider ecological systems--and that understanding the biology of other species is a foundation to both conservation and public health management.

Zika virus is recently making worrisome headlines and "there's a sudden surge in interest in mosquito biology across the United States," says Schall. "This is a reminder of the importance of parasite surveys and basic natural history."

In 1967, a renowned malaria researcher reported he'd discovered malaria in a single deer in Texas. But the received understanding was that "malaria wasn't supposed to be in mammals in the New World," says Schall, who has studied malaria for decades. "It was like the guy was reporting he saw Big Foot," and no other discoveries were made after that.

But now Martinsen and her colleagues have discovered that the deer malaria is widespread--though it's "cryptic" she says, because the parasites occur in very low levels in many of the infected deer. "Ellen spent days and days looking through a microscope at slides that were mostly empty," Schall says, but eventually found the parasites.

Combined with sensitive molecular PCR techniques to understand the genetics, the team confirmed a high prevalence of the disease--between eighteen and twenty-five percent--in sites ranging from New York to West Virginia to Louisiana.

Native species

The new discovery fundamentally changes our understanding of the distribution and evolutionary history of malaria parasites in mammals, Martinsen says. Some scientists wondered if the deer malaria could have jumped from people or zoo animals in the recent past.

But the new study suggests otherwise. The team's data shows that the deer actually carry two genetic lineages of the malaria parasites--"probably different species," she says--and that the two lineages are substantially different from each other.

This divergence between the two forms of malaria was used by the scientists as a kind of molecular clock. "We can date the evolutionary split between those two lineages,"

Martinsen says--to 2.3 to 6 million years ago. Which probably means that when the ancient evolutionary ancestors to white-tailed deer traveled from Eurasia across the Bering Land Bridge to North America in the Miocene, some 4.2 to 5.7 million years ago--malaria came along for the ride.

"We think malaria is native to the Americas," Martinsen says, "that it's been here for millions of years."

Malaria is a major problem for people in many parts of the world--and for many species of wildlife too. It has been devastating bird species in Hawaii and Bermuda, among many epidemics. Whether it is hurting white-tailed deer in America is an open question. Martinsen suspects not, because she'd expect to see more obviously sick animals.

But Schall wonders if, like some human malaria infections, the disease causes a low-level burden that hurts deer populations. They both agree that it is an area that calls for more research--and that the new study raises many other questions, including whether the parasite might infect dairy cows or other hoofed species.

Ellen Martinsen completed her undergraduate and doctoral training at UVM in Joe Schall's lab and went on to do her postdoctoral research at the Smithonian Conservation Biology Institute's Center for Conservation Genetics.

The new discovery drew on a team of scientists and veterinarians at the Smithsonian and other institutions, who studied samples from both live and necropsied deer as well as mosquitoes. Additionally, Martinsen returned to Schall's lab for some of the new research.

"Malaria is a top parasitic disease in humans and wildlife," Ellen Martinsen says. "It's important that we gain a better understanding of its diversity and distribution not just across humans but across other species too."

http://www.eurekalert.org/pub_releases/2016-02/uoc--afm020516.php

A flawed measure

BMI is not an accurate measure of health, according to research by UCSB psychologist Jeffrey Hunger and colleagues

In what could be the death knell for that once-vaunted measure of health known as BMI (body mass index), new research out of UC Santa Barbara and UCLA reveals that millions of Americans labeled overweight or obese based on their BMI are, in fact, "perfectly healthy."

Their findings, which appear in the International Journal of Obesity, suggest that 34.4 million Americans considered overweight by virtue of BMI are actually healthy, as are 19.8 million who are considered obese.

According to Jeffrey Hunger, a doctoral student in UCSB's Department of Psychological & Brain Sciences, and a co-author of the paper, BMI is a deeply flawed measure of health. "In the overweight BMI category, 47 percent are perfectly healthy," he said. "So to be using BMI as a health proxy -- particularly for everyone within that category -- is simply incorrect. Our study should be the final nail in the coffin for BMI."

Using data from the most recent National Health and Nutrition Examination Survey, the scientists analyzed the link between BMI -- calculated by dividing a person's weight in kilograms by the square of the person's height in meters -- and several health markers, including blood pressure, blood sugar and cholesterol. The results showed that more than 2 million people identified as "very obese" by virtue of having a BMI of 35 or higher are, in reality, healthy; that's about 15 percent of Americans so classified. The research also revealed that more than 30 percent of those with BMIs in the "normal" range -- about 20.7 million people -- are actually unhealthy based on their other markers.

"Not only does BMI mislabel 54 million heavier individuals as unhealthy, it actually overlooks a large group of individuals considered to have a 'healthy' BMI who are actually unhealthy when you look at underlying clinical indicators," said Hunger. "We used a fairly strict definition of health. You had to be at clinically healthy levels on four out of the five health indicators assessed."

Many U.S. companies use employees' BMI as a factor in determining their health insurance costs. And if a rule proposed by the Equal Employment Opportunity Commission (EEOC) is adopted, people with a BMI higher than 25 (the "healthy" range is 18.5 to 24.99) could find themselves paying higher health insurance premiums.

"We need to move away from trying to find a single metric on which to penalize or incentivize people and instead focus on finding effective ways to improve behaviors known to have positive outcomes over time," Hunger argued.

Lead author Janet Tomiyama, an assistant professor of psychology at UCLA, noted that healthy people with BMIs above 24.99 would be no more likely to incur higher medical expenses than those with lower BMIs, so requiring those individuals to pay out more in health insurance premiums would not be justified.

