http://www.eurekalert.org/pub_releases/2014-12/nlmc-fsv121714.php

First successful vaccination against 'mad cow'-like wasting disease in deer

'Gut vaccine' strategy may work for similar brain infections in humans

NYU Langone Medical Center / New York University School of Medicine

Researchers at NYU Langone Medical Center and elsewhere say that a vaccination they have developed to fight a brain-based, wasting syndrome among deer and other animals may hold promise on two additional fronts: Protecting U.S. livestock from contracting the disease, and preventing similar brain infections in humans.

The study, to be published in Vaccine online Dec. 21, documents a scientific milestone: The first successful vaccination of deer against chronic wasting disease (CWD), a fatal brain disorder caused by unusual infectious proteins known as prions. Prions propagate by converting otherwise healthy proteins into a disease state.

Equally important, the researchers say, this study may hold promise against human diseases suspected to be caused by prion infections, such as Creutzfeldt-Jakob disease, kuru, familial insomnia, and variably protease-sensitive prionopathy. Some studies also have associated prion-like infections with Alzheimer's disease.

"Now that we have found that preventing prion infection is possible in animals, it's likely feasible in humans as well," says senior study investigator and neurologist Thomas Wisniewski, MD, a professor at NYU Langone.

CWD afflicts as much as 100 percent of North America's captive deer population, as well as large numbers of other cervids that populate the plains and forests of the Northern Hemisphere, including wild deer, elk, caribou and moose. There is growing concern among scientists that CWD could possibly spread to livestock in the same regions, especially cattle, a major life stream for the U.S. economy, in much the same manner that bovine spongiform encephalopathy, or Mad Cow Disease, another prion-based infection, spread through the United Kingdom almost two decades ago.

According to Dr. Wisniewski and his research team, if further vaccine experiments prove successful, a relatively small number of animals (as few as 10 percent) could be inoculated to induce herd immunity, in which disease transmission is essentially stopped in a much larger group.

For the study, five deer were given the vaccine; another six were given a placebo. All of the deer were exposed to prion-infected brain tissue; they also were housed together, engaging in group activities similar to those in the wild. Scientists say this kept them in constant exposure to the infectious prions. The animals receiving the vaccine were given eight boosters over 11 months until key immune antibodies were detectable in blood, saliva, and feces. The deer also were monitored daily for signs of illness, and investigators performed biopsies of the animals' tonsils and gut tissue every three months to search for signs of CWD infection.

Within two years, all of the deer given the placebo developed CWD. Four deer given the real vaccine took significantly longer to develop infection - and the fifth one continues to remain infection free.

Wisniewski and his team made the vaccine using Salmonella bacteria, which easily enters the gut, to mirror the most common mode of natural infection - ingestion of prion-contaminated food or feces. To prepare the vaccine, the team inserted a prion-like protein into the genome of an attenuated, or no longer dangerous, Salmonella bacterium. This engineered the Salmonella to induce an immune response in the gut, producing anti-prion antibodies.

"Although our anti-prion vaccine experiments have so far been successful on mice and deer, we predict that the method and concept could become a widespread technique for not only preventing, but potentially treating many prion diseases," says lead study investigator Fernando Goni, PhD, an associate professor at NYU Langone.

Funding for the study was provided by the National Institutes of Health grants NIH NS047433, ARRA NS047433-06S1 and the Seix Dow Foundation.

In addition to Wisniewski and Goni, other NYU investigators involved in the study were Kinlung Wong, BSc; Daniel Peyser, MSc; and Jinfeng Zu, PhD. Research support was also provided by Candace Mathiason, PhD; Jeanette Hayes-Klug; Amy Nalls; Kelly Anderson; and Edward Hoover, DVM, MS, of the College of Colorado State University in Fort Collins, where the deer were kept; Lucia Yim, PhD; Veronica Estevez, MSc; and Jose A. Chabalgoity, PhD, at the University of Uruguay in Montevideo, where the vaccine was developed; and David R. Brown, MD, at the University of Bath in the United Kingdom.

http://www.eurekalert.org/pub_releases/2014-12/uoc-ilu121914.php

IMF lending undermined healthcare provision in Ebola-stricken West Africa

Writing today in the journal Lancet Global Health, researchers from Cambridge University's Department of Sociology examine the links between the International Monetary Fund (IMF) and the Ebola outbreak in West Africa.

According to the authors, joined by colleagues from Oxford University and the London School of Hygiene and Tropical Medicine, IMF programs over the years have imposed heavy constraints on the development of effective health systems of Guinea, Liberia and Sierra Leone - the cradle of the Ebola outbreak that has killed more than 6,800 since March this year.

The researchers say that economic policy reforms advocated by the IMF have undermined the capacity of health systems in these three nations - systems already fragile from legacies of conflict and state failure - to cope with infectious disease outbreaks and other such emergencies.

"A major reason why the Ebola outbreak spread so rapidly was the weakness of healthcare systems in the region, and it would be unfortunate if underlying causes were overlooked," said lead author and Cambridge sociologist Alexander Kentikelenis.

"Policies advocated by the IMF have contributed to under-funded, insufficiently staffed, and poorly prepared health systems in the countries with Ebola outbreaks."

By reviewing the policies enforced by the IMF before the outbreak - extracting information from the IMF lending programs between 1990 and 2014 - the researchers were able to examine the effects on the three West African nations, and identified three key policy impacts that led to the weakening of the already fragile healthcare systems in these countries:

Firstly, the IMF required economic reforms that reduced government spending. "Such policies have been extremely strict, absorbing funds that could be directed to meeting pressing health challenges," write the researchers. Although the IMF responded to concerns raised about the impact of these policies by incorporating "poverty-reduction expenditures" that aimed to boost health budgets, the researchers found these conditions were often not met.

"In 2013, just before the Ebola outbreak, the three countries met the IMF's economic directives, yet all failed to raise their social spending despite pressing health needs," said Professor Lawrence King, co-author and Cambridge sociologist.

Secondly, the IMF often requires caps on the public-sector wage bill, directly impacting the capacity of these nations to hire and adequately pay key healthcare workers such as doctors and nurses. An independent evaluation of the IMF in 2007 stated that these limits are "often set without consideration of the impact on expenditures in priority areas".

"Wage limits set by the IMF have been linked to a 'brain drain' of health workers in countries that need them most. For example, the IMF imposed restrictions on wage spending in Sierra Leone over the 2000s. At the same time, the number of health personnel in the country plummeted," said King.

Thirdly, the IMF campaigns for decentralised healthcare systems. While the idea behind this is to make healthcare more responsive to local needs, the researchers say that in practice this makes it difficult to mobilise coordinated responses to outbreaks of deadly diseases such as Ebola.

However, in recent months, the IMF has announced $430m of funding to help combat Ebola in West Africa, leading IMF Director Christine Lagarde to say it is "good to increase the fiscal deficit when it's a matter of curing the people [...] The IMF doesn't say that very often."

"The IMF's recent change of heart about prioritising public health instead of fiscal discipline is welcome, but this is not the first time we have heard such rhetoric from the IMF leadership. It remains to be seen whether this time is different," said Kentikelenis.

The authors of the Lancet article point to that journal's own Commission on Investing in Health, which calls for increases in public health spending and attention to hiring and training health workers. "The experience of Ebola adds a degree of urgency to the implementation of its recommendations," they write.

http://www.eurekalert.org/pub_releases/2014-12/uoc-fps121814.php

Forager past shows our fragile bones result from inactivity since invention of farming

New research across thousands of years of human evolution shows that our skeletons have become much lighter and more fragile since the invention of agriculture - a result of our increasingly sedentary lifestyles as we shifted from foraging to farming.

The new study, published today in the journal PNAS, shows that, while human hunter-gatherers from around 7,000 years ago had bones comparable in strength to modern orangutans, farmers from the same area over 6,000 years later had significantly lighter and weaker bones that would have been more susceptible to breaking.

Bone mass was around 20% higher in the foragers - the equivalent to what an average person would lose after three months of weightlessness in space.

After ruling out diet differences and changes in body size as possible causes, researchers have concluded that reductions in physical activity are the root cause of degradation in human bone strength across millennia. It is a trend that is reaching dangerous levels, they say, as people do less with their bodies today than ever before.

Researchers believe the findings support the idea that exercise rather than diet is the key to preventing heightened fracture risk and conditions such as osteoporosis in later life: more exercise in early life results in a higher peak of bone strength around the age of 30, meaning the inevitable weakening of bones with age is less detrimental.

There is, in fact, no anatomical reason why a person born today could not achieve the bone strength of an orangutan or early human forager, say researchers; but

even the most physically active people alive are unlikely to be loading bones with enough frequent and intense stress to allow for the increased bone strength seen in the 'peak point' of traditional hunter-gatherers and non-human primate bones.

"Contemporary humans live in a cultural and technological milieu incompatible with our evolutionary adaptations. There's seven million years of hominid evolution geared towards action and physical activity for survival, but it's only in the last say 50 to 100 years that we've been so sedentary - dangerously so," said co-author Dr Colin Shaw from the University of Cambridge's Phenotypic Adaptability, Variation and Evolution (PAVE) Research Group.

"Sitting in a car or in front of a desk is not what we have evolved to do."

The researchers x-rayed samples of human femur bones from the archaeological record, along with femora from other primate species, focusing on the inside of the femoral head: the ball at the top of the femur which fits into the pelvis to form the hip joint, one of the most load-bearing bone connections in the body.

Two types of tissue form bone: the cortical or 'hard' bone shell coating the outside, and the trabecular or 'spongy' bone: the honeycomb-like mesh encased within cortical shell that allows flexibility but is also vulnerable to fracture.

The researchers analysed the trabecular bone from the femoral head of four distinct archaeological human populations representing mobile hunter-gatherers and sedentary agriculturalists, all found in the same area of the US state of Illinois (and likely to be genetically similar as a consequence).

The trabecular structure is very similar in all populations, with one notable exception: within the mesh, hunter-gatherers have a much higher amount of actual bone relative to air.

"Trabecular bone has much greater plasticity than other bone, changing shape and direction depending on the loads imposed on it; it can change structure from being pin or rod-like to much thicker, almost plate-like. In the hunter-gatherer bones, everything was thickened," said Shaw.

This thickening is the result of constant loading on the bone from physical activity as hunter-gatherers roamed the landscape seeking sustenance. This fierce exertion would result in minor damage that caused the bone mesh to grow back ever stronger and thicker throughout life - building to a 'peak point' of bone strength which counter-balanced the deterioration of bones with age.

Shaw believes there are valuable lessons to be learnt from the skeletons of our prehistoric predecessors. "You can absolutely morph even your bones so that they deal with stress and strain more effectively. Hip fractures, for example, don't have to happen simply because you get older if you build your bone strength up earlier in life, so that as you age it never drops below that level where fractures can easily occur."

Other theories for humans evolving a lighter, more fragile skeleton include changes in diet or selection for a more efficient, lighter skeleton, which was never reversed.

While the initial switch to farming did cause a dip in human health due to monoculture diets that lacked variety, the populations tested were unaffected by this window in history. "Of course we need a level of calcium to maintain bone heath, but beyond that level excess calcium isn't necessary," said Shaw.

