http://www.eurekalert.org/pub_releases/2013-07/bc-spt_1071713.php

Scientists prove ticks harbor Heartland virus, a recently discovered disease in the United States

3 years after mysterious virus infected 2 Missouri Men, causing severe illness, scientists report isolating pathogen in nearby tick population

DEERFIELD, IL. - Scientists have for the first time traced a novel virus that infected two men from northwestern Missouri in 2009 to populations of ticks in the region, providing confirmation that lone star ticks are carrying the recently discovered virus and humans in the area are likely at risk of infection. The findings were published online today in the American Journal of Tropical Medicine and Hygiene.

Dubbed Heartland virus or HRTV, the infection causes fever, headaches, and low white blood cell and platelet counts. The two men infected in 2009, who live about 70 miles apart, were sufficiently ill to require hospitalization. They eventually recovered, and no other cases have been reported. Disease experts anticipate, however, that more people could become infected.

The Missouri Department of Health and Senior Services is working with the US Centers for Disease Control and Prevention (CDC) to identify additional cases and determine the role of this novel virus as a human pathogen.

"Ten samples of ticks tested positive for the Heartland virus, nine of which were collected from the property of one of the patients and one that came from conservation lands nearby," said Harry M. Savage, PhD, a research entomologist at CDC in Fort Collins, Colorado and the lead author of the paper. "It's pretty strong evidence that the virus is persisting from season to season in tick populations and that these ticks play an important role in disease transmission."

There is no treatment available for HRTV. Unlike other tick-borne diseases like Lyme, ehrlichiosis and Rocky Mountain spotted fever, HRTV is a virus and thus does not respond to antibiotics.

Disease Hunting

HRTV was discovered when a doctor at the hospital treating the two infected men, who had reported being bitten by ticks, sent blood samples to a CDC laboratory in Atlanta for testing. All involved assumed the tests would reveal ehrlichiosis, the tick-borne disease that is most common in the area. Instead, the tests revealed a virus that had never been recognized. Subsequent analysis showed that HRTV belongs to a group of viruses known as phleboviruses, which can be carried by sandflies, mosquitoes or ticks. Savage said there is a separate team of researchers conducting tests with animals in the area in an effort to identify the "reservoir hosts" that are carrying the virus and passing it along to ticks.

Since the HRTV discovery, which was reported in August 2012 in the New England Journal of Medicine, scientists have been combing the area for the source of the infection. Savage and his team, which included investigators from Missouri Western State University, collected 56,428 ticks from April to August of 2012. They employed a variety of collection methods. Flannel pads used in infant bedding were mounted to bamboo poles to act as a sort of fly-paper for ticks. Plastic food containers were "baited" with dry ice, which emits carbon dioxide—a natural attractant for the ticks. Researchers even removed ticks from horses and dogs.

"Finding a virus in ticks requires the collection and testing of large numbers of ticks," Savage said.

The virus infection rate in nymph stage ticks from one farm owned by a patient was about one in five hundred over the study period. Humans are likely to become infected when they are bitten by a tick carrying the virus. Savage said one of the HRTV patients recounted pulling dozens of ticks off his body each night before bed.

According to Savage, the ticks that carry the virus—known as lone star ticks for the single white spot found on females—are common in the area and in many other parts of the country as well.

Currently, there are no reports of HRTV in any other tick populations aside from those isolated in northwestern Missouri. He said it's hard to predict where the virus might be located in the US and if the virus will spread.

HRTV appears to be related to another new disease recently discovered in China, a life-threatening virus called severe fever with thrombocytopenia (SFTSV). SFTSV also appears to be tick-borne, though there is evidence of person-to-person transmission as well.

"This research illustrates the ever-changing world we live in and why we must sustain our nation's investment in research into these types of diseases that the majority of Americans will never hear of," said David H. Walker, MD, president of the American Society of Tropical Medicine and Hygiene, whose research has included a focus on tick-borne diseases. "It is only by getting trained experts into the field and doing the necessary work of collecting and testing thousands of specimens, as these scientists did, that we can be one step ahead of what could become another serious health threat carried by ticks."

To prevent Heartland virus and other diseases spread by ticks, CDC recommends taking the following steps:

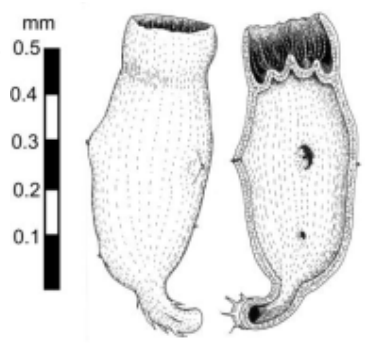
Wear repellent
Check for ticks daily
Shower soon after being outdoors
Call your doctor if you get a fever or rash

http://www.eurekalert.org/pub_releases/2013-07/uoo-got072213.php

Greening of the Earth pushed way back in time

Researchers say a newly named South African fossil points to rising oxygen and life 2.2 billion years ago

EUGENE, Ore. - Conventional scientific wisdom has it that plants and other creatures have only lived on land for about 500 million years, and that landscapes of the early Earth were as barren as Mars.



A new study, led by geologist Gregory J. Retallack of the University of Oregon, now has presented evidence for life on land that is four times as old -- at 2.2 billion years ago and almost half way back to the inception of the planet.

That evidence, which is detailed in the September issue of the journal Precambrian Research, involves fossils the size of match heads and connected into bunches by threads in the surface of an ancient soil from South Africa. They have been named Diskagma buttonii, meaning "disc-shaped fragments of Andy Button," but it is unsure what the fossils were, the authors say.

This is an interpretive view of Diskagma buttonii with exterior view, left, and cross section. The fossils are the size of match heads and were found connected into bunches by threads in the surface of an ancient soil from South Africa.
Courtesy of Gregory Retallack

"They certainly were not plants or animals, but something rather more simple," said Retallack, professor of geological sciences and co-director of paleontological collections at the UO's Museum of Natural and Cultural History. The fossils, he added, most resemble modern soil organisms called Geosiphon, a fungus with a central cavity filled with symbiotic cyanobacteria.

"There is independent evidence for cyanobacteria, but not fungi, of the same geological age, and these new fossils set a new and earlier benchmark for the greening of the land," he said. "This gains added significance because fossil soils hosting the fossils have long been taken as evidence for a marked rise in the amount of oxygen in the atmosphere at about 2.4 billion to 2.2 billion years ago, widely called the Great Oxidation Event."

By modern standards, in which Earth's air is now 21 percent oxygen, this early rise was modest, to about 5 percent oxygen, but it represented a rise from vanishingly low oxygen levels earlier in geological time.

Demonstrating that Diskagma are fossils, Retallack said, was a technical triumph because they were too big to be completely seen in a standard microscopic slide and within rock that was too dark to see through in slabs. The samples were imaged using powerful X-rays of a cyclotron, a particle accelerator, at the Lawrence Berkeley National Laboratory in California.

The images enabled a three-dimensional restoration of the fossils' form: odd little hollow urn-shaped structures with a terminal cup and basal attachment tube. "At last we have an idea of what life on land looked like in the Precambrian," Retallack said. "Perhaps with this search image in mind, we can find more and different kinds of fossils in ancient soils."

In their conclusion, the researchers noted that their newly named fossil Diskagma is comparable in morphology and size to Thucomyces lichenoides, a fossil dating to 2.8 billion years ago and also found in South Africa, but its composition, including interior structure and trace elements, is significantly different.

Diskagma also holds some similarities to three living organisms, which were illustrated microscopically in the study: the slime mold Leocarpus fragilis as found in Oregon's Three Sisters Wilderness; the lichen Cladonia ecmocyna gathered near Fishtrap Lake in Montana; and the fungus Geosiphon pyriformis from near Darmstadt, Germany.

The new fossil, the authors concluded, is a promising candidate for the oldest known eukaryote --an organism with cells that contain complex structures, including a nucleus, within membranes.

"Researchers at the UO are collaborating with scientists from around the world to create new knowledge with far-reaching applications," said Kimberly Andrews Espy, UO vice president for research and innovation, and dean of the graduate school. "This research by Dr. Retallack and his team opens new doors of inquiry about the origins of ancient life on Earth."

The three co-authors with Retallack on the study were: Evelyn S. Krull of the Land and Water Division of the Commonwealth Scientific and Industrial Research Organisation (CSIRO), Australia's national science agency; Glenn D. Thackray, professor of geology at Idaho State University; and Dula Parkinson of the Lawrence Berkeley National Laboratory.

http://scitechdaily.com/two-drug-combination-could-be-the-key-to-curing-cancer/

Two-Drug Combination Could Be the Key to Curing Cancer

Research Shows that Using Two Drugs in Targeted Therapy Can Help Eliminate Cancer

A new study from researchers at Harvard suggests that using a combination of two drugs in a "targeted therapy" effort could effectively cure nearly all cancers.

New research conducted by Harvard scientists is laying out a road map to one of the holy grails of modern medicine: a cure for cancer.

As described in a paper recently published in eLife, Martin Nowak, a professor of mathematics and of biology and director of the Program for Evolutionary Dynamics, and co-author Ivana Bozic, a postdoctoral fellow in mathematics, show that, under certain conditions, using two drugs in a "targeted therapy" — a treatment approach designed to interrupt cancer's ability to grow and spread — could effectively cure nearly all cancers.

Though the research is not a cure for cancer, Nowak said it does offer hope to researchers and patients alike.

"In some sense this is like the mathematics that allows us to calculate how to send a rocket to the moon, but it doesn't tell you how to build a rocket that goes to the moon," Nowak said. "What we found is that if you have a single point mutation in the genome that can give rise to resistance to both drugs at the same time, the game is over. We need to have combinations such that there is zero overlap between the drugs."

Importantly, Nowak said, for the two-drug combination to work, both drugs must be given together — an idea that runs counter to the way many clinicians treat cancer today.

"We actually have to work against the status quo somewhat," he said. "But we can show in our model that if you don't give the drugs simultaneously, it guarantees treatment failure."