Previous research by Tomiyama's Dieting, Stress and Health (DiSH) laboratory at UCLA found no clear connection between weight loss and health improvements related to hypertension, cholesterol and diabetes and blood glucose levels. The new study recommends that people focus on a healthy diet and regular exercise, rather than placing emphasis on their weight.

Others contributing to the research include Jolene Nguyen-Cuu and Christine Wells of UCLA. The research was funded by the Hellman Fellows Fund.

http://bit.ly/1PvZDEI

Race Is a Social Construct, Scientists Argue

Racial categories are weak proxies for genetic diversity and need to be phased out

By Megan Gannon, LiveScience on February 5, 2016

More than 100 years ago, American sociologist W.E.B. Du Bois was concerned that race was being used as a biological explanation for what he understood to be social and cultural differences between different populations of people. He spoke out against the idea of "white" and "black" as discrete groups, claiming that these distinctions ignored the scope of human diversity.

Science would favor Du Bois. Today, the mainstream belief among scientists is that race is a social construct without biological meaning. And yet, you might still open a study on genetics in a major scientific journal and find categories like "white" and "black" being used as biological variables.

In an article published today (Feb. 4) in the journal Science, four scholars say racial categories are weak proxies for genetic diversity and need to be phased out. [Unraveling the Human Genome: 6 Molecular Milestones]

They've called on the U.S. National Academies of Sciences, Engineering and Medicine to put together a panel of experts across the biological and social sciences to come up with ways for researchers to shift away from the racial concept in genetics research.

"It's a concept we think is too crude to provide useful information, it's a concept that has social meaning that interferes in the scientific understanding of human genetic diversity and it's a concept that we are not the first to call upon moving away from," said Michael Yudell, a professor of public health at Drexel University in Philadelphia.

Yudell said that modern genetics research is operating in a paradox, which is that race is understood to be a useful tool to elucidate human genetic diversity, but on the other hand, race is also understood to be a poorly defined marker of that diversity and an imprecise proxy for the relationship between ancestry and genetics.

"Essentially, I could not agree more with the authors," said Svante Pääbo, a biologist and director of the Max Planck Institute for Evolutionary Anthropology in Germany, who worked on the Neanderthal genome but was not involved with the new paper.

"What the study of complete genomes from different parts of the world has shown is that even between Africa and Europe, for example, there is not a single absolute genetic difference, meaning no single variant where all Africans have one variant and all Europeans another one, even when recent migration is disregarded," Pääbo told Live Science. "It is all a question of differences in how frequent different variants are on different continents and in different regions."

In one example that demonstrated genetic differences were not fixed along racial lines, the full genomes of James Watson and Craig Venter, two famous American scientists of European ancestry, were compared to that of a Korean scientist, Seong-Jin Kim. It turned out that Watson (who, ironically, became ostracized in the scientific community after making racist remarks) and Venter shared fewer variations in their genetic sequences than they each shared with Kim.

Assumptions about genetic differences between people of different races have had obvious social and historical repercussions, and they still threaten to fuel racist beliefs. That was apparent two years ago, when several scientists bristled at the inclusion of their research in Nicholas Wade's controversial book, "A Troublesome Inheritance" (Penguin Press, 2014), which proposed that genetic selection has given rise to distinct behaviors among different populations. In a letter to The New York Times, five researchers wrote that "Wade juxtaposes an incomplete and inaccurate account of our research on human genetic differences with speculation that recent natural selection has led to worldwide differences in IQ test results, political institutions and economic development."

The authors of the new Science article noted that racial assumptions could also be particularly dangerous in a medical setting.

"If you make clinical predictions based on somebody's race, you're going to be wrong a good chunk of the time," Yudell told Live Science. In the paper, he and his colleagues used the example of cystic fibrosis, which is underdiagnosed in people of African ancestry because it is thought of as a "white" disease.

Mindy Fullilove, a psychiatrist at Columbia University, thinks the changes proposed in the Science article are "badly needed." Fullilove noted that by some laws in the United States, people with one black ancestor of 32 might be called "black," but their 31 other ancestors are also important in influencing their health.

"This is a cogent and important call for us to shift our work," Fullilove said. "It will have an enormous influence. And it will make for better science."

So what other variables could be used if the racial concept is thrown out? Pääbo said geography might be a better substitute in regions such as Europe to define "populations" from a genetic perspective. However, he added that, in North America, where the majority of the population has come from different parts of the world during the past 300 years, distinctions like "African Americans" or "European Americans" might still work as a proxy to suggest where a person's major ancestry originated.

Yudell also said scientists need to get more specific with their language, perhaps using terms like "ancestry" or "population" that might more precisely reflect the relationship between humans and their genes, on both the individual and population level. The researchers also acknowledged that there are a few areas where race as a construct might still be useful in scientific research: as a political and social, but not biological, variable.