The research also counters the theory that, at some point in human evolution, our bones just became lighter - perhaps because there wasn't enough food to support a denser skeleton. "If that was true, human skeletons would be entirely distinct from other living primates. We've shown that hunter-gatherers fall right in line with primates of a similar body size. Modern human skeletons are not systemically fragile; we are not constrained by our anatomy."

"The fact is, we're human, we can be as strong as an orangutan - we're just not, because we are not challenging our bones with enough loading, predisposing us to have weaker bones so that, as we age, situations arise where bones are breaking when, previously, they would not have" Shaw said.

While the 7,000-year-old foragers had vastly stronger bones than the 700-year-old farmers, Shaw says that neither competes with even earlier hominids from around 150,000 years ago. "Something is going on in the distant past to create bone strength that outguns anything in the last 10,000 years."

The next step for Shaw's research team will be to look at how different types of loading and mobility shape bodies and bones by cross-referencing archaeological records with testing on modern ultra-marathon runners, who cover punishing distances over a range of terrains - from the Himalayas to the Namibian desert. He hopes this future work will provide insight into the kind of mobility that gave our ancient ancestors such powerful physical strength.

http://www.eurekalert.org/pub_releases/2014-12/sri-sun121914.php

Scientists uncover new, fundamental mechanism for how resveratrol provides health benefits

The ingredient found in red wine activates ancient stress response

LA JOLLA, CA and JUPITER, FL - Scientists at The Scripps Research Institute (TSRI) have found that resveratrol, the red-wine ingredient once touted as an elixir of youth, powerfully activates an evolutionarily ancient stress response in human cells. The finding should dispel much of the mystery and controversy about how resveratrol really works.

"This stress response represents a layer of biology that has been largely overlooked, and resveratrol turns out to activate it at much lower concentrations

than those used in prior studies," said senior investigator Paul Schimmel, professor and member of the Skaggs Institute for Chemical Biology at TSRI.

"With these findings we have a new, fundamental mechanism for the known beneficial effects of resveratrol," said lead author Mathew Sajish, a senior research associate in the Schimmel laboratory.

The discovery is reported in the advance online edition of Nature on December 22.

Resveratrol is a compound produced in grapes, cacao beans, Japanese knotweed and some other plants in response to stresses including infection, drought and ultraviolet radiation. It has attracted widespread scientific and popular interest over the past decade, as researchers have reported that it extended lifespan and prevented diabetes in obese mice and vastly increased the stamina of ordinary mice running on wheels.

More recently, though, scientists in this field have disagreed about the signaling pathways resveratrol activates to promote health, calling into question some of resveratrol's supposed health benefits - particularly given the unrealistically high doses used in some experiments.

Outsiders to the Controversy

Schimmel and Sajish came to this controversy as outsiders. Schimmel's laboratory is known for its work not on resveratrol but on an ancient family of enzymes, the tRNA synthetases. The primary and essential function of these enzymes is to help translate genetic material into the amino-acid building blocks that make proteins. But as Schimmel and others have shown since the late 1990s, tRNA synthetases have acquired an extensive set of added functions in mammals.

Earlier Xiang-Lei Yang, a TSRI professor in the Departments of Chemical Physiology and Cell and Molecular Biology and former member of Schimmel's laboratory, began to find hints that a tRNA synthetase called TyrRS, which links the amino acid tyrosine to the genetic material that codes for it, can move to the cell nucleus under stressful conditions - apparently taking on a protective, stress-response role.

Sajish noted that resveratrol appeared to have broadly similar stress-response properties and also resembled TyrRS's normal binding partner tyrosine. "I began to see TyrRS as a potential target of resveratrol," he said.

For the new study, Sajish and Schimmel put TyrRS and resveratrol together and showed with tests including X-ray crystallography that resveratrol does indeed mimic tyrosine, well enough to fit tightly into TyrRS's tyrosine binding pocket. That binding to resveratrol, the team found, takes TyrRS away from its protein translation role and steers it to a function in the cell nucleus.

Tracking the resveratrol-bound TyrRS in the nucleus, the researchers determined that it grabs and activates the protein, PARP-1, a major stress response and DNA-repair factor thought to have a significance influence on lifespan. The scientists confirmed the interaction in mice injected with resveratrol. TyrRS's activation of PARP-1 led, in turn, to the activation of a host of protective genes including the tumor-suppressor gene p53 and the longevity genes FOXO3A and SIRT6.

Compatible with Red Wine

The first studies of resveratrol in the early 2000s had suggested that it exerts some of its positive effects on health by activating SIRT1, also thought to be a longevity gene. But SIRT1's role in mediating resveratrol's reported health-boosting effects has been questioned lately in terms of its particular role.

The team's experiments showed, however, that the TyrRS-PARP-1 pathway can be measurably activated by much lower doses of resveratrol - as much as 1,000 times lower - than were used in some of the more celebrated prior studies, including those focused on SIRT1. "Based on these results, it is conceivable that moderate consumption of a couple glasses of red wine (rich in resveratrol) would give a person enough resveratrol to evoke a protective effect via this pathway," Sajish said. He also suspects that effects of resveratrol that only appear at unrealistically high doses may have confounded some prior findings.

Why would resveratrol, a protein produced in plants, be so potent and specific in activating a major stress response pathway in human cells? Probably because it does much the same in plant cells, and probably again via TyrRS - a protein so fundamental to life, due to its linkage to an amino acid, that it hasn't changed much in the hundreds of millions of years since plants and animals went their separate evolutionary ways.

"We believe that TyrRS has evolved to act as a top-level switch or activator of a fundamental cell-protecting mechanism that works in virtually all forms of life," said Sajish.

Whatever activity resveratrol naturally has in mammals may be an example of hormesis: the mild, health-promoting activation of a natural stress response. "If resveratrol brought significant benefits to mammals, they might have evolved a symbiotic relationship with resveratrol-producing plants," Sajish said.

"We think this is just the tip of the iceberg," said Schimmel. "We think there are a lot more amino-acid mimics out there that can have beneficial effects like this in people. And we're working on that now."

Schimmel and his laboratory also are searching for molecules that can activate the TyrRS stress response pathway even more potently than resveratrol does.

The National Cancer Institute (CA92577), the National Foundation for Cancer Research and aTyr Pharma, Inc. provided funding for the study, "A human tRNA synthetase is a potent PARP1-activating effector target for resveratrol." For more information, see http://www.nature.com

http://www.eurekalert.org/pub_releases/2014-12/uol-ufd122214.php

University of Louisville faculty discover mutation role involved in 75 percent of glioblastomas, melanomas

Researchers identify for first time mutations that destabilize DNA structure, turning gene off

LOUISVILLE, Ky. - Researchers at the University of Louisville's James Graham Brown Cancer Center have identified for the first time mutations that destabilize a DNA structure that turns a gene off. These mutations occur at four specific sites in what is known as the "hTERT promoter" in more than 75 percent of glioblastomas and melanomas.

The research is published this month in the online journal PLOS ONE and is authored by Brad Chaires, Ph.D., John Trent, Ph.D., Robert Gray, William Dean, Ph.D., Robert Buscaglia, Shelia Thomas and Donald Miller, M.D., Ph.D.

Telomerase is an enzyme largely responsible for the promotion of cell division. Within DNA, telomerase activation is a critical step for human carcinogenesis through the maintenance of telomeres. However, the activation mechanism during carcinogenesis - why cancer gets turned "on" - is not yet wholly understood. What is known is that transcriptional regulation of the human telomerase reverse transcriptase (hTERT) gene is the major mechanism for cancer-specific activation of telomerase.

Miller and his colleagues have been interested in turning genes off therapeutically for some time. "We know that human telomerase is over-expressed in most human cancers, but we've never known why," he said.

In 2013, two studies published in Science and another in Proceedings of the National Academy of Sciences gave the researchers a direction to explore. "These papers said that in most melanomas, mutations existed in the promoter of this telomerase gene. This was the first time that anyone reported common mutations in these promoters," said Miller, who is director of the James Graham Brown Cancer Center and a specialist in the treatment of melanoma.

The UofL team has now shown that the mutations all occur in a region of the hTERT promoter that previously has been shown to form quadruplex DNA. Using a combination of biophysics and molecular modeling, a new form of a quadruplex transcription regulation element is reported. The formation of these quadruplexes in telomeres has been shown to decrease the activity of telomerase.

"We speculated that the occurrence of these mutations could destabilize or alter the recognition of quadruplexes formed by this sequence," Miller said. "We found that the mutations inactivate the gene's 'off' switch so it becomes locked on, destabilizing the quadruplex and allowing it to be over-expressed. "This over-expression then drives the cells to continue to divide, which is the cause of the cancer."

The researchers are next examining how to unlock the switch from on to off, Miller said. "What we have described in this PLOS ONE article is the on-off switch and provided an entirely new model for that structure. Our next step is to look at how to turn it off that will help lead us to new therapeutics to prevent the occurrence of cancer."

The paper was posted online Dec. 19 in PLOS ONE.

http://www.eurekalert.org/pub_releases/2014-12/ru-aed122214.php

An existing drug, riluzole, may prevent foggy 'old age' brain

Treatment promoted neuroplasticity-promoting changes in aging rats

Forgetfulness, it turns out, is all in the head.

Scientists have shown fading memory and clouding judgment, the type that comes with advancing age, show up as lost and altered connections between neurons in the brain. But new experiments suggest an existing drug, known as riluzole and already on the market as a treatment for ALS, may help prevent these changes.

Researchers at The Rockefeller University and The Icahn School of Medicine at Mount Sinai found they could stop normal, age-related memory loss in rats by treating them with riluzole. This treatment, they found, prompted changes known to improve connections, and as a result, communication, between certain neurons within the brain's hippocampus.

"By examining the neurological changes that occurred after riluzole treatment, we discovered one way in which the brain's ability to reorganize itself - its neuroplasticity - can be marshaled to protect it against some of the deterioration that can accompany old age, at least in rodents," says co-senior study author Alfred E. Mirsky Professor Bruce McEwen, head of the Harold and Margaret Milliken Hatch Laboratory of Neuroendocrinology. The research was published online December 15 in the Proceedings of the National Academy of Sciences.

Neurons connect to one another to form circuits connecting certain parts of the brain, and they communicate using a chemical signal known as glutamate. But too much glutamate can cause damage; excess can spill out and excite connecting neurons in the wrong spot. In the case of age-related cognitive decline, this process damages neurons at the points where they connect - their synapses. In neurodegenerative disorders, such as Alzheimer's disease, this contributes to the death of neurons.

Used to slow the progress of another neurodegenerative condition, ALS (also known as Lou Gehrig's disease), riluzole was an obvious choice as a potential treatment, because it works by helping to control glutamate release and uptake, preventing harmful spillover. The researchers began giving riluzole to rats once

they reached 10 months old, the rat equivalent of middle age, when their cognitive decline typically begins.

After 17 weeks of treatment, the researchers tested the rats' spatial memory - the type of memory most readily studied in animals - and found they performed better than their untreated peers, and almost as well as young rats. For instance, when placed in a maze they had already explored, the treated rats recognized an unfamiliar arm as such and spent more time investigating it.