In earlier studies, Nowak and colleagues showed the importance of using multiple drugs. Though temporarily effective, single-drug targeted therapy will fail, the researchers revealed, because the disease eventually develops resistance to the treatment.

To determine if a two-drug combination would work, Nowak and Bozic turned to an expansive data set supplied by clinicians at New York's Memorial Sloan-Kettering Cancer Center that showed how patients respond to single-drug therapy. With data in hand, they were able to create computer models of how multidrug treatments would work. Using that model, they then treated a series of "virtual patients" to determine how the disease would react to the multidrug therapy.

"For a single-drug therapy, we know there are between 10 and 100 places in the genome that, if mutated, can give rise to resistance," Nowak explained. "So the first parameter we use when we make our calculations is that the first drug can be defeated by those possible mutations. The second drug can also be defeated by 10 to 100 mutations.

"If any of those mutations are the same, then it's a disaster," he continued. "If there's even a single mutation that can defeat both drugs, that is usually good enough for the cancer — it will become resistant, and treatment will fail. What this means is we have to develop drugs such that the cancer needs to make two independent steps — if we can do that, we have a good chance to contain it."

How good a chance?

"You would expect to cure most patients with a two-drug combination," Bozic said. "In patients with a particularly large disease burden you might want to use a three-drug combination, but you would cure most with two drugs."

The trick now, Nowak and Bozic said, is to develop those drugs.

To avoid developing drugs that are not vulnerable to the same mutation, Bozic said, pharmaceutical companies have explored a number of strategies, including using different drugs to target different pathways in cancer's development.

"There are pharmaceutical companies here in Cambridge that are working to develop these drugs," Nowak said. "There may soon be as many as 100 therapies, which means there will be as many as 10,000 possible combinations, so we should have a good repertoire to choose from.

"I think we can be confident that, within 50 years, many cancer deaths will be prevented," Nowak added. "One hundred years ago, many people died from bacterial infections, and now they would be cured. Today, many people die from cancer, and we can't help them, but I think once we have these targeted therapies, we will be able to help many people — maybe not everyone — but many people."

Publication: Ivana Bozic, et al., "Evolutionary dynamics of cancer in response to targeted combination therapy," eLife, 2013; DOI: 10.7554/eLife.00747

http://www.eurekalert.org/pub_releases/2013-07/asfm-npc072313.php

Natural pest control protein effective against hookworm: A billion could benefit

A benign crystal protein, produced naturally by bacteria and used as an organic pesticide, could be a safe, inexpensive treatment for parasitic worms in humans and provide effective relief to over a billion people around the world.

Researchers from the University of California, San Diego, La Jolla, CA, report on this potentially promising solution in a study published ahead of print in the journal Applied and Environmental Microbiology.

Hookworms, and other intestinal parasites known as helminths infect more than 1 billion people in poverty-stricken, tropical nations, sucking the vitality from the body, and leaving hundreds of millions of children physically and mentally stunted. Current drugs are insufficiently effective, and resistance is rising, but little effort has been made to develop better drugs because the relevant populations do not represent a profitable market for drug companies.

"The challenge is that any cure must be very cheap, it must have the ability to be mass produced in tremendous quantities, safe, and able to withstand rough conditions, including lack of refrigeration, extreme heat, and remote locations," says Raffi Aroian, a researcher on the study.

In earlier research, Aroian and his collaborators described a protein, Cry5B, that can kill intestinal nematode parasites—such as human hookworms—in infected test animals (hamsters). Cry5B belongs to a family of proteins that are generally accepted as safe for humans. These proteins are produced naturally in Bacillus thuringiensis (Bt), a bacterium which is applied to crops as a natural insecticide on some organic farms, and CryB proteins have been engineered into food crops such as corn and rice, to render them pest resistant.

As shown for the first time in this paper, Cry5B can also be expressed in a species of bacterium, Bacillus subtilis, which is closely related to Bacillus thuringiensis, and which is also related to bacteria which are present in some probiotics, says Aroian. In the current research researchers showed that a small dose of Cry5B, expressed in this bacterium can achieve a 93 percent elimination of hookworm parasites from infected hamsters. That, says Aroian, is substantially better than current drugs.

The scientific significance of the research, he says, is that "bacteria similar to those that are food grade—which are cheap and can readily be mass produced--can be engineered to produce molecules that can cure parasitic diseases."

Aroian notes that both the Bill and Melinda Gates Foundation, and the American taxpayer, via the National Institutes of Health, played an essential role in funding the research.

A copy of the manuscript can be found online at http://bit.ly/asmtip0713a. The paper is scheduled to be formally published in the September, 2013 issue of Applied and Environmental Microbiology.

http://www.eurekalert.org/pub_releases/2013-07/nrr-agb072313.php

A ginkgo biloba extract promotes proliferation of endogenous neural stem cells

Ginkgo biloba extract effectively and safely treats memory loss and cognitive impairments in patients with senile dementia

Neural stem cells proliferate in the subventricular zone and hippocampal dentate gyrus of adult mammals. However, the number of endogenous neural stem cells is insufficient to prevent cerebral ischemia/reperfusion injuries such as vascular dementia, so it is important to stimulate endogenous neural stem cell proliferation and differentiation. The ginkgo biloba extract EGb761 effectively and safely treats memory loss and cognitive impairments in patients with senile dementia. Prof. Yuliang Wang and team from Weifang Medical University observed the effects of EGb761 on proliferation of neural stem cells in the subventricular zone and dentate gyrus of rats with vascular dementia. Researchers found that the ginkgo biloba extract EGb761 promoted and prolonged the proliferation of neural stem cells in the subventricular zone and dentate gyrus of rats with vascular dementia. The cells continued to proliferate at 4 months. EGb761 also significantly improved learning and memory in rats with vascular dementia. These findings which were published in the Neural Regeneration Research (Vol. 8, No. 18, 2013) provide a new idea and approach to further explore the induced proliferation of neural stem cells in situ in the treatment of vascular dementia.

" A ginkgo biloba extract promotes proliferation of endogenous neural stem cells in vascular dementia rats " by Jiwei Wang2, Wen Chen1, Yuliang Wang1 (1 Department of Physiology, Weifang Medical University, Weifang 261042, Shandong Province, China; 2 School of Medicine, Shandong University, Jinan 250012, Shandong Province, China).

Wang JW, Chen W, Wang YL. A ginkgo biloba extract promotes proliferation of endogenous neural stem cells in vascular dementia rats. Neural Regen Res. 2013;8(18):1655-1662.

http://phys.org/news/2013-07-biologists-highly-complex-aquatic-cyanobacteria.html

Biologists discover highly complex communication system in aquatic cyanobacteria

Land plants can "see," but can microscopic plants see better?

New research from Indiana University has uncovered a give-and-take communication system between and within photoreceptors in freshwater-dwelling cyanobacteria that works at a level of complexity beyond those seen in plants or other organisms. The new work by IU Bloomington biologist David M. Kehoe and Ph.D. student Adam N. Bussell—published in the Proceedings of the National Academy of Sciences—not only identifies a new type of photoreceptor that can sense four different light colors at once, but for the first time demonstrates the regulation of the abundance of one photoreceptor by a second in cyanobacteria.

Kehoe, a professor in the College of Arts and Sciences' Department of Biology, said identification of both hierarchical control of one photoreceptor over another and of a complex communication system within a single photoreceptor that is simultaneously integrating information about four different light colors opens a new chapter in understanding cyanobacteria complexity and how their photoreceptors work.

Cyanobacteria are of tremendous evolutionary and ecological importance because the 3 billion-year-old aquatic bacteria, historically known as "blue-green algae," were the first group of organisms on Earth to release oxygen during photosynthesis. Their photosynthetic activity currently accounts for about 40 percent of our planet's oxygen production.

In analyzing the newly discovered photoreceptor, called IflA (influenced by far-red light), and its ability to use two distinct photosensory domains to respond to blue, green, red and far-red light, Kehoe and Bussell believe they may have uncovered unique and advantageous machinery for life in aquatic environments that are not needed on land.

The IflA photoreceptor attaches two chromophores using three cysteine residues (amino acids) and senses blue, green, red and far-red light. Replacements of these cysteines in various combinations create a rainbow of seven different colored IflAs (upper-right panels), which are visible in pelleted cells when each protein is overexpressed in E. coli , which is normally brown (upper-left panel).

"Unlike air, water absorbs far-red light very well, red light well, green light moderately and blue light poorly," Bussell said. "So aquatic cyanobacteria face big changes in the relative amounts of these colors just by moving up or down a few meters in the water column. These are not conditions that land plants have to face."

Living in a world where light color ratios and irradiance levels vary greatly at different depths, and where additional information about depth, competing photosynthesizers, time of day and other parameters can affect survival, the benefits of a complex communication system may be essential. In this case, it's the ability of the two IflA photoreceptor domains—the portion of a protein's sequence with a discrete structure and function that is independent of the rest of the protein chain—to affect each other's behavior during light sensing.

"Our results show that these domains are interacting with each other and integrating information about four different colors of light simultaneously," Kehoe said. "This kind of complexity has never been found in plants or other organisms. Although plants are known to integrate blue light information with red and far-red light information, multiple photoreceptors are used for this, and the information is integrated downstream of the photoreceptors, during signal transduction."

Specifically, the researchers isolated the IflA gene from cells of the filamentous, freshwater cyanobacterium Fremyella diplosiphon and then overexpressed various regions of the IflA protein in the bacteria Escherichia coli that had been engineered to produce the light-sensing chromophores needed by IflA for photoperception. They found the three amino acids that needed to bind the two chromophores used by IflA and that one protein domain sensed red and far-red light, while the other sensed blue and green light. Mutating these amino acids alone and in combination led to the creation of seven different colored forms of IflA when each was overexpressed in E. coli cells (see photo).

Adding to the complexity of this system is the first description, in bacteria, of the strong regulation of the abundance of IflA by a second photoreceptor called RcaE, which senses red and green light and represses the amount of IflA in red light. "The RcaE-mediated increase in IflA abundance as the ratio of green to red light increases resulted in IflA being about seven or eight times more abundant in green light than in red light," Kehoe said. "This has not ever been seen in prokaryotic phytochrome family members and demonstrates that complex interactions exist between these photoreceptors as well."