"While we argue phasing out racial terminology in the biological sciences, we also acknowledge that using race as a political or social category to study racism, although filled with lots of challenges, remains necessary given our need to understand how structural inequities and discrimination produce health disparities between groups," Yudell said.

http://bit.ly/1Q2S0oD

British Monks Discovered a Curry Recipe in a 200-Year-Old Cookbook

The Portuguese brought the dish to Europe when they began colonizing India

By Danny Lewis

As a dish, the spicy, saucy stew now called curry has deep roots. Archaeologists have uncovered dishware dating back more than 4,500 years in the town of Farmana (a two-hour drive west of Delhi, India, today), covered in the remains of ancient proto-curries made from ingredients like ginger, garlic and turmeric, which are all still used today in curries around the world. Over thousands of years, the stew evolved as trade brought new ingredients and cooking traditions to spice up the meal: Muslim traders introduced meat into curry sometime around the year 1,000, and later, Indians began incorporating cloves imported from Southeast Asia into the meal, Andrew Lawler writes for Slate. But it wasn't until the Portuguese began colonizing India that the spicy dish began to become popular in Europe. Recently, a group of British monks stumbled across a 200-year-old cookbook in their library that, among other things, includes a recipe for chicken curry.

The 1793 cookbook was discovered at Downside Abbey, a Catholic monastery in Somerset. The recipes were written out by hand and compiled instructions for meals made by generations of a wealthy local family, the Western Daily Press reports.

"You can tell it's been very well used," Simon Johnson, the abbey's librarian and archivist, tells the Western Daily Press. "It's in a pretty good condition, but there are a few splatters of something or other all over it...It seems to be a working kitchen cookbook as opposed to being for special occasions."

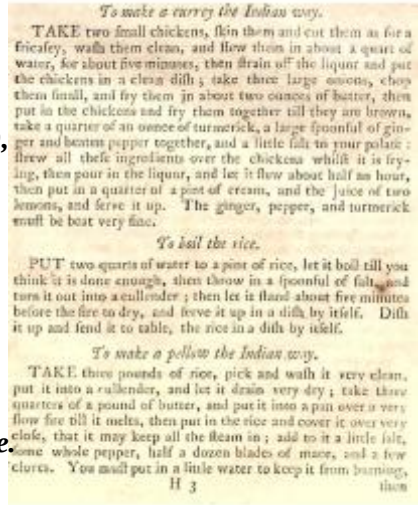
Along with recipes for pigeon pie and turtle soup, the book includes instructions on how to make a simple chicken curry. Because the book was clearly used in a working kitchen, it seems likely that the curry was already a popular dinner choice in England as far back as the 18th century, Nick Rose writes for *Munchies*.

"It's evoked so much interest because it's a Georgian, Regency cookbook," Johnson tells the Western Daily Press. "I think people are generally [interested] in the more domestic parts of history. The social history is forgotten – the day to day running of a house."

The word curry most likely comes from "kari," the Tamil word for "sauce." Over the years, it evolved into the modern "curry" and has became popular in kitchens all over the world. The first known curry recipe written in English was published in a 1747 cookbook written by Hannah Glasse, though it was already quite different from what people in India were making, Anna-Louise Taylor writes for the BBC.

"What had been an Indian sauce to go with rice, became an English stew with a little rice in it," food historian Alan Davidson tells Taylor.

You can check out Glasse's curry recipe here.



To make a currey the Indian way.

TAKE two small chickens, skin them and cut them as for a fricasey, wash them clean, and stew them in about a quart of water, for about five minutes, then drain off the liquor and put the chickens in a clean dish; take three large onions, chop them small, and fry them in about two ounces of butter, then put in the chickens and fry them together till they are brown, take a quarter of an ounce of turmerick, a large spoonful of ginger and beaten pepper together, and a little salt to your palate: strew all these ingredients over the chickens whilst it is frying, then pour in the liquor, and let it stew about half an hour, then put in a quarter of a pint of cream, and the juice of two lemons, and serve it up. The ginger, pepper, and turmerick must be beat very fine.

To boil the rice.

PUT two quarts of water to a pint of rice, let it boil till you think it is done enough, then throw in a spoonful of salt, and turn it out into a cullender; then let it stand about five minutes before the fire to dry, and serve it up in a dish by itself. Dish it up and send it to table, the rice in a dish by itself.

To make a pellow the Indian way.

TAKE three pounds of rice, pick and wash it very clean, put it into a cullender, and let it drain very dry; take three quarters of a pound of butter, and put it into a pan over a very slow fire till it melts, then put in the rice and cover it over very close, that it may keep all the steam in; add to it a little salt, some whole pepper, half a dozen blades of mace, and a few cloves. You must put in a little water to keep it from burning, then

H 3

"To make a currey the Indian way"

2 c. water
1 3-4 lb. chicken, cut-up and skinned
1 ½ large onions (about 12 oz. or 2 ½ c.), chopped small
1 oz. butter (2 T.)
1 T. ground turmeric
1 ½ t. dried, ground ginger
1 ½ t. fine-ground black peppercorns
1 t. kosher salt
1 c. cream
Juice of 1 lemon

Mix turmeric, ginger, pepper, and salt. Put aside.

Skin chicken parts.

In large pot, bring water to boil then add chicken pieces. Bring to simmer, cover (reduce heat further if necessary), and stew (i.e. fricasey) for 5 min. Strain off and save broth (i.e., liquor). Put chicken aside.

Heat a large, wide, heavy bottomed cooking pan over medium-high heat. Melt butter, add onions, and sauté them for about 3 min.

Add chicken to onions and fry together until onions and chicken begin to brown, about 3-4 more minutes.

Sprinkle (i.e. strew) spice mix over chicken. Stir quickly to coat chicken pieces.

Add broth. Stir and scrap any brown bits off the bottom of the pan. Bring to simmer, cover, and stew for about 30 minutes.