When the researchers looked inside the brains of riluzole-treated rats, they found telling changes to the vulnerable glutamate sensing circuitry within the hippocampus, a brain region implicated in memory and emotion.

"We have found that in many cases, aging involves synaptic changes that decrease synaptic strength, the plasticity of synapses, or both," said John Morrison, professor of neuroscience and the Friedman Brain Institute and dean of basic sciences and the Graduate School of Biomedical Sciences at Mount Sinai. "The fact that riluzole increased the clustering of only the thin, most plastic spines, suggests that its enhancement of memory results from both an increase in synaptic strength and synaptic plasticity, which might explain its therapeutic effectiveness."

In this case, the clusters involved thin spines, a rapidly adaptable type of spine. The riluzole-treated animals had more clustering than the young animals and their untreated peers, who had the least. This discovery led the researchers to speculate that, in general, the aged brain may compensate by increasing clustering. Riluzole appears to enhance this mechanism.

"In our study, this phenomenon of clustering proved to be the core underlying mechanism that prevented age-related cognitive decline. By compensating the deleterious changes in glutamate levels with aging and Alzheimer's disease and promoting important neuroplastic changes in the brain, such as clustering of spines, riluzole may prevent cognitive decline," says first author Ana Pereira, an instructor in clinical investigation in McEwen's laboratory.

Taking advantage of the overlap of neural circuits vulnerable to age-related cognitive decline and Alzheimer's disease, Pereira is currently conducting a clinical trial to test the effectiveness of riluzole for patients with mild Alzheimer's.

http://bit.ly/1A4zacL

Truffles Have a THC-like Substance in Them

This produces a euphoric smell that likely evolved as a way to trick animals into eating them

By Rachel Nuwer

Truffles don't particularly taste like much, but their smell is euphoric. It's their relative rarity combined with this blissful aroma that makes those little fungal nubs one of the most expensive delicacies in the world, often selling for hundreds of dollars per pound.

Now, Italian scientists have begun to crack the mystery of that unique truffle scent. As BBC Earth reports, they found that black truffles produce a natural chemical similar to the tetrahydrocannabinol, the psychoactive compound found in marijuana. Described by BBC Earth as a "bliss molecule," anandamide, the black truffle equivalent, causes the brain to release mood-enhancing chemicals. This effect isn't just confined to humans, either. According to the researchers, various mammals are likely susceptible to this chemical trick, too. This probably explains why truffle-sniffing dogs and hogs seem to fall under a frenzied spell when they begin to home in on one of those delectable subterranean treasures, BBC Earth reports.

So why do the truffles make anandamide? The truffles, the researchers found, do not have the requisite receptors that anandamide binds to, meaning the fungus itself has no use for the chemical. Given that, the researchers think that black truffles likely evolved this chemical profile precisely to encourage animals to devour them, BBC Earth writes. As the anandamide-intoxicated animal eats the truffle, the fungus' tiny spores are probably spread over a wider area than they would be if they just sat in the earth on their own.

Determining whether white, burgundy, and Bianchetto truffles also evolved the same propagation-friendly chemical profile will require further tests.

http://www.eurekalert.org/pub_releases/2014-12/pcrf-avs122314.php

Armed virus shows promise as treatment for pancreatic cancer

Study of improved effectiveness of Vaccinia oncolytic virus in treating pancreatic cancer by arming it with a gene modulating the body's immune system

The study, funded by the UK charity Pancreatic Cancer Research Fund, investigated whether the effectiveness of the Vaccinia oncolytic virus - a virus modified to selectively infect and kill cancer cells - as a treatment for pancreatic cancer, would be improved by arming it with a gene which modulates the body's immune system.

Despite laboratory studies which show that they can both kill cancer cells and provide immunity against cancer regrowth, oncolytic viruses have not performed well in clinical trials, as the immune system naturally attacks the virus before it can be effective.

The QMUL team, at Barts Cancer Institute, armed the Vaccinia virus with a copy of the interleukin-10 (IL-10) gene, which would express proteins in the cancer cell once infected by the Vaccinia. These proteins are important in cell signalling - but

are also known to dampen the immune response - and the researchers hoped that this would allow the virus to take hold and persist for longer.

"Many viruses use IL-10 to hide from the host's immune system, so we thought we'd use this natural strategy to investigate whether it would improve Vaccinia's effectiveness," said Dr Yaohe Wang, who led the research.

The research team first confirmed in the cell lines that arming Vaccinia with IL-10 would not compromise Vaccinia's anti-cancer effects.

They then conducted tests comparing the effectiveness of Vaccinia and Vaccinia armed with IL-10 on mice with pancreatic cancer and a group of transgenic mice specially bred to develop a more human form of the disease.

After six weeks, 87.5 per cent of all the mice treated with the combination approach were completely clear of tumours compared with 42.8 per cent of those treated with Vaccinia alone.

In the transgenic mice group, the average survival rate almost doubled from 69.7 days for 138.5 days.

Four weeks after being completely clear of primary tumours, pancreatic cancer cells were reintroduced into the mice.

No further doses of armed or unarmed virus were given. Whilst the cancer cells grew again in both groups, after 32 days all animals bar one were once again completely clear of cancer.

Interestingly, the regrowth was much slower in the mice originally treated with IL-10 armed Vaccinia and the mice in this group were free of cancer in only 18 days.

"This corresponds with recently published research which has suggested that IL-10 has its own anticancer properties when delivered directly into tumour tissue," says Dr Wang.

He continued: "These are exciting results, but we still have several questions. Our results show that in mice IL-10 suppresses antiviral immunity but boosts anti-tumour immunity - but exactly how IL-10 makes this happen remains unclear. This is something we're already investigating as understanding this mechanism will provide a foundation for designing clinical trials to treat pancreatic cancer with this IL-10 armed virus."

Maggie Blanks, CEO of the Pancreatic Cancer Research Fund said: "Much more research is needed, but these early results show there's some potential here. Pancreatic cancer desperately needs a radical new approach to see improvement in survival, so a new treatment that also offers protection against disease recurrence would be an extremely important development."

The study is published in the journal Clinical Cancer Research.

http://bit.ly/1JOu9c5

On the Front Lines of Ebola's Most Pressing Mystery

Gathering information about Ebola survivors in hopes of finding why some beat Ebola

By Erika Check Hayden

KENEMA, Sierra Leone - Alex Moigboi was panicking. He was preparing to enter the Ebola ward wearing just a pair of gloves and a plastic gown over his scrubs.

It was totally inadequate - like a firefighter entering a burning building wearing a pair of Ray-Bans - and Alex knew it. But he couldn't find the rest of the protective gear he needed: goggles, a Tyvek waterproof suit.

Alex was angry, crying, desperate. But his patients, piled three to a bed in the ward, needed him. He steeled himself to go inside. Alex later became one of dozens of health workers who died from Ebola here at Kenema Government Hospital this summer.

But others who entered the wards lived.

Mohammed Sankoh Yillah, an outreach worker, spent days in the Ebola ward caring for his sister, nurse Mbalu Fonnie. After Fonnie died in July, Yillah tested positive for the virus. He was transported to another hospital for treatment, but asked to come back to Kenema to die.

His wish was granted; he came back.

But Yillah survived.

Today Yillah sits with four colleagues in an office, discussing a new research project. The study is collecting information about survivors like him.

The hope is that the study might help explain why he and others beat Ebola, while their friends and colleagues - Alex, Mbalu - did not.

Epidemiologist Lina Moses runs the meeting.

Her colleagues back at Tulane University, she says, hope to analyze blood samples from survivors; she collected 29 such samples here in November.

"What they want to know in the laboratory," she says, "is what kind of antibodies Mohammed Yillah has that helped him to survive Ebola."

A lot of people want to know the answer to that question; scientists only have hints about why some live while others don't.

They know, for instance, that the body's first-responder immune cells seem to malfunction in those who don't make it, triggering a massive internal overreaction to the virus.

These patients suffer from fever, dehydration, organ failure and, finally, death.

But in those who survive, the first-responder cells manage to enlist the "adaptive" immune response that makes cells and proteins to attack specific viruses.

Those who survive make antibodies to the virus - proteins tailor-made to recognize and destroy Ebola itself.

Survivors like Yillah can make Ebola antibodies for years after they recover, and are therefore thought to be immune to the virus.

That's why they're being deployed all around the Ebola response; Yillah himself cares for children who are suspected of having the disease.

His antibodies also might, one day, help future patients. The Tulane study of survivor samples will search for antibodies that could serve as templates for better Ebola therapies and vaccines.

The drugs would work like ZMapp, the experimental antibody cocktail used to treat a handful of patients in this epidemic. But ZMapp is based on antibodies made in mice; this drug would be modeled on antibodies made by human Ebola survivors.

Moses hopes to do much more with the study, if she can get the funding.

There are 1,257 Ebola survivors in Sierra Leone - more in one country than have survived all previous Ebola outbreaks combined.

Moses hopes to work with the survivors for years, to understand how the disease has affected them; they face discrimination, poverty and ongoing health problems.

Moses is friends with many of the survivors in Kenema; though she is based at Tulane, she manages a project here on Lassa fever - like Ebola, a hemorrhagic illness.

Moses spent most of this year in Kenema, during the worst of the outbreak. Her colleagues - doctors, nurses, lab technicians - had been trained to fight Lassa.

She watched them enter the Ebola wards day after day; first as caregivers, then as patients.

On the day in June that Alex prepared to go into the ward in his paltry gear, Moses saw him. She ran to her lab to collect the rest of the protective equipment he needed.

As a result, Alex didn't become infected that day.

That happened later. Every day the nurses went into the wards in those early months - May, June, July - they risked their lives.

Many stopped showing up. Only the most dedicated, like Alex, continued to try.

When Moses found out that Alex was infected, she hoped for the best: "You think that the really good people are going to pull through," she says.

Why that didn't happen for Alex is a question that, Moses hopes, her research will help to answer one day.

Erika Check Hayden is a staff reporter for Nature. This piece was supported in part by The Pulitzer Center on Crisis Reporting.

http://bit.ly/1B5DZAp

New seismic survey technique could save dolphins' hearing

New method collects data that is normally discarded but still holds important information

Conventional seismic imaging transmits sound energy into the ground and builds a picture of the underlying geology by analysing how the energy waves are reflected back to the receiver.

These techniques create a series of loud bangs that can disturb marine wildlife, affecting their behaviour and migration habits. The University of Bath is already active in monitoring and reducing these impacts.

In contrast, a new technology called Acoustic Zoom, developed by University of Bath alumnus Professor Jaques Guigné in collaboration with his former PhD supervisor Professor Nick Pace, suppresses the reflected energy signal and instead records how the sound energy is scattered; data that is normally discarded but still holds important information.

This scattered sound data is used to create a highly detailed map of the underground geology, enabling subtle details such as fissures and fractures in the strata to be seen that cannot be detected using traditional seismic surveying.

The Acoustic Zoom technology goes further by reducing the original energy, using a marine trombone that can be adjusted to release smaller levels of energy at much higher frequencies, which reduces the effects on underwater animals.