More information: www.pnas.org/content/early/2013/07/10/1303371110.abstract

http://scitechdaily.com/stem-cell-study-reveals-the-brain-protective-powers-of-astrocytes/

Stem Cell Study Reveals the Brain-Protective Powers of Astrocytes

A newly published stem cell study from UC Davis uncovers the brain-protective powers of astrocytes, finding that astrocytes can protect brain tissue and reduce disability due to stroke and other ischemic brain disorders.

Sacramento, California – One of regenerative medicine's greatest goals is to develop new treatments for stroke. So far, stem cell research for the disease has focused on developing therapeutic neurons — the primary movers of electrical impulses in the brain — to repair tissue damaged when oxygen to the brain is limited by a blood clot or break in a vessel. New UC Davis research, however, shows that other cells may be better suited for the task. Published today in the journal Nature Communications, the large, collaborative study found that astrocytes — neural cells that transport key nutrients and form the blood-brain barrier — can protect brain tissue and reduce disability due to stroke and other ischemic brain disorders.

"Astrocytes are often considered just 'housekeeping' cells because of their supportive roles to neurons, but they're actually much more sophisticated," said Wenbin Deng, associate professor of biochemistry and molecular medicine at UC Davis and senior author of the study. "They are critical to several brain functions and are believed to protect neurons from injury and death. They are not excitable cells like neurons and are easier to harness. We wanted to explore their potential in treating neurological disorders, beginning with stroke."

Deng added that the therapeutic potential of astrocytes has not been investigated in this context, since making them at the purity levels necessary for stem cell therapies is challenging. In addition, the specific types of astrocytes linked with protecting and repairing brain injuries were not well understood.

The team began by using a transcription factor (a protein that turns on genes) known as Olig2 to differentiate human embryonic stem cells into astrocytes. This approach generated a previously undiscovered type of astrocyte called Olig2PC-Astros. More importantly, it produced those astrocytes at almost 100 percent purity.

The researchers then compared the effects of Olig2PC-Astros, another type of astrocyte called NPC-Astros and no treatment whatsoever on three groups of rats with ischemic brain injuries. The rats transplanted with Olig2PC-Astros experienced superior neuroprotection together with higher levels of brain-derived neurotrophic factor (BDNF), a protein associated with nerve growth and survival. The rats transplanted with NPC-Astros or that received no treatment showed much higher levels of neuronal loss.

To determine whether the astrocytes impacted behavior, the researchers used a water maze to measure the rats' learning and memory. In the maze, the rats were required to use memory rather than vision to reach a destination. When tested 14 days after transplantation, the rats receiving Olig2PC-Astros navigated the maze in significantly less time than the rats that received NPC-Astros or no treatment.

The investigators used cell culture experiments to determine whether the astrocytes could protect neurons from oxidative stress, which plays a significant role in brain injury following stroke. They exposed neurons co-cultured with both types of astrocytes to hydrogen peroxide to replicate oxidative stress. They found that, while both types of astrocytes provided protection, the Olig2PC-Astros had greater antioxidant effects. Further investigation showed that the Olig2PC-Astros had higher levels of the protein Nrf2, which increased antioxidant activity in the mouse neurons.

"We were surprised and delighted to find that the Olig2PC-Astros protected neurons from oxidative stress in addition to rebuilding the neural circuits that improved learning and memory," said Deng.

The investigators also investigated the genetic qualities of the newly identified astrocytes. Global microarray studies showed they were genetically similar to the standard NPC-Astros. The Olig2PC-Astros, however, expressed more genes (such as BDNF and vasoactive endothelial growth factor, or VEGF) associated with neuroprotection. Many of these genes help regulate the formation and function of synapses, which carry signals between neurons.

Additional experiments showed that both the Olig2PC-Astros and NPC-Astros accelerated synapse development in mouse neurons. The Olig2PC-Astros, however, had significantly greater protective effects over the NPC-Astros.

In addition to being therapeutically helpful, the Olig2PC-Astros showed no tumor formation, remained in brain areas where they were transplanted and did not differentiate into other cell types, such as neurons.

"Dr. Deng's team has shown that this new method for deriving astrocytes from embryonic stem cells creates a cell population that is more pure and functionally superior to the standard method for astrocyte derivation," said Jan Nolta, director of the UC Davis Institute for Regenerative Cures. "The functional improvement seen in the brain injury models is impressive, as are the higher levels of BDNF. I will be excited to see this work extended to other brain disease models such as Huntington's disease and others, where it is known that BDNF has a positive effect."

Deng added that the results could lead to stem cell treatments for many neurodegenerative diseases.
"By creating a highly purified population of astrocytes and showing both their therapeutic benefits and safety, we open up the possibility of using these cells to restore brain function for conditions such as Alzheimer's disease, epilepsy, traumatic brain disorder, cerebral palsy and spinal cord injury," said Deng.
Peng Jiang of UC Davis and Shriners Hospitals for Children was the study lead author. Deng and Jiang's co-authors were Chen Chen, Olga Chechneva, Seung-Hyuk Chung and David Pleasure of UC Davis and Shriners Hospitals for Children; Quanguang Zhang and Ruimin Wang of the Medical College of Georgia; Mahendra Rao of the National Institutes of Health (NIH) Center for Regenerative Medicine; and Ying Liu of the University of Texas Health Science Center.
This research was funded in part by the NIH (grants R01NS061983, R01ES015988 and R01NS025044), National Multiple Sclerosis Society, Shriners Hospitals for Children, California Institute for Regenerative Medicine, Memorial Hermann Foundation (Staman Ogilvie Fund) and the Bentsen Stroke Center.
Publication: Peng Jiang, et al., "hESC-derived Olig2+ progenitors generate a subtype of astroglia with protective effects against ischaemic brain injury," Nature Communications 4, Article number: 2196; doi:10.1038/ncomms3196

http://www.eurekalert.org/pub_releases/2013-07/uons-hlt072213.php

HPV's link to esophageal cancer

The human papillomavirus (HPV) triples the risk of people developing yet another cancer, oesophageal squamous cell carcinoma (OSCC), according to research led by University of New South Wales (UNSW) academics.

In addition to causing cervical, anal and genital cancers, HPV has more recently been found to cause some head and neck cancers. "One of the main issues is this form of oesophageal cancer is usually diagnosed quite late and so has a very high mortality," says the first author of the paper, Dr Surabhi Liyanage, a PhD candidate with the School of Public Health and Community Medicine, UNSW Medicine.
OSCC is the most common of two types of oesophageal cancer. While it is rare in Australia, it is the sixth highest cause of cancer-related deaths world-wide. It is particularly prevalent in China, South Africa and Iran among men in their mid-70s to 80s. It is unknown why the prevalence is so high in those countries, but it is thought to be linked to dietary, lifestyle and environmental factors. "HPV is another factor which we can add to a long list of causes of OSCC," says Dr Liyanage. "Smoking and alcohol are the main causes, as well as the consumption of extremely hot liquids, lots of red meat and possibly environmental toxins in the diet."
The findings, published today in PLOS ONE, could have implications for vaccination programs around the world. "This is an important new finding which resolves a previous uncertainty," says senior author, UNSW Professor Raina MacIntyre. "Given that the most common two cervical cancer-causing HPVs are now preventable by early vaccination, this may be significant in countries where OSCC is frequently found," says Professor MacIntyre, head of the School of Public Health and Community Medicine.
"In China, it is one of the leading causes of cancer death, so Chinese health authorities could consider this in any deliberations they are having about potential benefits of HPV vaccination in their population," she says.
Currently, HPV vaccinations are used most commonly in young people in developed countries to prevent cervical cancer. "Time will tell whether our universal HPV vaccination program has any additional benefit in prevention of cancers other than cervical cancer," says Professor MacIntyre.
"The findings from this meta-analysis should rekindle the debate about looking at the potential causative role for oncogenic HPVs in oesophageal cancer," says another of the authors and a leader in HPV vaccination, Dr Suzanne Garland, from the Royal Women's Hospital in Melbourne.
"These findings will assist the expert group, International Agency for Research on Cancer (IARC), which examines evidence for potential oncogenic roles in various cancers," says Dr Garland. "We look forward to a potential review by IARC of the meta-analysis and other studies in establishing a role or not for HPV."

http://www.scientificamerican.com/article.cfm?id=deadly-pig-virus-slips-through-us-borders

Deadly Pig Virus Slips through U.S. Borders

Researchers worldwide are racing to track the spread of a coronavirus that causes diarrhea and vomiting in pigs; it poses no threat to humans

By Beth Mole and Nature magazine | Wednesday, July 24, 2013 | 8

A lethal virus that causes diarrhea and vomiting in pigs has entered the United States and has been found in 14 states. With the country's $97-billion pork industry standing to lose millions of dollars in the event of a mass outbreak, scientists are working to track the virus and prevent its spread, even as they try to understand how it passed through biosecurity defenses in the first place.
"How this virus got here, that's the million-dollar question," says James Collins, director of the Veterinary Diagnostic Laboratory at the University of Minnesota in St Paul.

The pathogen, a type of coronavirus called porcine epidemic diarrhea virus (PEDV), was first identified in the United Kingdom in 1971, and it caused mass epidemics in Europe in the 1970s and 1980s. As pigs there developed immunity, the virus petered out and now causes only occasional, isolated outbreaks. It has since spread to Asia, where it has been considered endemic since 1982, causing substantial economic losses to pork producers. The virus can spread quickly by a fecal–oral route and infect entire herds. And although adult pigs typically recover, PEDV can kill 80–100% of the piglets it infects. The virus poses no health threat to humans.