Remove lid. At this point the broth should be well reduced, but the onions should still appear wet. Stir in cream and warm through but do not boil.

Remove from heat and add juice of one lemon.

Serve with boiled rice (see recipe below).

"To boil the rice"

1 quart (4 cups) water

1 c. rice

½ t. salt

Bring water to boil (this sounds like a lot of water, but it's not for a rapid boil).

Stir in rice. Boil 18-20 min. uncovered on med-high. Boil should be aggressive, but not raging.

Test for doneness at 18 min. Remove from heat and stir in salt.

Turn rice into colander and let sit for 5 min.

Fluff rice with fork before serving.

http://bit.ly/1Xd6GqP

Umbilical blood cells kill cancer quicker than adult cells

Immature but deadly. Immune cells in fetal blood are better at destroying leukaemia cells than adult cells, tests in mice suggest.

The results are a surprise because fetal immune cells haven't had the lifelong "training" that adult immune cells have had, yet they still seem to recognise and destroy abnormal cells.

People with blood cancers like leukaemia have to undergo chemotherapy to eradicate the blood cells that are causing their cancer. The collateral damage is that most, if not all their healthy blood cells go too.

Stem cells from bone marrow transplants are used to repopulate their circulatory system with healthy blood cells. The transplant has an extra benefit: the new immune cells in the blood can help finish off any residual cancer cells that survived the chemotherapy.

Increasingly, donated umbilical cord blood – which contains fetal stem cells – is being used instead of bone marrow transplants because the risk of rejection is lower with the immature cells. But doctors thought this came at a price – if the immune cells in the cord blood are less aggressive to the recipient, then presumably they are also less aggressive to any residual leukaemia cells.

Not so naive after all

Now, an experiment in mice has shown the exact opposite. "We thought the baby cells were much tamer," says team member Paul Veys of Great Ormond Street Hospital for Children in London. "The assumption has been that they won't fight but it's the complete reverse," he says.

Veys and his colleagues compared the impact of injecting immune cells from adult or cord blood into mice with a form of human blood cancer called B-cell lymphoma. Tumours rapidly disappeared in the mice that received the fetal immune cells, but kept growing in those that got the adult cells.

When the researchers examined tumour samples from the animals before they were destroyed, they found that the fetal cells triggered rapid production of CD4 cells, the white blood cells that orchestrate the immune system response to tumours and viruses. Moreover, the tumours rapidly filled up with CD8 cells, the killer cells that actually destroy cancerous tissue.

The result was a surprise because the assumption has always been that compared with "seasoned" adult cells, the immune cells in the cord blood would be too naive to recognise and kill abnormal cells. "Instead, it seems they can pitch straight in without practice," says Veys. He speculates that the cells may have special immunological abilities that provide immediate protection to a growing fetus. "The implication is that using cord blood may be a better choice to mop up leukaemia," he says. *Journal reference: Blood, DOI: 10.1182/blood-2015-06-654780*

http://nyti.ms/1mn67xl

How a Medical Mystery in Brazil Led Doctors to Zika

A sudden, sharp increase in babies with "no foreheads and very strange heads" was baffling doctors in Brazil. That set off a search for answers that led to a little-known pathogen, the Zika virus.

By DONALD G. McNEIL Jr., SIMON ROMERO and SABRINA TAVERNISE

Something strange was happening last August in the maternity wards of Recife, a seaside city perched on Brazil's easternmost tip, where the country juts into the Atlantic. "Doctors, pediatricians, neurologists, they started finding this thing we never had seen," said Dr. Celina M. Turchi, an infectious diseases researcher at the Oswaldo Cruz Foundation, a prominent scientific institute in Brazil.

"Children with normal faces up to the eyebrows, and then you have no foreheads and very strange heads," she recalled, referring to the condition known as microcephaly. "The doctors were saying, 'Well, I saw four today,' and, 'Oh that's strange, because I saw two.'"

Aside from their alarming appearance, many of the babies seemed healthy. "They cried," Dr. Turchi said. "They breast-fed well. They just didn't seem to be ill." Doctors were stumped.

They did not know it then, but they were seeing the first swell of a horrifying wave. A little-known pathogen — the Zika virus, carried by mosquitoes — had been circulating in Brazil for at least a year. It would later become the chief suspect in the hunt to work out what had happened to those newborns.

Since then, those tiny babies have led the World Health Organization to declare a public health emergency. They have prompted warnings to pregnant women to avoid countries where the virus is circulating, even to refrain from unprotected sex with men who have visited those countries, following a report of sexual transmission of the virus in Dallas last week.

They have led health ministers of five countries to say something so unthinkable that none had ever uttered it before: Women, please delay having children.

The virus now threatens the economies of fragile nations and the 2016 Summer Olympics in Rio de Janeiro. It has opened a new front in the debate in heavily Roman Catholic countries about a woman's right to birth control and abortion.

And the children stricken with microcephaly, or abnormally small heads, have doctors everywhere asking: What is this virus? How could it have been around for almost 70 years without us realizing its power? What do we tell our patients about a bug that can hide in a mosquito's proboscis and a man's semen, even in human saliva or urine? What do we tell young women who ask if their unborn babies are safe?

"This epidemic is an unfolding story," said Dr. Anthony S. Fauci, director of the National Institute of Allergy and Infectious Diseases. "As with Ebola, this virus is something that could exist for years under the radar, and we don't know until we get thousands of cases what it really does." "With Zika, we're seeing new twists and turns every week." To doctors in Recife, whatever was striking the babies seemed to have fallen like a bolt from the blue.