Professor Guigné explained: "We're really excited about this technology because it allows us to take virtual core samples, giving us a much more detailed understanding of subtle geological features, without any drilling.

"It works by analysing the scattered sound energy rather than the reflected energy that is normally recorded. The scattered signal is a lot weaker so it's been quite tricky to do successfully. "It's a bit like driving at night trying to focus on the face of the driver of an oncoming car through the glare of their headlights."

Acoustic Zoom is also gentler on the environment because it releases smaller amounts of energy over a longer time and at higher frequencies, so although marine life can still hear the sound waves, they are much less intrusive.

""We hope this new technology will help avoid unnecessary exploratory drilling by the oil industry and also reduce the impact of underwater surveys on the environment.""

Explore further: The sound of an atom has been captured

More information: Guigné et al (2014) Acoustic zoom high-resolution seismic beamforming for imaging specular and non-specular energy of deep oil and gas bearing geological formations" is published in Journal of Natural Gas Science and Engineering. www.sciencedirect.com/science/... ii/S1875510014002728

http://www.eurekalert.org/pub_releases/2014-12/hsop-mmb121814.php

Molecular mechanism behind health benefits of dietary restriction identified

A new study led by Harvard School of Public Health (HSPH) researchers identifies a key molecular mechanism behind the health benefits of dietary restriction, or reduced food intake without malnutrition.

Boston, MA - Also known as calorie restriction, dietary restriction is best known for its ability to slow aging in laboratory animals. The findings here show that restricting two amino acids, methionine and cysteine, results in increased hydrogen sulfide (H_2S) production and protection against ischemia reperfusion injury, damage to tissue that occurs following the interruption of blood flow as during organ transplantation and stroke. Increased H_2S production upon dietary restriction was also associated with lifespan extension in worms, flies, and yeast. Although H_2S gas is extremely toxic in high amounts, low levels present in naturally occurring sulfur springs have long been associated with health benefits. Mammalian cells also produce low levels of H_2S, but this is the first time that this molecule has been linked directly to the health benefits of dietary restriction.

"This finding suggests that H_2S is one of the key molecules responsible for the benefits of dietary restriction in mammals and lower organisms as well," said senior author James Mitchell, associate professor of genetics and complex diseases. "While more experiments are required to understand how H_2S exerts its beneficial effects, it does give us a new perspective on which molecular players to target therapeutically in our efforts to combat human disease and aging."

The study appears online December 23, 2014 in Cell.

Dietary restriction is a type of intervention that can include reduced overall food intake, decreased consumption of particular macronutrients such as protein, or intermittent bouts of fasting. It is known to have beneficial health effects, including protection from tissue injury and improved metabolism. It has also been shown to extend the lifespan of multiple model organisms, ranging from yeast to primates. The molecular explanations for these effects are not completely understood, but were thought to require protective antioxidant responses activated by the mild oxidative stress caused by dietary restriction itself.

First author Christopher Hine, research fellow in the Department of Genetics and Complex Diseases, and colleagues demonstrated that one week of dietary restriction increased antioxidant responses and protected mice from liver ischemia reperfusion injury, but surprisingly, this protective effect was intact even in animals that could not mount such an antioxidant response. Instead, the researchers found that the protection required increased production of H_2S, which occurred upon reduction of dietary intake of the two sulfur-containing amino acids, methionine and cysteine. When the diet was supplemented with these two amino acids, increased H_2S production and dietary restriction benefits were both lost. The investigators also found that genes involved in H_2S production were also required for longevity benefits of dietary restriction in other organisms, including yeast, worms, and flies.

"These findings give us a better understanding of how dietary interventions extend lifespan and protect against injury. More immediately, they could have important implications for what to eat and not to eat before a planned acute stress like surgery, when the risk of ischemic injury can be relatively high," said Hine.

Other Harvard School of Public Health authors include Eylul Harputlugil, Lear Brace, Humberto Trevino-Villarreal, Pedro Mejia, Yue Zhang, and William Mair.

The study was supported by grants from NIH (R01DK090629, R01AG036712) and the Glenn Foundation to James Mitchell. Christopher Hine was supported by T32CA0093823. Frank Madeo was supported by the Austrian Science Fund FWF (LIPOTOX, I1000, P23490-B12, and P24381-B20). William Mair was supported by R01AG044346. Vadim Gladyshev was supported by R01AG021518. C. Keith Ozaki was supported by American Heart Association 12GRNT9510001 and 12GRNT1207025. Rui Wang was supported by an operating grant from the Canadian Institutes of Health Research.

"Endogenous Hydrogen Sulfide Production is Essential for Dietary Restriction Benefits," Christopher Hine, Eylul Harputlugil, Yue Zhang, Christoph Ruckenstuhl, Byung Cheon Lee, Lear Brace, Alban Longchamp, Jose Trevino-Villarreal, Pedro Mejia, C. Keith Ozaki, Rui Wang, Vadim Gladyshev, Frank Madeo, William Mair, and James Mitchell, Cell, online December 23, 2014, doi:10.1016/j.cell.2014.11.048

After the embargo lifts, the paper will be available at http://www.cell.com/cell/abstract/S0092-8674(14)01525-6

http://www.eurekalert.org/pub_releases/2014-12/uob-asl121914.php

Ants show left bias when exploring new spaces

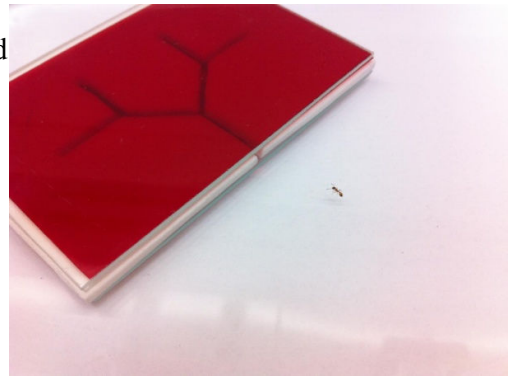
PhD student Edmund Hunt and colleagues studied how Temnothorax albipennis ants explore nest cavities and negotiate through branching mazes.

They found that ants were significantly more likely to turn left than right when exploring new nests. Such left bias was also present when the ants were put in branching mazes, though this bias was initially obscured by wall-following behaviour.

So why do the majority of rock ants turn left when entering unknown spaces? Edmund Hunt said: "The ants may be using their left eye to detect predators and their right to navigate. Also, their world is maze-like and consistently turning one way is a very good strategy to search and exit mazes.

"Furthermore, as their nest-mates are left-leaning too, there should also be safety in numbers. Consistent turning may also help the ants to monitor nest mates during house hunting. So perhaps leaning left is more shrewd than sinister."

Around ten percent of people are left-handed and brain lateralization is widespread in other vertebrates. There's also increasing evidence for sensory and motor asymmetries in the behaviour of invertebrates, but evidence for lateral biases in ants is relatively limited. Behavioural lateralization in invertebrates is an important field of study because it may provide insights into the early origins of lateralization seen in a diversity of organisms, the researchers said.



In a study published in Biology Letters, researchers from the University of Bristol, UK found that rock ants instinctively go left when entering unknown spaces. **Edmund Hunt, University of Bristol, UK**

'Ants show a leftward turning bias when exploring unknown nest sites' by Hunt ER, O'Shea-Wheller T, Albery GF, Bridger TH, Gumn M, Franks NR. in Biology Letters

http://www.eurekalert.org/pub_releases/2014-12/uoc--rsl122314.php

Researchers shed light on how 'microbial dark matter' might cause disease

Breakthrough by scientists from UCLA, J. Craig Venter Institute and U of Washington may be roadmap for study of other elusive bacteria

One of the great recent discoveries in modern biology was that the human body contains 10 times more bacterial cells than human cells. But much of that bacteria is still a puzzle to scientists.

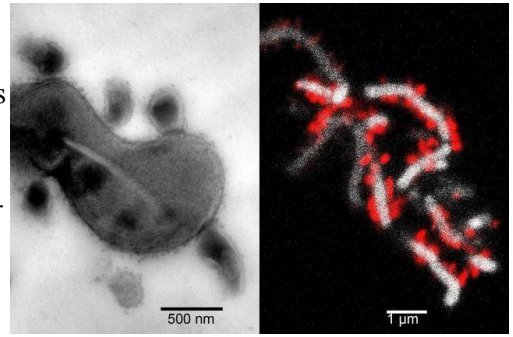
It is estimated by scientists that roughly half of bacteria living in human bodies is difficult to replicate for scientific research -- which is why biologists call it "microbial dark matter." Scientists, however, have long been determined to learn more about these uncultivable bacteria, because they may contribute to the development of certain debilitating and chronic diseases.

For decades, one bacteria group that has posed a particular challenge for researchers is the Candidate Phylum TM7, which has been thought to cause inflammatory mucosal diseases because it is so prevalent in people with periodontitis, an infection of the gums.

Now, a landmark discovery by scientists at the UCLA School of Dentistry, the J. Craig Venter Institute and the University of Washington School of Dentistry has revealed insights into TM7's resistance to scientific study and to its role in the progression of periodontitis and other diseases. Their findings shed new light on the biological, ecological and medical importance of TM7, and could lead to better understanding of other elusive bacteria.

The team's findings are published online in the December issue of the Proceedings of the National Academy of Sciences.

"I consider this the most exciting discovery in my 30-year career," said Dr. Wenyuan Shi, a UCLA professor of oral biology. "This study provides the roadmap for us to make every uncultivable bacterium cultivable."



The left image shows the tight physical association between TM7x cells and XH001. The right image shows TM7x cells (red) attach to the surface of XH001 (white). **Batbileg Bor/UCLA and Ryan Hunter/U of Minnesota**

The researchers cultivated a specific type of TM7 called TM7x, a version of TM7 found in people's mouths, and found the first known proof of a signaling interaction between the bacterium and an infectious agent called Actinomyces odontolyticus, or XH001, which causes mucosal inflammation.

"Once the team grew and sequenced TM7x, we could finally piece together how it makes a living in the human body," said Dr. Jeff McLean, acting associate professor at the University of Washington School of Dentistry. "This may be the first example of a parasitic long-term attachment between two different bacteria -- where one species lives on the surface of another species gaining essential nutrients and then decides to thank its host by attacking it."

To prove that TM7x needs XH001 to grow and survive, the team attempted to mix isolated TM7x cells with other strains of bacteria. Only XH001 was able to establish a physical association with TM7x, which led researchers to believe that TM7x and XH001 might have evolved together during their establishment in the mouth.

What makes TM7x even more intriguing are its potential roles in chronic inflammation of the digestive tract, vaginal diseases and periodontitis. The co-cultures collected in this study allowed researchers to examine, for the first time ever, the degree to which TM7x helps cause these conditions.

"Uncultivable bacteria presents a fascinating 'final frontier' for dental microbiologists and are a high priority for the NIDCR research portfolio," said Dr. R. Dwayne Lunsford, director of the National Institute of Dental and Craniofacial Research's microbiology program. "This study provides a near-perfect case of how co-cultivation strategies and a thorough appreciation for interspecies

signaling can facilitate the recovery of these elusive organisms. Although culture-independent studies can give us a snapshot of microbial diversity at a particular site, in order to truly understand physiology and virulence of an isolate, we must ultimately be able to grow and manipulate these bacteria in the lab."