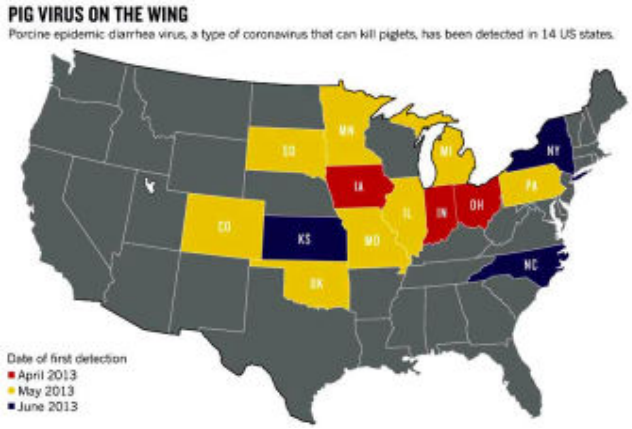
The US Department of Agriculture (USDA) had tried to keep PEDV and other diseases out of the country by restricting imports of pigs and pork products from certain nations, such as China. But on 10 May, the Veterinary Diagnostic Laboratory at Iowa State University in Ames confirmed that PEDV had infected pigs in Iowa, the leading producer of US pork. The lab then screened samples taken earlier from other states and found a case from Ohio submitted on 16 April that is now the earliest known US detection of PEDV, according to Gregory Stevenson, a pathologist at Iowa State. The fact that the virus has now spread to 14 states in total is a sign that the outbreak is still flaring and could become an epidemic (see 'Pig virus on the wing').

"It's a real threat," says Lisa Becton, a veterinary surgeon and director of swine health information at the National Pork Board, an industry group in Des Moines, Iowa.

To understand the virus's enigmatic US entry, scientists are sequencing viral DNA isolated from pigs and comparing it with PEDV variants from elsewhere in the world. Researchers are also trying to create rapid diagnostic tests and vaccines to prevent the virus from spreading. The National Pork Board has approved $800,000 to fund research and education.

But PEDV must first be grown in labs — a notoriously difficult exercise because the pathogen thrives in the specific conditions found in pig guts. Researchers in Europe and Asia have already managed to infect cells, but only after years of working with the virus. In the United States, the same import restrictions that were set up to help to prevent PEDV from entering the country have made it difficult to import the necessary lab materials for working with the virus, such as vaccines, infected cells and pig antibodies.

"What's hampering the research is that we don't have regents," says Linda Saif, a virologist at Ohio State University in Wooster. Access to the virus and good tests in hand "would have helped us identify which herds have been exposed, and one could have imposed more stringent control measures", she says.



SOURCE: US Department of AGRICULTURE

The USDA's National Veterinary Services Laboratories in Ames is one of just a few US facilities to have grown the virus successfully. But it had a head start: the lab imported the virus around 15 years ago from Asia, after a lengthy security-clearance process, in preparation for just such an outbreak. Lab scientists have spent recent months tweaking cell-culture protocols, and plan to distribute the virus to researchers on request in the coming weeks.

In the meantime, other research groups have focused on detecting viral DNA in sick pigs and on sequencing viral genes. In August, a team led by Douglas Marthaler, a scientist at the University of Minnesota's Veterinary Diagnostic Laboratory, will publish the sequence of a virus genome taken from a Colorado farm. They found it to be 99.4% identical to a Chinese strain of PEDV. On the basis of that sequence, many researchers suspect that the virus originated in China, but Marthaler says that he is surprised by the level of similarity, because he would have expected the US virus to have evolved more in the time since it arrived.

In any case, he says, the potential origin of the virus does not say anything about the route that it took to reach the United States. Canada, the main source of pigs entering the United States, does not import pigs from China either. And although researchers know that the virus can be transported in feces, they do not know how long it can survive outside pigs' intestines, so it is unclear if a dirty boot, a contaminated package or an illegal import carried PEDV into the country.

Vets say that pig farmers are now restricting access to farms, and are cleaning pig manure more carefully off their clothes and trucks as they move between barns. And researchers still hope that they can elucidate the virus's international and domestic path by looking for subtle evolutionary changes in viral genome sequences of samples from Asia and different US states.

Saif, who has feared such an outbreak for decades, wonders what the virus will do next. Agriculture experts speculate that it may be more stable in cooler temperatures, and thus more dangerous, making the current outbreak a mild precursor to what could come in the winter. “We have to be vigilant,” says Saif.

http://www.bbc.co.uk/news/health-23436589

Cat allergy research offers new clues

Scientists have discovered how allergic reactions to cats are triggered, raising hopes of preventative medicine.

By Helen Briggs BBC News

A University of Cambridge team has identified how the body's immune system detects cat allergen, leading to symptoms such as coughing and sneezing. New treatments to block this pathway raise hopes of developing medicines to protect sufferers, they say.

Allergy UK says the research is "a big step forward" in understanding how cat allergen causes allergic reactions.

Researchers led by Dr Clare Bryant of the University of Cambridge studied proteins found in particles of cat skin, known as cat dander, which is the most common cause of cat allergy. They found that cat allergen activates a specific pathway in the body, once in the presence of a common bacterial toxin.

This triggers a large immune response in allergy sufferers, causing symptoms such as coughing, wheezing, sneezing and a runny nose.

Dr Bryant told BBC News: "We've discovered how the cat allergy proteins activate the host immune cells.

"By understanding the triggering mechanism, there are now drugs that have been designed that are in clinical trials for other conditions, such as sepsis, that could potentially then be used in a different way to treat cat allergy and to prevent cat allergy."

Cat allergies

Cats are among the most common culprits for pet allergies
People with cat allergies are allergic to proteins in the cat's saliva, urine, and dander (dried flakes of skin)
Symptoms of a cat allergy can develop in a few minutes or take hours to appear
Some people with allergic asthma have severe flare-ups after coming in contact with a cat

The charity Allergy UK said the research, published in Journal of Immunology, was a big step forward in understanding how cat allergen causes such severe allergic reactions.

"Cat allergen is particularly difficult to avoid as it is a 'sticky' molecule that is carried into every building on people's shoes and clothes," said director of clinical services Maureen Jenkins. "It can also still be found in a home, on the walls and ceiling or fittings, even a few years after a cat has ceased to live there.

"Therefore, this new information identifying the specific receptor interaction in the immune system could pave the way for treatments for those with persistent disease triggered by cat allergen and, in the future, potentially dog and house dust mite allergen."

Allergic reactions happen when the immune system overreacts to a perceived danger. Instead of responding to a harmful virus or bacteria, it misidentifies allergens, such as cat dander, and mounts an immune response.

The research was funded by the Wellcome Trust and the Medical Research Council.

http://www.eurekalert.org/pub_releases/2013-07/uos-stc072513.php

Suffocating tumors could lead to new cancer drugs

Scientists have discovered a new molecule that prevents cancer cells from responding and surviving when starved of oxygen and which could be developed into new treatments for the disease, according to new research published in the Journal of the American Chemical Society* today (Friday).

Cancer Research UK scientists at the University of Southampton found that this molecule targets the master switch -- HIF-1 -- that cancer cells use to adapt to low oxygen levels, a common feature in the disease.

The researchers uncovered a way to stop cancer cells using this switch through an approach called 'synthetic biology'. By testing 3.2 million potential compounds, made by specially engineered bacteria, they were able to find a molecule that stopped HIF-1 from working.

All cells need a blood supply to provide them with the oxygen and nutrients they require to survive. Cancer tumours grow rapidly and as the tumour gets bigger it outstrips the supply of oxygen and nutrients that the surrounding blood vessels can deliver.

But, to cope with this low-oxygen environment, HIF-1 acts as a master switch that turns on hundreds of genes, allowing cancer cells to survive. HIF-1 triggers the formation of new blood vessels around tumours, causing more oxygen and nutrients to be delivered to the starving tumour, which in turn allows it to keep growing.

Dr Ali Tavassoli, a Cancer Research UK scientist whose team discovered and developed the compound at the University of Southampton, said: "We've found a way to target the steps that cancer cells take to survive and we hope that our research will one day lead to effective drugs that can stop cancers adapting to a low oxygen environment, stopping their growth. The next step is to further develop this molecule to create an effective treatment."

Dr Julie Sharp, senior science information manager at Cancer Research UK, said: "Finding ways to disrupt the tools that cancer cells use to adapt and grow when starved of oxygen has been a hot topic in cancer research, but finding drugs that do this effectively has proved elusive.
"For the first time our scientists have found a way to block a master switch controlling cells response to low levels of oxygen -- an important step towards creating drugs that could halt cancer in its tracks."

http://www.eurekalert.org/pub_releases/2013-07/aafc-whl072213.php

Women's height linked to cancer risk

The taller a postmenopausal woman is, the greater her risk for developing cancer, according to a study published in Cancer Epidemiology, Biomarkers & Prevention, a journal of the American Association for Cancer Research.

PHILADELPHIA - Height was linked to cancers of the breast, colon, endometrium, kidney, ovary, rectum, and thyroid, as well as to multiple myeloma and melanoma, and these associations did not change even after adjusting for factors known to influence these cancers, in this study of 20,928 postmenopausal women, identified from a large cohort of 144,701 women recruited to the Women's Health Initiative (WHI).
"We were surprised at the number of cancer sites that were positively associated with height. In this data set, more cancers are associated with height than were associated with body mass index [BMI]," said Geoffrey Kabat, Ph.D., senior epidemiologist in the Department of Epidemiology and Population Health at Albert Einstein College of Medicine of Yeshiva University in New York, N.Y. "Ultimately, cancer is a result of processes having to do with growth, so it makes sense that hormones or other growth factors that influence height may also influence cancer risk."
Some genetic variations associated with height are also linked to cancer risk, and more studies are needed to better understand how these height-related genetic variations predispose some men and women to cancer, according to the authors. Kabat and colleagues used data from the WHI, a large, multicenter study that recruited postmenopausal women between the ages 50 and 79, between 1993 and 1998. At study entry, the women answered questions about physical activity, and their height and weight were measured.
The researchers identified 20,928 women who had been diagnosed with one or more invasive cancers during the follow-up of 12 years. To study the effect of height, they accounted for many factors influencing cancers, including age, weight, education, smoking habits, alcohol consumption, and hormone therapy.
They found that for every 10-centimeter (3.94 inches) increase in height, there was a 13 percent increase in risk of developing any cancer. Among specific cancers, there was a 13 percent to 17 percent increase in the risk of getting melanoma and cancers of the breast, ovary, endometrium, and colon. There was a 23 percent to 29 percent increase in the risk of developing cancers of the kidney, rectum, thyroid, and blood.
Of the 19 cancers studied, none showed a negative association with height. Because the ability to screen for certain cancers could have influenced the results, the researchers added the participants' mammography, Pap, and colorectal cancer screening histories to the analyses and found the results remained unchanged.
"Although it is not a modifiable risk factor [A modifiable risk factor can be changed, controlled, or treated, e.g., diet, lifestyle. Height is a non-modifiable risk factor because it cannot be changed], the association of height with a number of cancer sites suggests that exposures in early life, including nutrition, play a role in influencing a person's risk of cancer," said Kabat. "There is currently a great deal of interest in early-life events that influence health in adulthood. Our study fits with this area."

http://www.eurekalert.org/pub_releases/2013-07/cp-bns071813.php

Bad night's sleep? The moon could be to blame

Many people complain about poor sleep around the full moon, and now a report appearing in Current Biology, a Cell Press publication, on July 25 offers some of the first convincing scientific evidence to suggest that this really is true.