In reality, it had been building for months. It had even been frequently discussed among clinicians — but no one had realized what was on the horizon.

Seeing the Same Symptoms

A year earlier, doctors say, the first patients had started trickling into public hospitals in Natal, capital of the state of Rio Grande do Norte, about 200 miles up the coast from Recife.

It was a few weeks after the 2014 World Cup, and Natal had been one of the host cities of the soccer championship, which draws fans from all over the world.

Many patients lived on the city's margins, others in settlements dotted across the sertão, northeast Brazil's arid hinterland.

Almost all had the same symptoms: a flat pinkish rash, bloodshot eyes, fever, joint pain and headaches. None were desperately ill, but the similarities were striking.

"That scared some patients and doctors, and my team," said Aline Bezerra, a nurse and the municipal epidemiologist. "We knew nothing other than that it might be some kind of light dengue."

Tests ruled that out, along with other common viruses, but the patients kept coming. One day in January 2015, 100 showed up at the state's hospitals.

"We alerted the federal authorities that we were dealing with something urgent and new," said Dr. Kleber Luz, an infectious diseases specialist at the Federal University of Rio Grande do Norte. "But their reaction was sluggish."

By last March, the spread of a "doença misteriosa" — the mystery disease — had become impossible to ignore. It appeared in two more states nearby. Then it reached Salvador, a city of 2.5 million.

Doctors speculated that it was an allergy; that it was roseola, a childhood illness; that it was a new variant of Fifth Disease, a facial rash that gives children a "slapped-cheek" look.

"People were claiming it was polluted water," said Dr. Gúbio Soares, a virologist at the Federal University of Bahia in Salvador. "I began thinking it was something transmitted by mosquitoes." Working in his modest lab with a colleague, Dr. Silvia Sardi, Dr. Soares kept testing blood samples.

Other doctors were doing the same. Over 6,800 samples were tested, according to news reports, from victims ranging from 4 months to 98 years old. Parvovirus, dengue, chikungunya and other suspects were all ruled out.

Finally, in April, Dr. Soares and Dr. Sardi were sure: It was Zika.

"I actually felt a sense of relief," Dr. Soares said. "The literature said it was much less aggressive than viruses we already deal with in Brazil."

In the capital, Brasília, the health minister at the time, Dr. Arthur Chioro, felt the same way. "Zika virus doesn't worry us," he told reporters in May, after the Oswaldo Cruz Foundation had confirmed Dr. Soares's findings. "It's a benign disease." Dengue hemorrhagic fever, on the other hand, killed hundreds of Brazilians each year.

But on ProMED Mail, an online service run by the International Society for Infectious Diseases, the reaction was not so sanguine. "The arrival of Zika virus in Brazil is not good news," wrote Thomas M. Yuill, an emeritus professor of veterinary science and wildlife ecology at the University of Wisconsin-Madison.

Not only did Brazil have "abundant mosquitoes and a large population of susceptible people," he wrote, but so did much of the Americas.

Two weeks earlier, an American mosquito disease expert working in Rio de Janeiro had scoffed on ProMED about an unconfirmed report that it was Zika. The virus fit the symptoms, he wrote, but it was circulating only in Africa and Asia, and in the South Pacific, half a world away, in a different ocean.

An Island-Hopping Virus

For years, virus hunters on ProMED and other outbreak alert networks had been watching, fascinated, as Zika made long, slow and erratic progress eastward across the Pacific, island-hopping as American forces had done during World War II, albeit in reverse.

In 2007, it hit Yap Island, in Micronesia, east of the Philippines and north of Australia. It could have come to Yap from anywhere in Asia.

In October 2013, the Zika virus raced through the many islands of French Polynesia, including Tahiti and Bora Bora. In early 2014, it bounced to the Cook Islands, just to the west, and New Caledonia, close to Australia.

It also leapt to Easter Island, home of the giant stone heads, its official arrival in the Western Hemisphere.

It is still island-hopping. American Samoa and Tonga are having outbreaks now.

Scott C. Weaver, a virologist at the University of Texas Medical Branch in Galveston, wrote an article in 2009 warning that Zika was approaching the Americas. The virus was so obscure that, trying to be helpful in an interview, he explained: "Its closest relative is Spondweni" — a virus named for a place in South Africa that is no longer even on maps.

The Zika Forest in Uganda still is; the virus was discovered there in a monkey in 1947. Since then, the Zika virus had been considered mild compared to its killer cousins: yellow fever, dengue, West Nile and Japanese encephalitis. Until 2013, there was no evidence Zika had ever hospitalized anyone.

Tracking Its Path to Brazil

Back in Brazil, on May 14, it was definite. The mysterious outbreaks — by then in cities all over Brazil, including Rio de Janeiro — had all been caused by Zika.

Who had brought the virus to Brazil? There are two theories.

The first, offered by Brazilian scientists who analyzed airline flight patterns, was that it arrived in the crowds of soccer fans who had flocked to the 12 host cities in the 2014 World Cup. If the Natal outbreak was truly the first, that theory has credence.

A second, proposed by French scientists connected to the Pasteur Institute in Paris who had investigated the outbreak in Polynesia, was that it arrived a few weeks later, during the Va'a World Sprint, a canoe race in Rio that attracted teams from several Polynesian islands.