It was previously known that XH001 induces inflammation. But by infecting bone marrow cells with XH001 alone and then with the TM7x/XH001 co-culture, the researchers also found that inflammation was greatly reduced when TM7x was physically attached to XH001. This is the only known study that has provided evidence of this relationship between TM7 and XH001.

The researchers plan to further study the unique relationship between TM7X and XH001 and how they jointly cause mucosal disease. Their findings could have implications for potential treatment and therapeutics.

Other collaborators on the study were Drs. Xuesong He, Renate Lux and Anna Edlund of the UCLA School of Dentistry; Shibu Yooseph, Adam Hall and Karen Nelson of the Venter Institute; Su-Yang Liud and Genhong Cheng of the UCLA department of microbiology, immunology and molecular genetics; Pieter Dorresteine of UC, San Diego; Eduardo Esquenazi of Sirenas Marine Discovery; and Ryan Hunterg of the University of Minnesota. Dr. Shi is part-time chief science officer at C3 Jian, Inc., which has licensed technologies from the University of California Regents that could be indirectly related to this research project.

The work was supported in part by the NIH's National Institute of Dental and Craniofacial Research (grants 1R01DE023810-01, 1R01DE020102 and 1R01DE021108, and T90 training award DE022734) and the National Institute of General Medical Sciences (grant 1R01GM095373).

http://www.eurekalert.org/pub_releases/2014-12/uotm-trt122314.php

To remove the gallbladder or not -- that is the question

Gallbladder removal is one of the most common operations performed in older adults.

Yet, research from the University of Texas Medical Branch at Galveston shows many patients who would benefit most from the surgery don't get it.

A previous study by the UTMB researchers showed that a combination of factors -- age, sex, race, other associated illnesses and severity of gallbladder symptoms, for example -- put a patient in the most danger for acute gallbladder attack. This study resulted in the creation of a predictive model for determining patients at most risk -- and thus most likely to benefit from having their gallbladders removed. Then the researchers put their predictive model to the test.

Their new study looks at 11 years of billing records of more than 160,000 Texas Medicare patients, 66 and older, who had an initial episode of gallstone trouble. The researchers used their predictive model to determine which of these patients was most likely headed for a dangerous gallbladder attack over the course of two years. The patients in the highest risk category should be receiving gallbladder removal surgery most often. But the UTMB study, available online in the January edition of the Journal of the American College of Surgeons, showed the reverse to be true. Removal of the gallbladder did not seem to depend on risk and in the healthiest patients, those in the most danger had their gallbladders removed least often.

Dr. Taylor Riall, professor of surgery at UTMB and lead author of the study, said that even though gallbladder removal is recommended for patients with gallstone problems, "Less than a quarter of patients in this study had their gallbladders removed. We sought to determine whether the decision to have the gallbladder removed was actually based on their risk of having gallstone-related complications in the next two years."

Using their model, the researchers identified which patients were in low, moderate or high-risk categories for an acute gallbladder episode that required hospitalization. This new study validates the accuracy of their predictive model. Among those who did not have their gallbladder removed, less than 20 percent in the low-risk group ended up being hospitalized for gallbladder-related issues -- for the high-risk group, 65 percent were hospitalized within two years of first symptoms.

In looking at patients who had the surgery, the study showed risk was not related to removal of the gallbladder. Only 22 percent of people in the low-risk group, 21 percent in the moderate-risk group, and 23 percent in the high-risk group had their gallbladder removed. In the healthiest older patients, gallbladder removal was performed in 34 percent of low-risk patients but in only 27 percent of the highest-risk patients. Also, less than 10 percent of patients who did not have their gallbladder removed even saw a surgeon after the initial episode.

The risk of developing gallstones increases with age. While a person under 40 has about an 8 percent chance of developing gallstones, the risk jumps to more than 50 percent in people 70 years and older. Gallbladder disease is the most common cause of acute abdominal pain in older patients and removal of the gallbladder accounts for a third of abdominal operations in patients over 65.

The UTMB risk prediction model provides a starting point for individualized care and shared decision making in older patients with gallstones. Integrating this model into clinical practice, especially at the level of the primary care physician, may improve outcomes by increasing elective gallbladder removal rates in the patients at highest risk and prevent future complications in this vulnerable population. This information would also allow physicians to avoid gallbladder removal in patients who are a high surgical risk and at low risk for developing complications from their gallstones.

In patients with lower risk and the decision for gallbladder removal is based on preference, this information can help patients make decisions in the context of their symptoms and the impact on their quality of life.

The other authors of this paper include Deepak Adhikari, Abhishek Parmar, Suzanne Linder, Francesca Dimou, Winston Crowell, Nina Tamirisa, Courtney Townsend, Jr. and James Goodwin.

This paper was supported by the National Institutes of Health and the Agency for Healthcare Research and Quality. The study was presented at the 126th Annual Meeting of the Southern Surgical Association.

http://bit.ly/140Gytw

It's Beginning to Smell a Lot Like Christmas: The Neuroscience of Our Nostalgia

Have you ever smelled something so familiar that it felt like you were transported back through time into one of your earlier memories?

By Amanda Baker | December 23, 2014 |

Have freshly baked cookies, your grandmother's chili sauce, or a specific brand of sunscreen after a long winter actually affected the way you feel?

It turns out that science can explain this link between important memories and the smells associated with them. By studying the activity in our brains, scientists may even have an explanation why your uncle tries to tell you the same five stories about his childhood during the holidays each year.

Researchers have long known that episodic memories – the memories you have of particular events – are made up of the combined information from all of your senses (sights, sounds, smells, tastes, and touch). But trying to understand how this sensory information becomes a memory, or how it affects the way you are able to remember an event, is more complicated.

By looking at the structure of the brain, researchers have found that olfactory (smell) information takes a different path through the brain than input from the other senses. Most sensory information has to first go through a region called the thalamus before being interpreted by the other specialized parts of the brain. The thalamus receives input from many sources and then directs it to the right part of the brain. Scientists often consider this the step when a person becomes consciously aware of something.

When it comes to smells, the olfactory information actually passes through the olfactory memory and processing parts of the brain first. That means that we are actually processing the content and memory of a smell before we are consciously figuring out what that smell is.

This smell center of the brain, the olfactory cortex, is also strongly connected with two other areas of the brain: the limbic system and the amygdala. Both of these areas play strong roles in the emotional components of how memories are created and retrieved.

To understand the possible effect of this connection, one group of researchers exposed people to specific smells at the same time as emotionally intense videos. A week later the volunteers were given cues of either sounds, images, or smells from the earlier experience and asked to recall as many details as they could. The people who were reminded of the smell from the week before actually recalled more details about the emotional experience than the people who were given visual or auditory triggers.

Another group of researchers asked volunteers to create a story in their minds to form a link either between two images or an image and a particular smell. The scientists scanned the brains of the volunteers during the process of forming these connections and later when the people were asked to recall the story that they had created for themselves. They found that the part of the olfactory cortex that was activated when the volunteers were exposed to the smell the first time was activated again during the memory – even if the smell was not actually there.

But what does this mean for the smells we are exposed to and the things we remember every day?

A group of researchers in Sweden asked 72 people between the ages of 65 and 80 to describe the memories that came to mind after being exposed to a certain trigger. These triggers were 20 smells, 20 smells with names, or just the names of those 20 smells.

The researchers kept track of how many memories were brought up with each type of trigger, how old the people were at the time of these memories, how pleasant they felt, and how much they felt like they had been transported back in time.

Though the triggers with the names of smells led to the recall of more memories, the people who had been exposed just to smells not only felt more pleasant and felt more like they had been transported back in time, but also recalled memories from younger ages than the people in the other groups. In fact, more than twice as many of the memories were from under the age of ten when smells were one of the triggers.

Think about this in the context of all of the smells that are unique to the holiday season. Depending on where you are from or what holidays you celebrate, these might be burning candles, evergreen trees, cinnamon, crispy latkes, mulled wine, fresh tamales, or even meat on the grill if you live in the southern hemisphere. Whatever the holiday, when we are flooded with the smells of the season the olfactory cortex and our episodic memories may be transporting us back to memories from our childhoods.

So think about that the next time your grandmother wants to tell you about the first time she had gingerbread as a child. You could explain to her how that smell has made a journey through the olfactory parts of the brain, linked up with the thalamus, and probably even brought in the emotion centers of the brain just to bring on that strong sense of nostalgia. Or you could just listen to the story.

http://nyti.ms/1CG6Orr

Mysterious Virus That Killed a Farmer in Kansas Is Identified

Researchers have identified a previously unknown virus, thought to be transmitted by ticks or mosquitoes, that led to the death of a farmer in Kansas last summer.

By DENISE GRADY DEC. 23, 2014

The illness was fast-moving and severe, causing lung and kidney failure, and shock. The man, previously healthy, died after about only 10 days in the hospital, according to Dr. Dana Hawkinson, an infectious disease specialist who treated the patient at the University of Kansas Hospital in Kansas City.

The newly discovered microbe has been named the Bourbon virus, for the county where the patient lived, the Kansas Department of Health and Environment said in a statement released Monday. The virus was identified by scientists at the federal Centers for Disease Control and Prevention through a process that took several months, according to Dr. J. Erin Staples, a medical epidemiologist at the C.D.C. laboratory in Fort Collins, Colo.

She said the virus was a type of thogotovirus, part of a larger family known as orthomyxoviruses. Its nearest relatives are found in Eastern Europe, Africa and Asia, Dr. Hawkinson said. Those viruses are spread by ticks and mosquitoes.

Researchers do not yet know whether there have been other cases in the United States. They hope to test stored blood samples from people who had similar illnesses in the past that could not be identified. “I think we have to assume this has been around for some time, and we haven’t been able to diagnose it,” Dr. Hawkinson said. He added, “We suspect there have been milder cases and people have recovered from them, but we don’t have a lot of information.”

There is no treatment for the disease. The best defense is to avoid insect bites by wearing pants and long sleeves outdoors and applying bug spray that contains the repellent DEET.

The medical mystery began late last spring, when the patient was admitted to the hospital with a high fever, muscle aches and loss of appetite. He worked outdoors and often had tick bites. That history and his symptoms, combined with abnormal results on blood tests — his liver enzymes were too high, his platelets and white cells too low — made doctors suspect tick-borne diseases like Rocky Mountain spotted fever or ehrlichiosis. But tests for those illnesses came back negative.

Dr. Hawkinson suspected another, recently discovered tick-borne illness caused by the Heartland virus and sent blood samples to the C.D.C. for testing. But those tests also came back negative.

Researchers at the C.D.C. noticed that something else seemed to be growing in the samples that were tested for the Heartland virus, and they eventually identified the Bourbon virus. But the researchers are not certain that ticks or mosquitoes transmit the virus, or whether other animals might carry it.

“We will be working with state and local health departments come springtime to do extensive field investigations,” Dr. Staples said.