The findings add to evidence that humans—despite the comforts of our civilized world—still respond to the geophysical rhythms of the moon, driven by a circalunar clock.
"The lunar cycle seems to influence human sleep, even when one does not 'see' the moon and is not aware of the actual moon phase," says Christian Cajochen of the Psychiatric Hospital of the University of Basel.
In the new study, the researchers studied 33 volunteers in two age groups in the lab while they slept. Their brain patterns were monitored while sleeping, along with eye movements and hormone secretions.
The data show that around the full moon, brain activity related to deep sleep dropped by 30 percent. People also took five minutes longer to fall asleep, and they slept for twenty minutes less time overall. Study participants felt as though their sleep was poorer when the moon was full, and they showed diminished levels of melatonin, a hormone known to regulate sleep and wake cycles.

"This is the first reliable evidence that a lunar rhythm can modulate sleep structure in humans when measured under the highly controlled conditions of a circadian laboratory study protocol without time cues," the researchers say.
Cajochen adds that this circalunar rhythm might be a relic from a past in which the moon could have synchronized human behaviors for reproductive or other purposes, much as it does in other animals. Today, the moon's hold over us is usually masked by the influence of electrical lighting and other aspects of modern life. The researchers say it would be interesting to look more deeply into the anatomical location of the circalunar clock and its molecular and neuronal underpinnings. And, they say, it could turn out that the moon has power over other aspects of our behavior as well, such as our cognitive performance and our moods.
Current Biology, Cajochen et al.: "Evidence that the lunar cycle influences human sleep."

http://www.eurekalert.org/pub_releases/2013-07/haog-rla072513.php

Rapamycin: Limited anti-aging effects

Substance makes mice live longer, but hardly slows down the aging process

The body's repair mechanisms begin to fail with increasing age. As a result, signs of wear and tear appear and the risk for many diseases, including Alzheimer's disease, diabetes, cardiovascular disorders and cancer, increases. "Current efforts to develop therapies against age-related diseases target these disorders one by one," says Dr. Dan Ehninger, research group leader at the DZNE site in Bonn. "Influencing the aging process itself may be an alternative approach with the potential to yield broadly effective therapeutics against age-related diseases." The findings are reported in the current issue of the "Journal of Clinical Investigation" (published online on July 25, 2013).
In this context, the substance rapamycin is noteworthy. Rapamycin is used in recipients of organ transplants, as it keeps the immune system in check and can consequently prevent rejection of the foreign tissue. In 2009, US scientists discovered another effect: Mice treated with rapamycin lived longer than their untreated counterparts. "Rapamycin was the first drug shown to extend maximal lifespan in a mammalian species. This study has created quite a stir," says Ehninger.
For Ehninger and his team, this finding motivated further studies: "We wanted to address if rapamycin slows down aging in mice or, alternatively, if it has an isolated effect on lifespan - without broadly modulating aging."

Not a youth elixir

Together with scientists from the Helmholtz Zentrum München and other colleagues, Ehninger's group investigated if rapamycin influences aging in mice. The results are sobering: "Our results indicate that rapamycin extends lifespan, but it has only limited effects on the aging process itself," is Ehninger's summary of the findings. "Most aging traits were not affected by rapamycin treatment. Although we did observe positive effects on some aging traits, such as memory impairments and reduced red blood cell counts, our studies showed that similar drug effects are also seen in young mice, indicating that rapamycin did not influence these measures by slowing aging, but rather via other, aging-independent, mechanisms."
The researchers believe that such aging-independent drug effects also underlie rapamycin's effect on lifespan. "We assume that the lifespan of mice is extended because rapamycin inhibits tumor formation. This is a well-known rapamycin effect, which we were able to confirm. Cancer is the leading cause of death in the relevant mouse strains" says the specialist in molecular medicine. "Rapamycin, therefore, seems to have isolated effects on specific life-limiting pathology, but lacks broad effects on aging in mice."

A comprehensive assessment of aging

The research team assessed more than 150 traits, which typically change during the course of aging. These analyses included an assessment of vision, reflexes, cardiovascular function, learning and behavior, immune functions and the integrity of the arterial wall, to just name a few. "Aging is a complex process, which cannot be captured by assessing a single parameter. This is why we analysed a large number of structural and functional signs of aging," explains Ehninger. "The present study is one of the most comprehensive assessments of a putative anti-aging intervention."
The analysis comprised three different age cohorts, in which rapamycin treatment was either initiated in young adulthood, in midlife or late in life. "At the time, the US study showed that rapamycin extends lifespan irrespective of whether the treatment is given to young or aged animals," says the Bonn-based researcher. "We, therefore, chose a study design, in the context of which we also investigated rapamycin's effects on different age groups. This enabled us to examine whether the possible effects of rapamycin depend on the age at which treatment started."
The animals were genetically identical twin mice. All of the animals received rapamycin regularly over a period of approximately one year. For each age cohort there was also a control group, which did not take the substance.

Need for comprehensive analyses

"Generally speaking, our studies show that a number of different parameters have to be considered when assessing the efficacy of possible anti-aging interventions. The interpretation of the data depends heavily on the overall picture of findings. Lifespan measures alone are not a reliable indicator of anti-aging effects," emphasises Ehninger. "This makes the search for anti-aging medicines tedious, but it is also very promising, because such substances could open up new possibilities for medicine. However, this is still some way off."
"Rapamycin extends murine lifespan but has limited effects on aging ", Frauke Neff, Diana Flores-Dominguez etc., Journal of Clinical Investigation (published online on July 25, 2013), http://dx.doi.org/10.1172/JCI67674

http://www.bbc.co.uk/news/health-23431833

Study confirms cancers-family link

Having cancer in the family can increase your chances of developing not only the same cancer but other types too, research suggests.

By Helen Briggs BBC News

A study of 23,000 people in Italy and Switzerland found that for each of 13 cancers, close relatives had an increased risk of the same disease. But there was also evidence that a family history of one cancer could significantly raise the risk of others. Cancer charities say risk depends on genes, lifestyle and environment.
The research, published in the journal Annals of Oncology, followed 12,000 patients with cancer at different sites in the body. They were compared with 11,000 people without cancer.
The researchers collected information on family history of cancer, particularly in a first-degree relative (those who share about 50% of their genes - namely a parent, sibling or child).
They found people with a first-degree relative with cancer of the larynx had triple the normal risk of developing oral and pharyngeal cancer. Those closely related to someone with oral and pharyngeal cancer had a fourfold increased risk of oesophageal cancer, while breast cancer doubled the risk of ovarian cancer for female family members. Men had a 3.4-fold increased risk of prostate cancer if a first-degree relative had bladder cancer.
The research also confirmed some known cancer risks. They include a raised risk of women developing breast cancer if they have a family history of bowel cancer.

Cancers studied
Mouth and pharynx
Nasopharynx
Oesophagus
Stomach
Bowel
Liver
Pancreas
Larynx
Breast
Womb
Ovaries
Prostate
Kidney

Lifestyle factors
Study leader Dr Eva Negri, of the Mario Negri Institute for Pharmacological Research in Milan, Italy, told BBC News: "If you have a relative with one type of cancer your risk of the same type of cancer is increased.
"What this study has highlighted is that sometimes if you have a relative with one cancer your risk of another cancer can be increased. "The relative risk of a different cancer generally tends to be lower than for the same cancer." In some cases, the links between different cancers may be due to shared environmental factors, such as family smoking and drinking habits, she said. But there was also evidence of genetic factors affecting multiple cancer sites in the body.
Jessica Harris, Cancer Research UK's senior health information manager, said cancer risk is determined by a combination of genes we inherit from our parents, our lifestyles, and our environment. "Whether or not someone in your family has had cancer, living a healthy life can really help to stack the odds in our favour, and reduce the risk of cancer," she said. "The main things you can do are to be a non-smoker, cut down on alcohol, and stay in shape by being active and eating a balanced diet."
Eluned Hughes, from the charity Breakthrough Breast Cancer, said some breast cancers do run in the family, however it was vital that women remembered most cases were not hereditary. "In order to fully understand the causes of breast cancer, we need to study more women over a longer period of time," she said.

http://bit.ly/13GKQlQ

Light completely stopped for a record-breaking minute

The fastest thing in the universe has come to a complete stop for a record-breaking minute. At full pelt, light would travel about 18 million kilometres in that time – that's more than 20 round trips to the moon.

09:00 25 July 2013 by Jacob Aron

"One minute is extremely, extremely long," says Thomas Krauss at the University of St Andrews, UK. "This is indeed a major milestone."
The feat could allow secure quantum communications to work over long distances.
While light normally travels at just under 300 million metres per second in a vacuum, physicists managed to slow it down to just 17 metres per second in 1999 and then halt it completely two years later, though only for a fraction of a second. Earlier this year, researchers kept it still for 16 seconds using cold atoms.