Since the virus is believed to persist in the blood for up to 10 days, it presumably came from an island then having an outbreak. But in a world as interconnected as ours has become, it may be spread not by a foreigner from faraway lands, but by any international traveler.

The first case of Zika infection detected in New York City was found in December 2013 — six months before the virus is thought to have reached Brazil — in a 48-year-old traveler who lives near Central Park but has asked to remain unidentified.

When he walked into Traveler's Medical Service on Madison Avenue, he had just returned from a long trek through Ecuador, Peru, Bolivia, Chile, Easter Island and Hawaii, with a stopover in French Polynesia.

Dyan J. Summers, the nurse practitioner who first saw him, said he pulled his shirt out of his bluejeans and peeled it off, exposing a pinkish rash he said he had had for 11 days.

"I took one look and said, 'Dengue fever,'" she recalled in an interview last week. "He said, 'I'm not so sure. I think it's Zika.'"

Ms. Summers was startled: "I'd heard of Zika, but nobody was thinking about Zika."

"But this is a very, very bright guy," she continued. "He travels a lot, he knows about safe water and safe altitudes for malaria. He was right on the money, that guy. In Polynesia, he had read articles in the local paper about Zika."

She took blood immediately and again 20 days later, and sent both samples to the Centers for Disease Control and Prevention in Atlanta. Their tests showed that he had antibodies to dengue, West Nile and Zika, but the count of Zika antibodies had shot up.

In researching Zika, Ms. Summers said, her very bright patient had found an article about a scientist in Colorado who had infected his wife with the virus after returning from Africa.

"Because of that paper, I advised him not to have unprotected sex with his common-law wife," she said.

"What's weirder," she added. "He knew there were cases of Guillain-Barré connected to it."

Their exchange was strangely prescient.

At the time, Polynesian and French doctors were just beginning to diagnose Guillain-Barré syndrome, a form of temporary paralysis that starts in the hands and feet. Along with infant microcephaly, the syndrome has turned out to be one of the Zika epidemic's chief fears.

It is an autoimmune attack on nerve cells that can be triggered by several viruses or bacteria. It is usually temporary, though it can last for weeks; but if the paralysis reaches the muscles powering the lungs, and the patient is not quickly put on a respirator, it can kill.

Ms. Summers's caution was right: Last week, the Centers for Disease Control and Prevention gave similar advice about unprotected sex to all Americans.

Hints a Virus Isn't Benign

In May, after it was confirmed that Zika was circulating in Brazil, it took only a few weeks for doctors to suspect that Dr. Chioro, the health minister, had been mistaken. There were hints that the virus was anything but benign.

In Maceió, Recife and other cities, cases of Guillain-Barré began to spike. Dr. María Lúcia Brito, a neurologist in Recife, saw 50 patients with it in 2015, up from 14 the year before.

"It was obvious — a shift occurred when Zika cases started to rise," she said.

Then, in July, a pair of twins were born in Recife. One was healthy; the other was microcephalic. Their parents took them in early August to be examined by Dr. Vanessa van der Linden, a prominent neurologist.

She diagnosed the cause as an infection that had reached one baby in the womb, and tested mother and baby for rubella, syphilis and toxoplasmosis, three known causes of microcephaly.

The results were negative, so she started testing for genetic mutations like Down syndrome.

In September, the Hospital Barão de Lucena, the public hospital in Recife where she works, saw a surge in cases: five microcephalic babies were suddenly in her care. The same thing was happening elsewhere. The hospital where her mother was a pediatric neurologist suddenly had seven cases.

"That's when I thought, 'Something is terribly wrong,' " Dr. van der Linden said.

She soon learned that several of the mothers remembered having the "mystery disease" — the Zika rash — early in their pregnancies.

But tests of the infants for the Zika infection were all negative. Their mothers had been ill months earlier, and in adults the virus usually disappears in 10 days or less. It is still unclear how long it persists in a fetus.

In early October, the national health ministry asked Dr. Turchi, the Oswaldo Cruz Foundation epidemiologist, to investigate. She went to hospitals, including those in Recife. Doctors were running tests for various viruses, but they were all coming up blank.

"The pediatricians were saying, 'We've never seen anything like this,' " she said. "These kids are different. This is something new."

Young Mothers in Shock

Dr. Kátia Petribu, a hospital psychiatrist in Recife, remembers the mothers. They were ghosts — mute, expressionless figures in corridors holding babies whose foreheads seemed to have vanished. Many of the mothers were young, one just 14.

"They were in a state of shock," she said. "They were unable to talk."

Speaking as a medical epidemiologist, I think that the writers of the article confused case control study with cohort study. Comparing kids...

Normally, she worked with patients with obsessive-compulsive disorder. But she decided to refocus on these women, who so clearly needed help.

"They come with nothing," she said. "No food. They travel by bus for hours, arrive at 7 a.m., and wait for hours to be seen."

Many were young rural women with no understanding of why their children looked so different. A 16-year-old showed up with her own mother, who was worried about missing a perfect day to sell cold drinks on the beach.

Dr. Mauricio L. Nogueira, a doctor from southern Brazil who had seen no cases in his region, which is as far from the tropical north as Quebec is from Miami, remembers visiting a hospital in the northern city of Salvador. He is still haunted by what he saw: 25 microcephalic children, all born in the previous 10 days.

That was "really shocking for me," he said. "Until then, I was just reading reports."

One mother, he said, looked up at him and asked, "Hey, doctor, his head is going to grow, right?"