For now, the risk to the public is low because ticks and mosquitoes are not active in cold weather. But ticks rebound earlier in the year than mosquitoes do, she said, once the temperature starts consistently reaching 55 degrees Fahrenheit.

http://vult.re/16SOf6h

In an All-Digital Future, It’s the New Movies That Will Be in Trouble

When it comes to preserving movies for the long haul, the digital revolution may turn out to be something of a catastrophe

By Bilge Ebiri

The speed with which digital cinema took over the world has been nothing short of astonishing. Back in 2007, researchers forecasted that around 50 percent of the world’s movie screens would be digital by 2013 — which seemed like a pretty sci-fi prognostication at the time. In fact, by the end of 2013, the figure was closer to 90 percent. Last month, Christopher Nolan made news by actually daring to release *Interstellar* early to some theaters on 35mm (and 70mm) film. Within a few years, photochemical film has gone from an industry standard to a novelty act.

Progress, right? Digital files, as we’ve been told over and over again, don’t decay and fade and damage the way celluloid film does. The movie looks exactly the same the 100th time it’s projected as it did the first time. No rewinding. No lost reels, scratched prints, or pesky splices. You can store films on one smallish hard drive and easily copy them to another. Making a DCP (digital cinema package) of a film costs around $150, whereas striking a film print costs about $1500. Plus, the DCP can be shipped around the country more easily and cheaply than huge, heavy, clunky film reels. That has saved studios and distributors untold amounts of money — and, indeed, the studios were the ones who pushed theatrical conversion to digital hardest, in some cases refusing to make film prints available for theaters.

But when it comes to preserving movies for the long haul, the digital revolution may turn out to be something of a catastrophe. “At this time, the longevity of digital files of moving images is anybody’s guess,” says Paolo Cherchi Usai,

senior curator at George Eastman House, one of the nation's most significant motion-picture archives. “We do know that it is much, *much* shorter than the longevity of photochemical film.” If hard drives aren’t occasionally turned on, he notes, they start to become unusable.

“Digital preservation is really just an oxymoron at this point,” says Jan-Christopher Horak, director of the UCLA Film and Television Archive. “It’s really just putting plus and minus electronic charges on plastic — and that plastic has an extremely short half-life. So that most digital media, even if you take it and store it correctly, is probably not going to last more than eight or ten, maybe 15 years.” By contrast, with 35mm film, “we just need to put it into a cold, dark, dry place, pay the electricity bill, and it will last for 500 to a thousand years.”

In one of the most famous examples of the perils of digital preservation, when the makers of *Toy Story* attempted to put their film out on DVD a few years after its release, they discovered that much of the original digital files of the film — as much as a fifth — had been corrupted. They wound up having to use a film print for the DVD. “That was the first major episode to draw public attention to the fact that digital files are a challenge when it comes to conservation,” says Usai. (Somewhat hilariously and almost tragically, a similar fate came close to befalling *Toy Story 2*, which nearly got nuked when someone accidentally hit a “delete” button.)

The fate of *Toy Story* highlights a sad irony of the digital revolution: It’s the *newer* movies that are in trouble. For a long time, it was assumed that the real loser in our rapidly approaching all-digital future would be older films shot on celluloid, as they would have to be digitized at great cost in a world where movie theaters had forsaken film prints. (Horak estimates that only about 2 percent of UCLA’s 350,000-plus print archive has been digitized.) “My museum has preserved over 28,000 films, representing the history of cinema,” says Usai. “And it’s all largely photochemical. We try to make them available in digital form. But as long as the film itself is taken good care of as an object, I’m not worried, because if I lose the digital file, I can create a new one. But when we deal with something that was born in digital form, all we can do is migrate the digital files as often as possible.” And when you consider the sheer number of films that are out there, that requires technology and resources that go beyond what nonprofit organizations like film archives are able to handle. In other words, new movies that were *never* on film should be worried about what will happen to them in the future.

The physical deterioration of drives and discs and chips isn’t the only thing digital filmmakers need to worry about. Digital files are also prone to become outdated, with software upgrades and new programs that render previous ones obsolete or unusable. “We are still in the developmental stages of this digital technology, and formats are changing every 18 months to two years,” says Horak. “And many of these digital formats are not compatible with each other.” And each change in format can mean a cost of between $10,000 to $20,000 per film. (Meanwhile, movie theaters that converted to digital are in a similar conundrum, with formats and industry standards changing and each new wave of very expensive projectors breathlessly touted at tech conventions. By contrast, a movie theater could, with proper upkeep, use the same film projector for decades.)

Part of the problem, of course, is that preservation isn’t a for-profit endeavor. Movie companies, as they never cease reminding us, are businesses, and the idea of spending a lot of money and space to keep a bunch of old movies in storage probably seems like a waste of resources to a cost-cutting executive. But take a somewhat longer view, and the situation changes. “There’s this notion, which is not true, that digital is very inexpensive,” says Horak. “Filmmakers and studios *are* saving a lot of money in production and post-production costs because of digital, and that’s a good thing. But because of that, many people don’t really understand that they’re putting their assets at risk by wholesale transferring to digital and then not keeping the originals.” He adds: “This is not a new problem. In the 1970s and '80s, some film companies took all of their motion-picture film and transferred it to ¾-inch video, which was thought of as a preservation medium. They threw away their originals! And ¾-inch video was not a good format. In fact, it was a terrible format! This is happening with digital now. They’ve already sloughed off their nitrate collections, and there are actually discussions in some of the studios to get rid of their 35mm collections as well.”

Neither Horak nor Usai are complete pessimists in this regard, however. For starters, film may not be as dead as some seem to think. Archives around the world have discussed the idea of pooling their resources and manufacturing film themselves, if and when companies like Kodak or Agfa or Fuji go out of business. And the truth is that when it comes to digital technology, we’re still at the beginning, Horak notes. It will develop further, and sooner or later, there will be other strategies for the long-term preservation of digital material. “I even saw someone discussing the idea of shooting it all up into space and then waiting for it to come back around again,” he says. “That sounded like pure science-fiction, but who knows?”

So, despite the terrifying example of what almost happened to *Toy Story* and *Toy Story 2*, the big digital films are probably safe. You can bet that James Cameron and Fox are likely making sure that all the digital files associated with *Avatar* and its upcoming sequels will be duplicated many times and securely placed in multiple locations; that’s a multi-billion-dollar property, after all.

But what about everyone else? What about all those smaller, low-budget films that were lured by the promise of digital into shooting, storing, and projecting exclusively on video? What will happen to them over the course of what is sure to be multiple format changes? Is somebody making sure to turn *their* hard drives on every now and then to make sure that the files are still usable? Have *they* been distributed into multiple locations? Will their producers and distributors remain solvent enough over the years to make sure to update their files, or to splurge for a film print?

One hopes so, because the story of cinema is the story of discovery. Movies once considered afterthoughts — think of B-movie Westerns and noirs — or flops — think of *It's a Wonderful Life* — can, over time, become beloved classics. A print of a film long forgotten might turn up in a Norwegian archive and get revived. That may not be possible in an all-digital future, where moving-image files will need regular maintenance and upgrades to keep them viable. A forgotten movie, in other words, will be an extinct movie.

Celluloid is far from a perfect medium, but ironically, all those splices and scratches that we used to see as problems now seem like *strengths*: You can scratch a piece of film and it can still run through a projector. You can lose a frame or two — hell, you can lose an entire reel — and the rest of the film will still remain intact. Indeed, many of the great classics of the silent era were restored and reconstructed from multiple, incomplete film elements; usable parts of one print found in one corner of the world could be joined together with usable parts from another print found elsewhere. Not unlike with books, the simplicity of the physical medium held the key to its longevity. "Five hundred years from now, someone could look at a strip of film and probably reverse-engineer a projector from it," says Horak. "Not that it would ever last this long, but if somebody looked at a DVD 500 years from now, they wouldn't know what the hell it was."

http://www.bbc.com/news/world-us-canada-30591002

Gay US blood donor ban 'should end'

Gay and bisexual men should no longer be banned from donating blood in the US, the Food and Drug Administration (FDA) has recommended.

The ban has been in place since the HIV/Aids epidemic in the mid-1980s.

Men who have had sex with other men in the previous 12 months will still be banned, the FDA said in a statement. The move brings the US into line with other countries such as the UK, Australia and Spain, which have also lifted outright bans in recent years. New guidelines will be drafted in early 2015 and then a consultation period will follow.

Currently the ban excludes men who have had sex with other men at any time since 1977. Campaigners have long argued the ban discriminates against gay and bisexual men and reinforces negative stereotypes. And due to advances in HIV testing, the American Medical Association and other groups say the lifetime ban is no longer supported by science.

Blood donation around the world:

UK (excluding Northern Ireland), Japan and Australia have a one-year ban on men who have had sex with another man

Canada has a five-year ban

No ban in Italy, Mexico, Poland, Portugal, Russia and Spain, but some of those countries have tougher screening questions

"Over the past several years, in collaboration with other government agencies, the FDA has carefully examined and considered the available scientific evidence relevant to its blood donor deferral policy," the statement said.

It added: "The agency will take the necessary steps to recommend a change to the blood donor deferral period for men who have sex with men from indefinite deferral to one year since the last sexual contact."

The 12-month deferral period is because it takes on average two to four weeks to pick up an HIV infection when testing blood and a couple of months to detect Hepatitis B. But some activists are unhappy there are still any restrictions.

"While this proposed change is certainly historic, it would still mean that countless gay and bisexual men will be turned away from blood banks simply because of who they are," said the Gay and Lesbian Alliance Against Defamation.

Men and women of any sexual orientation are not permitted to give blood for one year after having sex with someone with HIV, with a prostitute or with an intravenous drug user.

http://www.bbc.com/news/world-africa-30595352

Ebola crisis likely to last a year, says expert

West Africa's Ebola crisis is likely to last until the end of 2015, says a leading researcher who helped to discover the virus.

Peter Piot, who has just returned from Sierra Leone, told the BBC that he was encouraged by progress there and by the promise of new anti-viral therapies.

But he also warned that vaccines would take time to develop.

The current Ebola outbreak, the deadliest to date, has so far killed more than 7,300 people. Most of the victims have been in Sierra Leone, Liberia and Guinea.

Prof Piot was one of the scientists who discovered Ebola in 1976 and is now Director of the London School of Hygiene and Tropical Medicine. He said that even though the outbreak has peaked in Liberia and was likely to peak in Sierra Leone in the next few weeks, the epidemic could have a "very long tail and a bumpy tail".

"The Ebola epidemic is still very much there. People are still dying, new cases are being detected," he told the BBC World Service's Newsday programme. "We need to be ready for a long effort, a sustained effort [for] probably the rest of 2015."

But he added that he was impressed by the progress that he had seen in Sierra Leone. "Treatment centres have now been established across the country with British help. You don't see any longer the scenes where people are dying in the streets," he said.

He also said he was also encouraged that thanks to simple treatments such as intravenous fluids and antibiotics, mortality rates had fallen to as low as one in three. "Getting it below that will require specific therapies that are now going to be tested," he said, adding that he hoped that within three months it would be clear which anti-viral therapies were effective.