Stripy light

To break the minute barrier, George Heinze and colleagues at the University of Darmstadt, Germany, fired a control laser at an opaque crystal, sending its atoms into a quantum superposition of two states. This made it transparent to a narrow range of frequencies. Heinze's team then halted a second beam that entered the crystal by switching off the first laser and hence the transparency.

The storage time depends on the crystal's superposition. A magnetic field extends it but complicates the control laser configuration. Heinze's team used an algorithm to "breed" combinations of magnet and laser, leading them to one that trapped light for a minute.

They also used the trap to store and then retrieve an image consisting of three stripes. "We showed you can imprint complex information on your light beam," says Heinze.

Tens of seconds of light storage are needed for a device called a quantum repeater, which would stop and then re-emit photons used in secure communications, to preserve their quantum state over long distances.

It should even be possible to achieve longer light storage times with other crystals, says Heinze, as they have pushed their current material close to its physical limit.

Journal reference: Physical Review Letters, doi.org/m86

http://www.wired.com/wiredscience/2013/07/cre-chronology-nature/

Where 'Nightmare Bacteria' Came From, And How Our Inattention Helped Them Emerge

Form of antibiotic resistance that is relatively new, and has suddenly emerged as much more serious is still relatively unknown

By Maryn McKenna

Cast your minds back a few months ago, to when the director of the US Centers for Disease Control and Prevention announced, "We have a very serious problem" with "nightmare bacteria," and the chief medical officer of the United Kingdom backed him up a few days later, describing a "ticking time bomb" that threatens national security as seriously as terrorism.

Both public health chiefs were talking about a form of antibiotic resistance that is relatively new, and has suddenly emerged as much more serious — yet that the general public, and even much of the medical establishment, knows relatively little about. The acronym for this resistance is CRE, for carbapenem-resistant Enterobacteriaceae; breaking it down, that's a family of gut-dwelling bacteria, a very common cause of hospital infections, that no longer respond to a group of last-ditch antibiotics called carbapenems.

Because they are effectively untreatable, CREs are very serious — as many as half of the patients who contract these infections die from them — and they are surprisingly common. In the United States, they have been found in 42 states to this point, in more than 4 percent of all hospitals, and more than 18 percent of "long term acute care" institutions, which offer critical care such as ventilator support for patients who need that level of care for months.

When Dr. Tom Frieden of the CDC and Professor Dame Sally Davies of the UK made their announcements in March — in language that (I suspect) was intended to jar people out of complacency about the future unreliability of antibiotics — a fairly common reaction among my readers was, "How on earth did this happen?" It seemed a reasonable question.

This week, with the help of excellent editors at the journal Nature, I've tried to provide an answer. In a 3,000-word piece, I trace the emergence and spread of CREs from their first sighting by the CDC in a sample from 1996. The story is told in episodes extracted from the chronology of CREs' rampage around the globe; from each episode, we've tried to extract a vital lesson. Among them:

When carbapenem resistance was first sighted — by chance, in a small surveillance program — there was no federal funding or political will to expand surveillance to see if the problem was occurring widely.

When the problem came to notice again — several years later and hundreds of miles away — the automated testing systems medicine uses to detect resistance had not been tuned to this new threat.

Once hospitals realized how persistent these resistant bugs were in the hospital environment, they took steps to corral patients and rigorously clean rooms — but they could do nothing to prevent the spread of these same bugs within the nursing homes and long-term care institutions from which they accept patients, undermining all their protective care.

When it became recognized that these highly resistant bacteria could travel asymptomatically in the guts of unknowing patients, not enough thought was given to how far they might spread, and so CREs disseminated across the globe.

There's much more, and I hope you'll take a look. If you can't spare the time, you can get the tl;dr in the first 5 minutes of Nature's weekly podcast. ***"Too long; didn't read"***

http://www.medscape.com/viewarticle/808417?src=rss

Dietary Guidelines Aim to Reduce Alzheimer's Risk

New dietary guidelines http for the prevention of Alzheimer's disease have been developed by the Physicians Committee for Responsible Medicine (PCRM).

Sue Hughes

PCRM is a nonprofit organization that advocates preventive medicine, especially good nutrition; conducts clinical research; and advocates for higher ethical standards in research, according to their Web site. The new guidelines were released last week at the International Conference on Nutrition and the Brain held in Washington, DC, sponsored jointly by PCRM and George Washington University School of Medicine.

PCRM president and lead author of the guidelines, Neal Barnard, MD, said, "The current generation of clinicians is in a battle over food — especially Alzheimer's-promoting foods, such as those which contain saturated and trans fats. We potentially have the capabilities to prevent a disease that is poised to affect 100 million people worldwide by 2050. Why wait?"

The guidelines are very similar to the habits that prevent heart disease in that they recommends avoiding saturated and trans fats, grounding the diet in plant-based foods, and adding sources of vitamin E and B. "Combining this diet with physical exercise and avoiding excess metals, such as iron and copper in multivitamins, can maximize protection for the brain," Dr. Barnard claimed.

The 547 healthcare providers who attended the conference sampled the dietary recommendations themselves by eating meals such as roasted broccoli salad, spiced chickpea curry, baby bok choy, and blueberry sorbet.

Several Alzheimer's experts asked to comment on the guidelines for *Medscape Medical News* all had similar opinions: that the recommendations were for a healthy diet and exercise, which was always good general advice, but that high levels of evidence that following these guidelines would definitively reduce Alzheimer's risk are lacking.

The 7 Dietary Principles to Reduce Alzheimer's Risk
1. Minimize saturated fats and trans fats.
2. Vegetables, legumes (beans, peas, and lentils), fruits, and whole grains should be the primary staples of the diet.
3. One ounce of nuts or seeds (one small handful) daily provides a healthful source of vitamin E.
4. A reliable source of vitamin B_{12}, such as fortified foods or a supplement providing at least 2.4 µg per day for adults) should be part of the daily diet.
5. Choose multivitamins without iron and copper, and consume iron supplements only when directed by your physician.
6. Avoid the use of cookware, antacids, baking powder, or other products that contribute dietary aluminium.
7. Engage in aerobic exercise equivalent to 40 minutes of brisk walking 3 times per week.

The Evidence

Dr. Barnard cited several studies that supported these guidelines. For example, in the Chicago Health and Aging Project, individuals who consumed the most saturated fat (around 25 g each day) were 2 to 3 times more likely to develop Alzheimer's disease than those who consumed only half that amount.

He acknowledged that not all studies agree.

For example, a Dutch study found no protective effect of avoiding saturated fats, although the population was somewhat younger than that in the Chicago study He suggested that high-fat foods and/or the increases in cholesterol they may cause can contribute to the production of β-amyloid plaques in the brain. High-fat foods also increase the risk for obesity and type 2 diabetes, common risk factors for Alzheimer's disease, he added.

A large study of Kaiser Permanente patients showed that participants with total cholesterol levels above 250 mg/dL in midlife had a 50% higher risk for Alzheimer's disease 3 decades later compared with participants with cholesterol levels below 200 mg/dL, Dr. Barnard reported. And he noted that the *APOE* ε4 allele, which is strongly linked to Alzheimer's risk, produces a protein that plays a key role in cholesterol transport.

On the recommendation for vegetables, legumes, fruits, and whole grains, Dr. Barnard pointed out that these foods are rich in vitamins, such as folate and vitamin B_6, that play protective roles for brain health. Studies of Mediterranean-style diets and vegetable-rich diets, such as the Chicago Health and Aging Project, have shown reduced risk for cognitive problems compared with other dietary patterns.

Dr. Barnard cited an Oxford University study of older people with elevated homocysteine levels and memory problems, in which supplementation with B vitamins improved memory and reduced brain atrophy.

On potentially harmful metals, he noted that excessive iron and copper have been linked to cognitive problems. And while the role of aluminium in Alzheimer's disease remains controversial, he pointed out that aluminum has been demonstrated in the brains of individuals with Alzheimer's disease, and studies in the United Kingdom and France have found increased Alzheimer's prevalence in areas where tap water contained higher aluminium concentrations.

He added that several studies have found a correlation between exercise and a reduced risk for Alzheimer's.

Experts Cautious

Commenting on the guidelines for *Medscape Medical News*, Heather Snyder, PhD, director of scientific operations at the Alzheimer's Association, said, "There is evidence supporting the idea that physical activity reduces risk of Alzheimer's. And it is always good advice to keep to a healthy diet and stay active. So we would endorse those views. But there is really not enough evidence to support some of the other specific aspects in these guidelines to the level of prescription given."

She added that some positive studies suggest benefits with certain foods/vitamins, but there were also others that show completely opposite results. "So it is difficult to reach a consensus on individual foods."

She noted that benefits have been suggested with some foods, such as dark leafy vegetables (eg, spinach, which is low in saturated fat and has antioxidant effects), but these are not definitive.

Malaz Boustani, MD, Indiana University Center for Aging Research, Indianapolis, had broadly similar views. "Unfortunately there is no high level evidence to support these guidelines," he commented. "But the adverse effects of following such a diet would be minimal. It is a very healthy diet that they are recommending so there would be no harm in it. But whether it provides value in reducing risks of Alzheimer's is not known. Yes, there are some observational studies suggesting that some of these guidelines may be beneficial. But there is no hard evidence from randomized trials."

Dr. Boustani pointed out that recently, the National Institute on Aging reviewed the literature and did not find any strong evidence to support issuing these type of guidelines and that in a randomized controlled trial vitamin E did not slow Alzheimer's disease or the underlying pathology.

"I would say the major caveat would be whether people can afford to pay for the supplements recommended. If you can afford it then there is no harm in trying it. It is always good to eat a diet low in saturated fat and trans fat and do physical exercise every day. We should all do that anyway," he added.

Samuel Gandy, MD, Mount Sinai Center for Cognitive Health, New York, said, "The diet recommendations makes good sense but must be subjected to one or more randomized clinical trials before one can say that they truly modify the risk for Alzheimer's."