"It was really painful," he said.

Frustration was growing, too, for Dr. Turchi, the epidemiologist. "If we had known what was going on, that would have been one thing," she said. "But there was no book to follow. We had no map."

She shelved her work on the dengue virus and skipped Christmas with her mother.

"I couldn't sleep for several weeks," she said. "It was the most important thing I have seen in my entire career. It was a tragedy, but it was like we were seeing history in front of us, day by day. It was a living history, and we were part of it."

Zika's connection to microcephaly was suspected but very difficult to confirm. Dr. Turchi set up a quick "case control" study, the epidemiologist's classic tool, comparing babies born with the condition and those without it.

Dr. David L. Heymann, chairman of the World Health Organization committee that recommended the declaration of the public health emergency, said in an interview last week that very tool — a case control study following two sets of pregnant women, some who had Zika and some who did not — was what his committee needed to prove whether Zika causes microcephaly, and whether it does so alone or requires a cofactor like a prior infection with dengue.

"Sorting out a rare event will take a lot of women," he said, and they must be followed for months.

At Last, ‘a Road to Follow’

But Dr. Turchi did not have months.

She called every scientist she knew, and they came from all over Brazil. One flew in from London. Dr. Turchi gave the group a name: MERG, the Microcephaly Epidemic Research Group.

“It was like a house on fire — everyone grabs a bucket and does what they can,” she said. Some scientists stayed in her apartment, talking late into the night. “It’s like when you like something and you have people who like the same thing, you can talk for hours without feeling tired,” she said. “It’s like discussing football. You never stop talking. It’s an obsession.”

A turning point came in early November: Dr. Adriana Melo in Paraíba State, just north of Recife, had drawn amniotic fluid from a pregnant woman and found Zika virus in it. Then brain tissue from two stillbirths was tested. Again, Zika.

“At last we had a road to follow,” Dr. Turchi said. “A map.”

One of those who flew in to help in the detective work was Dr. Laura C. Rodrigues, an epidemiologist at the London School of Hygiene and Tropical Medicine on contract to the Pan American Health Organization.

“It was the kind of call where you dropped everything,” she said. “There had never been a congenital malformation by mosquito before, not ever. It was totally outside our experience.”

With the discovery of Zika in malformed fetuses, Dr. Turchi’s team has been able to turn to the kind of task Dr. Heymann described. They have recruited about 1,000 pregnant women with Zika symptoms, and are following healthy and microcephalic newborns in the same areas. They work nights and weekends, eating sandwiches from the institute’s shop or meals of rice, beans and chicken provided by a research assistant’s mother.

There are now so many reported cases of microcephaly that a new problem has arisen: too many false alarms.

Anxious obstetricians across Brazil have reported babies who merely have small heads, or babies whose mothers had other problems, like severe alcoholism or family histories of malformations, conditions that should have excluded them from the research.

Brazil has already changed its definition of a small head, to 32 centimeters around from 33 centimeters, and may revise it again soon.

Dr. Turchi defends those decisions, saying a broad net had to be cast at first because so little was known.

“We didn’t want to get just the severe cases; we wanted to look at the broadest possible spectrum of the disease,” she said. “Then we can narrow it later.”

‘Perfect Epidemic Curve’

Loosed on a continent where no one is immune, Zika has the potential to infect tens of millions of people. It is now being transmitted in 33 countries with about 600 million inhabitants, the W.H.O. says. Health officials in Brazil are investigating thousands of reported cases of microcephaly that may be linked to the virus. Now a bright spot has appeared.

In Recife, and Pernambuco State around it, microcephaly cases have been declining for about three weeks. It is unclear exactly why, but researchers are starting to wonder if the epidemic has peaked. “It looks like a perfect epidemic curve,” Dr. Turchi said. “You see where it started, then went up, and now it’s going down.”

But that decline, and the general sigh of relief it portends, is occurring only in the one spot in the hemisphere where transmission of the virus hit earliest and was most intense. Zika was just getting started there a year before the microcephaly cases began. And now the virus is virtually everywhere south of Florida and Texas.

And Guillain-Barré, the harbinger of microcephaly, is being spotted farther from the epidemic’s epicenter in Brazil. Colombia, Venezuela, Suriname and El Salvador, where mosquitoes thrive year-round, all have reported Guillain-Barré cases. Colombia has “an explosion” of them, its health minister said, with three deaths.

There have also been dozens of confirmed Zika rashes and fevers in the United States, all so far in returning travelers, except for the person infected through sex in Texas by a traveler returning from Venezuela.

Air travel maps show the United States’ potential to be a kind of viral pincushion; Zika may arrive from anywhere. Since four out of five victims never have any symptoms, there is no way to spot it at the border. The C.D.C. thinks it is all but inevitable that there will be at least small outbreaks here. But how far they spread will depend on how aggressively mosquitoes are killed.

Now that the world is alert to the danger and is fighting back, and women are even contemplating delaying pregnancies, scientists say it is unlikely that Brazil’s national nightmare will be repeated elsewhere on such a scale.

In Recife, Dr. Turchi was hopeful.

“I’m more comfortable now,” she said. “I see so many people working as a team and so much international concern. Now it has become clear to the whole world.”