Developing a vaccine would be more complicated, he said, but must be done "so that when there is another epidemic or maybe when this epidemic drags on for a long time, that we have that vaccine available".

http://phys.org/news/2014-12-economics-newly-veterinarians.html

The economics of newly graduated veterinarians

Animals can teach us more about the human body than we might realize.

December 24th, 2014 by Katie Allen in Biology / Other

Crack open New York Times bestseller "Zoobiquity," and you'll learn about a human cardiologist's experiences at the Los Angeles Zoo that allowed her to more closely connect human and animal medicine. Even in the first chapter—Dr. House, Meet Doctor Dolittle—author Barbara Natterson-Horowitz reveals how doctors and veterinarians could learn from each other to effectively diagnose and treat all species.

Indeed, veterinarians are needed not only to treat our pets and livestock, but in a broader context, to help with zoonotic disease maintenance. The interaction between animals and humans secures the continuous demand for the profession, and the fewer veterinarians we have, the larger potential for catastrophic disease, according to Michael Dicks, director of the economics division for the American Veterinary Medical Association (AVMA).

Although the profession is needed, Dicks said financial struggles do exist, especially for those beginning to practice. Many veterinarians who are just starting out find that they need to make enough money to pay off their high educational debt while trying to make a living, which can pose a major challenge.

According to Kansas State University's College of Veterinary Medicine, the average debt reported by its 2014 graduates was $170,380, and graduates in 2013 had similar debt at $170,919.

The average practice salary reported by 2014 K-State graduates was $64,678 and for 2013 graduates, $63,294. For those practicing outside of Kansas, the average starting salaries were a bit higher at $66,057 for 2014 graduates and $66,939 for those who graduated in 2013.

"The downturn of the economy impacted veterinary medicine and what graduates could earn in their first year," said Roger Fingland, executive associate dean for K-State's College of Veterinary Medicine and director of the Veterinary Health Center. "It is important to educate people who want to be veterinarians about the financial realities. But, I think the value of being a veterinarian has to always be in the discussion."

Seeking opportunities

Dicks, a veteran agricultural economist, said the objective of the AVMA's economics division is to find ways to enhance the lifelong value of a veterinary degree. Understanding the market for veterinary services and how individual veterinary practices make money are important components that add value.

The biggest area of demand in veterinary medicine is working with companion animals, or pets, which accounts for about six out of 10 practicing veterinarians, he said. Food animal veterinarians, those who work with cattle, sheep and pigs as examples, account for one out of 10. Other veterinarians might choose mixed animal practice or work in zoos, animal hospitals, the education field or other industries.

Scholarship opportunities are available for students in many interest areas, particularly for those who want to work in rural areas and seek mixed animal practice or large animal practice, including food animals and other livestock.

Fingland said at K-State, rural scholarship recipients receive $25,000 a year if they intend to practice in a Kansas county that is declared rural. Most counties in Kansas have a rural designation.

If the students take the scholarship and don't practice in a rural area, however, they have to pay that money back, he said. To his knowledge, no students have had to pay the money back, but some graduates will find that some rural areas don't have enough animals to support a full-time veterinarian. Or, perhaps the environment will not allow the veterinarian to charge the going rate for various services.

"I believe there is a need for large animal and mixed animal practitioners in some rural areas," Fingland said. "Need means there are consumers in that area who perceive that they need veterinary services. Needing veterinary services and having an environment that financially supports veterinary services are two different things. Some communities can't financially support the service."

Certain aspects of veterinary medicine are different than others, and veterinarians can choose certain avenues of practice to potentially increase their salaries. Industry veterinarians typically have higher earning potential than mixed animal practitioners, Fingland said.

Like human doctors, veterinarians can specialize as cardiologists, surgeons, internists and radiologists, as examples. Veterinarian specialists tend to make higher salaries, Fingland said, but they also have to go through much more training than general practitioners. Practice ownership also lends itself to higher pay.

Combining business with a calling

Most people go into business, because that's what they want to do. Veterinarians, no matter what area of practice they prefer, are no different, Dicks said. He encourages veterinarians, like any other business-minded professionals, to plan ahead to maximize their opportunities.

"We know just like farmers and ranchers, we all weigh life in some ways with the amount of money we're making," Dicks said. "People must give up a little return to have the style of life they want. For veterinarians, that huge (college) debt can be a restraint. If I have debt when I get out of school, that means I may be driving a 10-year-old car, living with my roommate for another five years, and not going out or buying anything. I may be paying my debt and trying to make a life."

Fingland, who teaches veterinary business courses, said on the first day of orientation at K-State, he presents new students and their parents with numbers showing what the education will cost for the next four years of veterinary school to make them aware.

"There is no question that there is a financial problem at work, and I worry about it like other people in my position worry about it," Fingland said. "But, there is value in doing what is your calling. I understand as a veterinarian that I'm not going to make as much money as someone in another profession. I don't want to be in that other profession, so what difference does it make if that person makes more money than I do?"

"We can't tell young people who aspire to be veterinarians, 'You shouldn't do this, because you won't make as much money as you could doing something else,'" he continued. "Is that what we're going to tell people who want to teach? Imagine if somebody would have turned away the wonderful teachers that we had in grade school, high school and college. That would have been very unfortunate."

Fingland said preparing students to be business-minded veterinarians involves work in and out of the classroom. In addition to one required business and finance course, veterinary students at K-State are allowed to take elective business courses and join organizations such as the Veterinary Business Management Association. They also learn about planning and budgeting through K-State's Powercat Financial Counseling, available as a free resource for all students.

Involving the veterinarian

As a livestock producer, Dicks said he believes it is essential to have a veterinarian as part of your health team if you own animals. Sometimes the veterinarian might be considered a provider of last resort or someone whose job can be handled by salesmen or technicians for artificial insemination, embryo transfer, ultrasounding for carcass characteristics or pregnancy, hoof trimming and nutritional planning, as examples.

But, he said in the last 10 years the cattle industry has experienced diseases such as trichomoniasis and curly calf syndrome, among others, that may have surprised some producers. Having a close relationship with a veterinarian could help protect animals from diseases and producers from major financial losses.

"Maybe because a veterinarian wasn't part of our herd health program, we only found out about these diseases once they happened to us," Dicks said. "Some of those things cause 20 to 30 percent losses."

"What we focus on is teaching veterinary students to thrive in a competitive environment, not in an unrealistic environment where there's no competition," Fingland said. "There are many things veterinarians can do that others can't do who don't have the level of training. No one will ever replace the veterinarians' intellect, when they go to a farm to analyze the nutrition that the rancher or farmer is providing, and the environment and how that environment might lead to disease."

http://phys.org/news/2014-12-fecal-microbes-earth-evolution.html

What the 'fecal prints' of microbes can tell us about Earth's evolution

The distinctive "fecal prints" of microbes potentially provide a record of how Earth and life have co-evolved over the past 3.5 billion years as the planet's temperature, oxygen levels, and greenhouse gases have changed.

But, despite more than 60 years of study, it has proved difficult, until now, to "read" much of the information contained in this record. Research from McGill University and Israel's Weizmann Institute of Science, recently published in the *Proceedings of the National Academy of Sciences* (*PNAS*), sheds light on the mysterious digestive processes of microbes, opening the way towards a better understanding of how life and the planet have changed over time.

Everything that eats must excrete, and microbes are no exception.

Microbes have dominated the Earth's ecology for at least the past 3.5 billion years. They play a vital role in the planet's carbon cycle by digesting organic matter. So

their waste potentially carries information about how the planet's temperature, greenhouse gas composition, and even oxygen levels have changed over time, along with information about how life itself has evolved to accommodate these changes. But though scientists have been trying to grasp how to interpret the information from these microbial "fecal prints" for more than sixty years, the solution has proved to be elusive until now.

Microbes are ultra-picky diners

In a paper recently published in the *Proceedings of the National Academy of Sciences* (*PNAS*), researchers from McGill University and Israel's Weizmann Institute of Science describe a new technique they have developed to interpret these distinctive metabolic traces. They chose to focus on the microbes that live on the ocean floor where the microbes consume the sulfate found in seawater because oxygen is in short supply.

Global temperatures, carbon dioxide concentrations, and oxygen levels all determine whether these sulfate-using microbes are living in times of plenty, and growing fast, or in times of need, and growing slowly. The record of these changes is to be found in the microbial wastes and more specifically in how much, or how little, of the sulfate compound the microbes trim off.

The microbes are ultra-picky diners. Like many humans, they prefer to keep their consumption light. And just as careful carnivores will trim the fat from the edge of their steak, these microbes tend to reject sulfur if it is just a neutron or two heavier than normal. (Neutrons are atomic particles, and so are very, very small.) In times of plenty, as growth speeds up and the microbes need to take in more sulfate they are less discriminating and will trim off, or "fractionate" less. Like a glutton is wolfing down a slab of roast beef, they don't spend time cutting off the fat. But in times when resources are more limited, as growth slows down, microbes trim off or fractionate more. It's like dining with a freegan who insists on trimming off the rank exterior of an expired rump roast to find the single unspoiled piece of meat inside.

The new research by Boz Wing and Itay Halevy explains these peculiar dining preferences for microbes and, for the first time, links it to how much of what they consume is stored inside their cells. Although the "fecal print" analysis was developed for the sulfate-reducing microbes found under the seabed, as Wing's co-author Halevy of points out, the new work has larger implications. "It can be applied to many other microbial metabolisms that are important to earth system functioning today, from the denitrifiers that drive Earth's nitrogen cycle to the microbes that produce the greenhouse gas methane."

Wing credits a sharp McGill undergrad with asking the question that inspired this research. "When I started at McGill I told an undergrad that we were going to grow some microbes in the lab to see how they fractionated, so that then we could look for this as a bio signature in some wicked old rocks," says Wing. "She gave me a super skeptical look and asked if I knew that microbes evolve. That fundamental question is now behind the majority of the research in my lab, where we are trying to understand ever-evolving relationship between our planet and its microbial majority."

More information: *"Intracellular metabolite levels shape sulfur isotope fractionation during microbial sulfate respiration." Boswell A. Wing, PNAS, 18116–18125, DOI: 10.1073/pnas.1407502111*

http://phys.org/news/2014-12-phosphorus-river.html

Project cuts phosphorus levels in river

A seven-year pilot project has helped to reduce the amount of phosphorus entering the Pecatonica river in southwestern Wisconsin

December 24th, 2014 by Bob Mitchell in Earth / Environment

Conservation experts and farmers alike are rather pleased with the news out of southwestern Wisconsin. A seven-year pilot project in the 12,000-acre Pleasant Valley subwatershed of the Pecatonica River has helped to reduce the amount of phosphorus and sediment entering the river after major storms by more than a third. The project involved changing practices on just 10 of the valley's 61 farms. Certain practices, such as reducing tillage and planting crops that leave more residue to protect the soil, caused the estimated annual amounts of phosphorus and sediment entering the river to drop by 4,400 pounds and 1,300 tons, respectively.