"The new dietary guidelines are mainly based on findings from observational studies and seem reasonable," added Joe Verghese, MBBS, Albert Einstein College of Medicine, New York. "However, there is a paucity of proof from well-conducted clinical trials that supplementation prevents Alzheimer's disease in elders without nutritional deficiencies." *The full guidelines document is available at the PCRM Web site.*

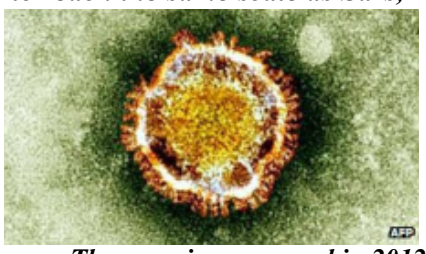
http://www.bbc.co.uk/news/health-23447687

Mers: New virus 'not following Sars' path'

The new Mers virus, which has killed half of those infected, is "unlikely" to reach the same scale as Sars, ministers in Saudi Arabia say.

By James Gallagher Health and science reporter, BBC News

Most of the 90 Mers cases reported so far have been in Saudi Arabia. Mers is from the same group of viruses as the common cold and Sars, which killed 774 people. However, a detailed analysis of the Saudi cases, published in Lancet Infectious Diseases, did warn of "major gaps" in understanding of the virus.



The new virus emerged in 2012

The Middle East respiratory-syndrome coronavirus (Mers) emerged in 2012 and has infected 90 people worldwide, 45 of them have died. The global concern is that cases could spread much further, echoing the Sars outbreak. Cases have been centred on the Middle East - with patients in Jordan, Qatar, Saudi Arabia and the United Arab Emirates. Additional cases in France, Germany, Italy, Tunisia and the UK have all been linked to travel to the Middle East.

Researchers in Saudi Arabia have published details of the 47 cases reported in the country. They suggest a pattern of mostly older men being infected. Most cases were also in people with other medical problems, more than two-thirds of the reported cases also had diabetes.

Low threat

The lead researcher and Deputy Minister for Public Health, Prof Ziad Memish, said: "Despite sharing some clinical similarities with Sars, there are also some important differences. "In contrast to Sars, which was much more infectious especially in healthcare settings and affected the healthier and the younger age group, Mers appears to be more deadly, with 60% of patients with co-existing chronic illnesses dying, compared with the 1% toll of Sars.

"Although this high mortality rate with Mers is probably spurious due to the fact that we are only picking up severe cases and missing a significant number of milder or asymptomatic cases. "So far there is little to indicate that Mers will follow a similar path to Sars."
A report earlier this month showed that the virus struggled to spread in people. However, it and the latest Saudi investigation both highlighted the need to find where the virus was coming from.
Prof Memish's report said: "Reducing the rate of introduction of Mers coronavirus into human beings is unpredictable because the source of the virus is not yet known. "We are searching vigorously for the source."

http://www.sciencedaily.com/releases/2013/07/130726092427.htm

Chronic Fatigue Syndrome: Inherited Virus Can Cause Cognitive Dysfunction and Fatigue

A common virus, Human Herpesvirus 6, is the possible cause of some CFS cases

Many experts believe that chronic fatigue syndrome (CFS) has several root causes including some viruses. Now, lead scientists Shara Pantry, Maria Medveczky and Peter Medveczky of the University of South Florida's Morsani College of Medicine, along with the help of several collaborating scientists and clinicians, have published an article in the Journal of Medical Virology suggesting that a common virus, Human Herpesvirus 6 (HHV-6), is the possible cause of some CFS cases.
Over 95 percent of the population is infected with HHV-6 by age 3, but in those with normal immune systems the virus remains inactive. HHV-6 causes fever and rash (or roseola) in infants during early childhood, and is spread by saliva. In immunocompromised patients, it can reactivate to cause neurological dysfunction, encephalitis, pneumonia and organ failure.
"The good news reported in our study is that antiviral drugs improve the severe neurological symptoms, including chronic pain and long-term fatigue, suffered by a certain group of patients with CFS," said Medveczky, who is a professor of molecular medicine at USF Health and the study's principal investigator. "An estimated 15,000 to 20,000 patients with this CFS-like disease in the United States alone may ultimately benefit from the application of this research including antiviral drug therapy."
The link between HHV-6 infection and CFS is quite complex. After the first encounter, or "primary infection," all nine known human herpesviruses become silent, or "latent," but may reactivate and cause diseases upon immunosuppression or during aging. A previous study from the Medveczky laboratory showed that HHV-6 is unique among human herpesviruses; during latency, its DNA integrates into the structures at the end of chromosomes known as telomeres.
Furthermore, this integrated HHV-6 genome can be inherited from parent to child, a condition commonly referred to as "chromosomally integrated HHV-6," or CIHHV-6. By contrast, the "latent" genome of all other human herpesviruses converts to a circular form in the nucleus of the cell, not integrated into the chromosomes, and not inheritable by future generations.
Most studies suggest that around 0.8 percent of the U.S. and U.K. population is CIHHV6 positive, thus carrying a copy of HHV-6 in each cell. While most CIHHV-6 individuals appear healthy, they may be less able to defend themselves against other strains of HHV-6 that they might encounter. Medveczky reports that some of these individuals suffer from a CFS-like illness. In a cohort of CFS patients with serious neurological symptoms, the researchers found that the prevalence of CIHHV-6 was over 2 percent, or more than twice the level found in the general public. In light of this finding, the authors of the study suggest naming this sub-category of CFS "Inherited Human Herpesvirus 6 Syndrome," or IHS.
Medveczky's team discovered that untreated CIHHV-6 patients with CFS showed signs that the HHV-6 virus was actively replicating: determined by the presence of HHV-6 messenger RNA (mRNA), a substance produced only when the virus is active. The team followed these patients during treatment, and discovered that the HHV-6 mRNA disappeared by the sixth week of antiviral therapy with valganciclovir, a drug used to treat closely related cytomegalovirus (HHV-5). Of note, the group also found that short-term treatment regimens, even up to three weeks, had little or no impact on the HHV-6 mRNA level.
The investigators assumed that the integrated virus had become reactivated in these patients; however, to their surprise, they found that these IHS patients were infected by a second unrelated strain of HHV-6.
The USF-led study was supported by the HHV-6 Foundation and the National Institutes of Health.
Further studies are needed to confirm that immune dysregulation, along with subsequent chronic persistence of the HHV-6 virus, is the root cause of the IHS patients' clinical symptoms, the researchers report.
Shara N. Pantry, Maria M. Medveczky, Jesse H. Arbuckle, Janos Luka, Jose G. Montoya, Jianhong Hu, Rolf Renne, Daniel Peterson, Joshua C. Pritchett, Dharam V. Ablashi, Peter G. Medveczky. Persistent human herpesvirus-6 infection in patients with an inherited form of the virus. Journal of Medical Virology, 2013; DOI: 10.1002/jmv.23685

http://www.sciencedaily.com/releases/2013/07/130726131256.htm

Buying a Used Car? Be Sure to Flatter the Seller

Consumers set high prices when selling their possessions because they feel threatened, according to a new study in the Journal of Consumer Research.

"When consumers consider selling a product they own, they feel threatened by the impending loss. In order to counter this threat, they increase the product's value," write authors Promothesh Chatterjee (University of Kansas), Caglar Irmak (University of Georgia), and Randall L. Rose (University of South Carolina).

Due to a phenomenon called the "endowment effect," consumers seek much higher prices when selling a product they own than they would be willing to pay to purchase the same product.

In one study, consumers were assigned either a seller or buyer role and presented with a coffee mug. Sellers were told they could keep the mug or sell it, while buyers were asked to evaluate the mug. Then, both sellers and buyers were shown a series of words on a computer screen consisting of threat-related words (endanger), neutral words (wood), and non-words (tlun). Sellers responded to threat-related words much more quickly than buyers, and this difference in their response time led to significantly higher selling prices compared to buying prices.

Consumers should be aware that sellers can feel threatened when parting with even the most mundane possessions. Complimenting or flattering a seller can make them feel less threatened and lead them to lower their selling prices.

"Affirming a seller leads to elimination of the endowment effect. Buyers may want to affirm sellers to make them feel less threatened by the loss of a possession and therefore willing to set lower prices. Next time you are buying a second-hand car, for example, you may want to start the negotiation by telling the car owner what a wonderful family she has," the authors conclude.

Promothesh Chatterjee, Caglar Irmak, and Randall L. Rose. The Endowment Effect as Self-Enhancement in Response to Threat. Journal of Consumer Research, October 2013

http://bit.ly/1brBOP6

Tiny Patch Hides You From Mosquitoes

Brightly colored little patches that stick on clothing or backpacks could represent an effective new way to fight mosquitoes and the deadly illnesses they spread.

Jul 26, 2013 02:36 PM ET // by Alyssa Danigelis

A California-based startup is close to getting the patches produced and distributed in areas where they're needed the most.

Current mosquito repellents can be cumbersome, expensive, limited and in some cases even toxic. Bed nets can only do so much and nobody enjoys getting sprayed with who-knows-what just to keep the bugs away for a brief spell. A company in Riverside, California called Kite thinks it has the answer in the form of a 1.5-inch square patch that goes on clothing, backpacks, baby carriers and other items to make the wearer invisible to mosquitoes.

Photo: The Kite Mosquito Patch goes on clothing and backpacks to hide the wearer from mosquitoes. **Credit: Kite**

The company reports that its patch can protect the wearer for up to 48 hours. Usually I'd be highly skeptical of any company making such a claim but Kite appears to have solid credentials. They're using technology developed by entomologist Anandasankar Ray at the University of California, Riverside, and they're supported by grants from the Bill and Melinda Gates foundation and the National Institutes of Health.

Although the formula that Kite uses in its patches is patent-pending and proprietary, the company does say that its scientists developed the formula from food-grade, FDA-approved compounds and International Fragrance Association-approved fragrances that "specifically target the mosquitoes' receptor neurons used to detect carbon dioxide."

The carbon dioxide part is crucial because that's how mosquitoes can find us, suck our blood, and spread deadly diseases. "For more than half of the world's population, primarily in Africa, Southeast Asia and South America, effective mosquito control means the difference between life and death," Kite co-founder Torrey Tayenaka said in a video about the patch.