Correction: February 7, 2016 *An earlier version of this article misstated the location of Yap Island, in Micronesia. It is west of the Philippines, not east.*

Donald G. McNeil Jr. reported from New York; Simon Romero from Recife, Brazil; and Sabrina Tavernise from Washington.

http://wapo.st/1QQ4GCi

Colombia: 3,177 pregnant women with Zika; no microcephaly

Colombia's President said there's no evidence Zika has caused any cases of microcephaly in his country

By Ch/jr | AP February 6

BOGOTA, Colombia - Colombia's President Juan Manuel Santos said Saturday that there's no evidence Zika has caused any cases of the birth defect known as microcephaly in his country, though it has diagnosed 3,177 pregnant women with the virus. Santos also announced that a U.S. medical-scientific team will arrive in Colombia to help investigate the mosquito-borne virus.

Brazilian officials say they suspect Zika is behind a seemingly unusual number of microcephaly cases, in which children are born with unusually small heads. The link is not confirmed, but it has helped prompt the World Health Organization to declare an emergency over the virus.

Santos says Zika apparently has affected more than 25,600 Colombians overall.

Colombian officials said Friday that three people had died of the paralyzing Guillain-Barre syndrome they attributed to cases of Zika. To date, the mosquito-borne virus has spread to more than 20 countries in the Americas.

With global concern over the Zika virus growing, health officials are warning pregnant women to be careful about who they kiss and calling on men to use condoms with pregnant partners if they have visited countries where the virus is present. The flurry of recommendations began in Brazil, where a top health official said that scientists have found live virus in saliva and urine samples, and the possibility it could be spread by the two body fluids requires further study.

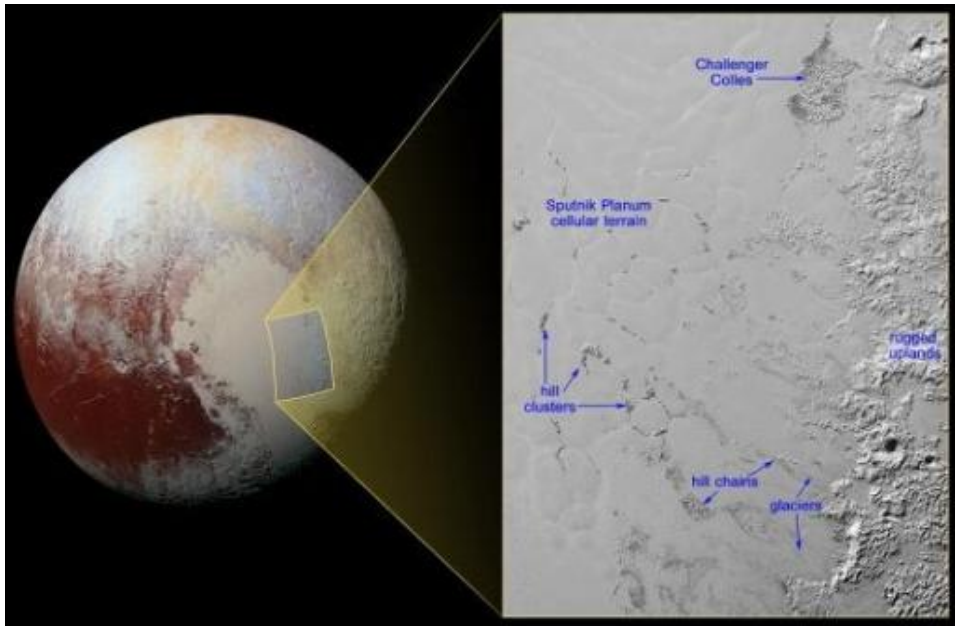
http://bit.ly/1KAB7oQ

NASA announces that Pluto has icebergs floating on glaciers of nitrogen ice

New Horizons continues to return data from the dwarf planet on the edge of the solar system

When Pluto was first discovered in 1930 by Clyde Tombaugh it was a tiny dot that could be seen only by a telescope. All of that changed in July 2015 when NASA's New Horizons space probe flew by the dwarf planet in a signature feat of space exploration and took images and garnered data that has transformed it into a totally alien and fascinating world.

Pluto features plains of nitrogen ice and hills of water ice that are harder than rock. It has a hydrological cycle, of a kind, in which the nitrogen evaporates in the air and then falls as sleet and snow onto Pluto's surface. The dwarf planet is enveloped in a haze of nitrogen gas that ranges in layers from near the surface to 60 miles high.



Pluto from the New Horizons **(Credit NASA)**

The most recent finding from New Horizons show that ice bergs have broken off from the hills surrounding the Sputnik Planum, a glacier of nitrogen ice, and are floating slowly across its surface, eventually to cluster together in places like the Challenger Colles, informally named after the crew of the space shuttle Challenger, which was lost just over 30 years ago. The feature is an especially high concentration of icebergs, measuring 37 by 22 miles. The icebergs float on the nitrogen ice plain because water ice is less dense than nitrogen ice.

New Horizons was launched in 2006 and spent nine and a half years on its voyage to Pluto. The NASA space probe is currently voyaging deeper into the Kuiper Belt, an area cluttered with objects made of ice and rock that are thought to be debris left over from the birth of the solar system. The probe is due to encounter one of these objects sometime in 2019. In the meantime New Horizons continues to transmit data and images that it acquired during its brief flyby of Pluto.

Ironically, when New Horizons rocketed from Earth, Pluto was still considered a planet. But, in a controversial move, a group of astronomers designated it as a "dwarf planet." The matter is still disputed in some corners of the scientific community, with some voices raised to restore Pluto's status as a full-fledged planet.