So far Kite's ingredients appear to be working in the lab. Next, the company wants to do a six-month pilot program in malaria-stricken parts of Uganda. After that they're aiming for EPA approval on the compounds so they can begin producing enough patches for global distribution. While they initially set out to raise $75,000 through Indiegogo.com, Kite has raised more than $268,000 so far and still has 35 days left in their campaign.

Mosquito-repellent patches are desperately needed in foreign countries, but mosquito-borne illness is no joke in parts of North America, either. Cases of West Nile Virus are rising again in the U.S., according to the CDC. When it comes down to it, I'd rather go for an effective little sticky patch than a strange chemical cloud.

http://www.eurekalert.org/pub_releases/2013-07/uouh-ssa072713.php

Shocking: Surgical anesthetic appears to treat drug-resistant depression

University of Utah study shows isoflurane may provide alternative to electroconvulsive therapy

Salt Lake City - Although electroconvulsive therapy (ECT) has long been considered the most effective treatment of medication-resistant or refractory depression, millions of people who might benefit don't take advantage of it because of the treatment's side effects and public misperception of the procedure.

If the results of a campuswide collaboration of University of Utah researchers are borne out by larger studies and trials, patients with refractory depression might one day have an alternative that is as effective as ECT but without the side effects – the surgical anesthetic drug isoflurane.

"We need to expand our research into a larger, multicenter trial, but if the results of our pilot study pan out, it would change the face of treating depression," says Howard R. Weeks, M.D., assistant professor of psychiatry and first author on a study published in Friday, July 26, 2013, in the journal PLOS One online.

Also known as shock therapy, ECT is effective in 55 percent to 90 percent of depression cases, with significant reductions in symptoms typically occurring within two to four weeks. When medications work, they can take six to eight weeks to become effective. But ECT is associated with side effects including amnesia, concentration and attention problems, and other cognitive issues. Many people also mistakenly believe ECT is painful and causes brain damage, which has given the treatment a social stigma that makes millions of patients reluctant to have the therapy. Isoflurane potentially offers an alternative to ECT that could help many of those people, according to Weeks and his colleagues from eight University of Utah departments and programs.

In a pilot study with 20 patients who received ECT treatments compared to eight patients who received the isoflurane treatments, the researchers found that both therapies provided significant reduction in symptoms of depression. Immediately following the treatments, ECT patients showed declines in areas of memory, verbal fluency, and processing speed. Most of these ECT-related deficits did resolve by four weeks. However, autobiographical memory, or recall of personal life events, remained below pretreatment levels for ECT patients four weeks after the treatment. In contrast, the patients treated with isoflurane showed no real impairment but instead had greater improvements in cognitive testing than ECT patients both immediately and four weeks after the treatments.

Recently, another anesthetic, Ketamine, has drawn interest as a potential treatment for depression. But studies so far have not shown long-lasting effects from using Ketamine. In contrast, isoflurane showed continued antidepressant effects four weeks after the treatments.

In the mid-1980s, researchers in Europe studied isoflurane as a potential depression therapy. Later studies by other scientists, however, failed to confirm the results of the original work and isoflurane research fell out of favor. But these later studies didn't adhere to the first study's protocol regarding type of anesthetic, dosing size and number of treatments, according to Weeks, and he believes that's why isoflurane's antidepressant effects weren't confirmed in subsequent trials. For their research, Weeks and his University colleagues followed the original study's protocol. "Our data reconfirm that isoflurane had an antidepressant effect approaching ECT with less adverse neurocognitive effects, and reinforce the need for a larger clinical trial," the researchers wrote.

Researchers don't know what produces the relief of depression symptoms from ECT or isoflurane. Weeks believes further study might identify a molecular pathway that both therapies target and is responsible for the improvement in depression. One common effect of both ECT and isoflurane treatments is a brief state of low electrical activity in which the brain becomes unusually quiet. ECT induces a seizure to reach that state, but isoflurane does not. After inhaling the anesthesia, patients are "under" for about 45 minutes, with 15 minutes of that time being a deep state of unconsciousness, according to Weeks. This period of electrical rest for the brain may be a potential explanation for why ECT and isoflurane improve depression.

If isoflurane proves to be a viable alternative to ECT, a device invented by three University of Utah anesthesiology faculty members can make the anesthetic an even more attractive therapy. The Aneclear™ device (Anecare, Salt Lake City, UT) invented by Dwayne R. Westenskow, Ph.D., Derek J. Sakata, M.D., and Joseph A. Orr, Ph.D., from the University of Utah Department of Anesthesiology, uses hyperventilation and allows patients to rebreathe their own carbon dioxide (C02). Hyperventilation removes anesthesia from the lungs and C02 encourages blood flow to the brain, which encourages quicker removal of anesthetic. The Aneclear™ also minimizes or even eliminates vomiting, nausea and extreme fatigue that some patients experience from anesthesia.

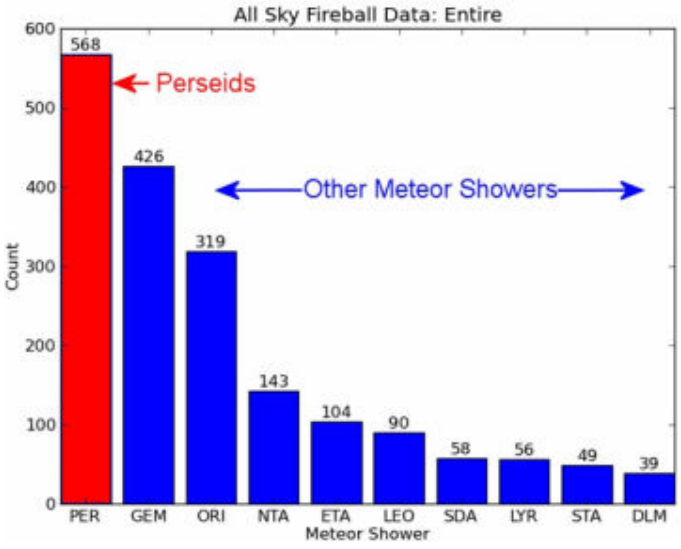
"With the Aneclear™, we can wake people up from the anesthesia much quicker," Weeks says. "This makes the treatment a potentially viable clinical treatment by reducing the time required in an operating room."
Weeks and his co-researchers now are looking for grants to fund a larger study that will include several U.S. centers.
Other authors on this study include Scott C. Tadler, M.D., Kelly W. Smith, M.D., Kathleen C. Light, Ph.D., Michael K. Cahalan, M.D., Derek J. Sakata, M.D., Eli Iacob, Ph.D., Joshua D. Landvatter, M.A., and Alan R. Light, Ph.D., all Department of Anesthesiology; Andrea T. White, Ph.D., Department of Exercise and Sport Science; Gordon J. Chelune, Ph.D., Department of Neurology; Yana Suchy, Ph.D., Departments of Psychology and Neurology; Elaine Clark, Ph.D., and Mikala Saccoman, Ph.D., Department of Educational Psychology; and Lowry A. Bushnell, M.D., Department of Psychiatry.

http://scitechdaily.com/astronomers-prepare-for-a-fireball-display-from-the-perseid-meteor-shower/

Astronomers Prepare for a Fireball Display from the Perseid Meteor Shower

On the nights of August 12th and 13th, astronomers will have the opportunity to watch the Perseid meteor shower, a meteor shower that has recently produced more fireballs than any other.

New research from NASA's Meteoroid Environment Office identifies the Perseids as the "fireball champion" of annual meteor showers. This year's Perseid display peaks on August 12th and 13th.
In astronomy, there's nothing quite like a bright meteor streaking across the glittering canopy of a moonless night sky. The unexpected flash of light adds a dash of magic to an ordinary walk under the stars.
New research by NASA has just identified the most magical nights of all.
"We have found that one meteor shower produces more fireballs than any other," explains Bill Cooke of NASA's Meteoroid Environment Office. "It's the Perseid meteor shower, which peaks on August 12th and 13th."
Using a network of meteor cameras distributed across the southern USA, Cooke's team has been tracking fireball activity since 2008, and they have built up a database of hundreds of events to analyze. The data point to the Perseids as the 'fireball champion' of annual meteor showers.
A fireball is a very bright meteor, at least as bright as the planets Jupiter or Venus. They can be seen on any given night as random meteoroids strike Earth's upper atmosphere. One fireball every few hours is not unusual. Fireballs become more numerous, however, when Earth is passing through the debris stream of a comet. That's what will happen this August.
The Perseid meteor shower comes from Comet Swift-Tuttle. Every year in early- to mid-August, Earth passes through a cloud of dust sputtered off the comet as it approaches the sun. Perseid meteoroids hitting our atmosphere at 132,000 mph produce an annual light show that is a favorite of many backyard sky watchers.
Cooke thinks the Perseids are rich in fireballs because of the size of the parent comet.
"Comet Swift-Tuttle has a huge nucleus–about 26 km in diameter," comments Cooke. "Most other comets are much smaller, with nuclei only a few kilometers across. As a result, Comet Swift-Tuttle produces a large number of meteoroids, many of which are large enough to produce fireballs."



Astronomers Identify a Meteor Shower that Produces More Fireballs than Any Other
Since 2008, the Perseids have produced more fireballs than any other annual meteor shower. The Geminids are a close second, but they are not as bright as the Perseids. "The average peak magnitude for a Perseid observed by our cameras is -2.7; for the Geminids, it is -2," explains Bill Cooke. "So on average, Geminid fireballs are about a magnitude fainter than those in the Perseids."
Cooke recommends looking on the nights of August 12th and 13th between the hours of 10:30 PM to 4:30 AM local time. Before midnight the meteor rate will start out low, then increase as the night wears on, peaking before sunrise when the constellation Perseus is high in the sky.
For every fireball that streaks out of Perseus, there will be dozens more ordinary meteors.
"Get away from city lights," advises Cooke. "While fireballs can be seen from urban areas, the much greater number of faint Perseids is visible only from the countryside."
In total, the Perseid meteor rate from dark-sky sites could top 100 per hour.
That's a lot of magic. Enjoy the show.