"We can say with 90 percent confidence that this project made a real reduction in the phosphorus losses," says Laura Ward Good, a soil scientist at the University of Wisconsin-Madison College of Agricultural and Life Sciences (CALS). "Farmers who changed their management practices reduced their estimated phosphorus and sediment losses by about half." The project was launched in 2006 by a multi-institutional team that included University of Wisconsin-Madison scientists, public agencies, local farmers and The Nature Conservancy.

Researchers collected baseline data on water quality in the Pecatonica south of Pleasant Valley and a nearby watershed that served as a control. Team members then identified high-risk fields and worked with landowners to assess the likely impacts of switching practices in selected fields—not just on runoff, but also on yields, expenses, feed supplies and other factors. A key tool for the team was SnapPlus, a software program developed at CALS that estimates each field's potential for phosphorus runoff under various management scenarios.

"In many cases the higher risk areas were fields on steep slopes, where silage had been grown in consecutive years so there wasn't much crop residue to hold the

soil, and where soil phosphorus levels were high—possibly because past manure applications had supplied more phosphorus than crops required," Good says.

The results indicate that farmers can make changes without reducing their bottom lines if the practices are tailored to individual needs and growers can proceed gradually. For example, farmers who don't till can still see high production from their acres, though that approach requires a closer attention to detail and leaves little room for mistakes, says UW-Extension specialist Jim Leverich, the project's ongoing farm research coordinator.

"The trick is to give farmers the time to adapt, to search among the best management practices to see how they fit into their systems," Leverich says. "If they have time to utilize the practice on a small scale first, they'll start to see the advantages and maybe start to use it on more acres."

http://bit.ly/13GqPPF

Scrapie could breach the species barrier

INRA scientists have shown for the first time that the pathogens responsible for scrapie in small ruminants (prions) have the potential to convert the human prion protein from a healthy state to a pathological state.

In mice models reproducing the human species barrier, this prion induces a disease similar to Creutzfeldt-Jakob disease. These primary results published in Nature Communications on 16 December 2014, stress the necessity to reassess the transmission of this disease to humans.

Scrapie is a neurodegenerative disease that has been known for centuries and which affects sheep and goats. Similar to Bovine Spongiform Encephalopathy (BSE) or mad cow disease, scrapie is caused by a transmissible pathogen protein called prion.

However, and contrary to BSE, epidemiological studies have never been able to establish a link between this disease and the occurrence of prion diseases in humans. "Risks of transmitting scrapie to humans (zoonose) were hitherto considered negligible because of the species barrier that naturally prevents prion propagation between species", said Olivier Andreoletti, INRA scientist who led the present study.

Researchers at INRA studied the permeability of the human transmission barrier to pathogens responsible for scrapie, using animal models specifically developed for this purpose. This approach previously allowed the confirmation of the zoonotic nature of prions responsible for BSE in cows and of the variant of Creutzfeldt-Jakob disease in humans (vCJD).

Unexpectedly, in these rodent models, certain pathogens responsible for scrapie were able to cross the transmission barrier. Moreover, the pathogens that propagated through this barrier were undistinguishable from the prions causing the sporadic form of Creutzfeldt-Jakob disease (sCJD). This data suggest a potential link between the occurrence of certain sCJD and these animal prions. "Since CJD is scarce, about 1 case per million and per year, and incubation periods are usually long -several decades- it is extremely difficult for epidemiological studies to try and make this link", explains Olivier Andreoletti.

In their conclusions, the authors stress the fact that CJD cases are rare though scrapie has been circulating for centuries in small ruminants for which we eat the meat. Even if in future studies scrapie is finally confirmed to have a zoonotic potential, the authors consider that this disease does not constitute a new major risk for public health.

More information: Hervé Cassard, Juan-Maria Torres, Caroline Lacroux, Jean-Yves Douet, Sylvie Benestad, Frédéric Lantier, Séverine Lugan, Isabelle Lantier, Pierrette Costes, Naima Aron, Fabienne Reine, Laetitia Herzog, Juan-Carlos Espinosa, Vincent Beringue and Olivier Andréoletti. "Evidence for zoonotic potential of ovine scrapie prions." Nature Communications, 16 December 2014. DOI: 10.1038/10.1038/NCOMMS6821

http://www.eurekalert.org/pub_releases/2014-12/sfu-ssh122314.php

SFU scientists help put bedbugs to bed forever

The world owes a debt of gratitude to Simon Fraser University biologist Regine Gries.

Her arms have provided a blood meal for more than a thousand bedbugs each week for five years while she and her husband, biology professor Gerhard Gries, searched for a way to conquer the global bedbug epidemic.

Working with SFU chemist Robert Britton and a team of students, they have finally found the solution--a set of chemical attractants, or pheromones, that lure the bedbugs into traps, and keep them there.

This month, after a series of successful trials in bedbug-infested apartments in Metro Vancouver, they have published their research, Bedbug aggregation pheromone finally identified, in Angewandte Chemie, a leading general chemistry journal. They're working with Victoria-based Contech Enterprises Inc. to develop the first effective and affordable bait and trap for detecting and monitoring bedbug infestations. They expect it to be commercially available next year.

"The biggest challenge in dealing with bedbugs is to detect the infestation at an early stage," says Gerhard, who holds an NSERC-Industrial Research Chair in Multimodal Animal Communication Ecology. "This trap will help landlords, tenants, and pest-control professionals determine whether premises have a bedbug problem, so that they can treat it quickly. It will also be useful for monitoring the treatment's effectiveness." It's a solution the world has been waiting for.

Over the last two decades the common bedbug (Cimex lectularius), once thought eradicated in industrialized countries, has reappeared as a global scourge. These

nasty insects are infesting not just low-income housing but also expensive hotels and apartments, and public venues such as stores, movie theatres, libraries and even public transit. And while these blood-sucking pests were previously not considered a carrier of disease, scientists have recently discovered they can transmit the pathogen that causes Chagas disease, which is prevalent in Central and South America. Yet until now, tools for detecting and monitoring these pests have been expensive and technically challenging to use.

The research was funded with a Natural Sciences and Engineering Research Council of Canada industry grant in partnership with Contech Enterprises Inc.

Backgrounder: The research story--180,000 bedbug bites later

The Gries' began their research eight years ago when Gerhard, who is internationally renowned for his pioneering work in chemical and bioacoustic communication between insects, began searching for pheromones that could lure and trap bedbugs.

Regine worked with him, running all of the lab and field experiments and, just as importantly, enduring 180,000 bedbug bites in order to feed the large bedbug colony required for their research. She became the unintentional "host" because, unlike Gerhard, she is immune to the bites, suffering only a slight rash instead of the ferocious itching and swelling most people suffer.

The Gries' and their students initially found a pheromone blend that attracted bedbugs in lab experiments, but not in bedbug-infested apartments. "We realized that a highly unusual component must be missing--one that we couldn't find using our regular gas chromatographic and mass spectrometric tools," says Gerhard.

That's when they teamed up with Britton, an expert in isolating and solving the structure of natural products, and then synthesizing them in the lab. He used SFU's state-of-the-art NMR spectrometers to study the infinitesimal amounts of chemicals Regine had isolated from shed bedbug skin, looking for the chemical clues as to why the bedbugs find the presence of skin so appealing in a shelter. It was like looking for a needle in a haystack.

After two years of frustrating false leads, Britton, his students and the Gries duo finally discovered that histamine, a molecule with unusual properties that eluded identification through traditional methods, signals "safe shelter" to bedbugs. Importantly, once in contact with the histamine, the bedbugs stay put whether or not they have recently fed on a human host.

Yet, to everyone's disbelief, neither histamine alone nor in combination with the previously identified pheromone components effectively attracted and trapped bedbugs in infested apartments. So Regine began analyzing airborne volatile compounds from bedbug faeces as an alternate source of the missing components. Five months and 35 experiments later, she had found three new volatiles that had never before been reported for bedbugs. These three components, together with two components from their earlier research and, of course, histamine, became the highly effective lure they were seeking.

Their research isn't over yet, however. They continue to work with Contech Enterprises to finalize development of the commercial lure--which means Regine is still feeding the bedbugs every week. "I'm not too thrilled about this," admits Regine, "but knowing how much this technology will benefit so many people, it's all worth it."

http://bit.ly/1wJkiPK

World's Largest Floating Solar Plant Planned for Japan

Engineers in Japan hope to harness sun's power with construction of what will be the world's largest floating solar power installation

Dec 24, 2014 10:14 AM ET // by Glenn McDonald

If you've ever been out in a boat on a hot summer day, you know that open water gathers a lot of sun and heat. Engineers in Japan are hoping to harness that power with the construction of what will be the planet's largest floating solar power installation.

Japan's Kyocera Corporation has already leveraged the power of open water with shoreline solar installations like the fixed Kagoshima Nanatsujima plant, pictured above. The new project, however, will be built around 50,000 solar collection modules actually afloat on the Yakamura Dam reservoir.

An image of the Kyocera Corporation's existing Kagoshima Nanatsujima power plant in Japan. The company's new project will be the largest fully-floating solar installation in the world.

The modules will cover a water surface area of around 180,000 square meters. Engineers estimate the plant will generate more than 15.6 megawatt hours (MWh) per year. That's enough to power approximately 4,700 average households.

More numbers: According to the company's projections, the floating power plant will gather enough solar power from the surface of the dam to offset about 7,800 tons of carbon dioxide emissions annually. The facility will also include an education center adjacent to the plant, to provide classes for local students on environmental issues.

"When we first started R&D for solar energy in the mid 1970's, the technology was only viable for small applications such as street lamps, traffic signs and

telecommunication stations in mountainous areas," said Nobuo Kitamura, Kyocera senior executive officer, in press materials for the project.
"Since then, we have been working to make solar energy use more ubiquitous in society. We are excited to work with our partners on this project, taking another step forward by utilizing untapped bodies of water as solar power generation sites."

http://www.eurekalert.org/pub_releases/2014-12/wios-hpc121814.php

Human primordial cells created in the lab

A cell programming technique developed at the Weizmann Institute turns them into the earliest precursors of sperm and ova

Groups at the Weizmann Institute of Science and Cambridge University have jointly managed the feat of turning back the clock on human cells to create primordial germ cells - the embryonic cells that give rise to sperm and ova - in the lab. This is the first time that human cells have been programmed into this early developmental stage. The results of their study, which were published today in Cell, could help provide answers as to the causes of fertility problems, yield insight into the earliest stages of embryonic development and potentially, in the future, enable the development of new kinds of reproductive technology.
"Researchers have been attempting to create human primordial germ cells (PGCs) in the petri dish for years," says Dr. Jacob Hanna of the Institute's Molecular Genetics Department, who led the study together with research student Leehee Weinberger. PGCs arise within the early weeks of embryonic growth, as the embryonic stem cells in the fertilized egg begin to differentiate into the very basic cell types. Once these primordial cells become "specified," they continue developing toward precursor sperm cells or ova "pretty much on autopilot," says Hanna. The idea of creating these cells in the lab took off with the 2006 invention of induced pluripotent stem (iPS) cells - adult cells that are "reprogrammed" to look and act like embryonic stem cells, which can then differentiate into any cell type. Thus several years ago, when researchers in Japan created mouse iPS cells and then got them to differentiate into PGCs, scientists immediately set about trying to replicate the achievement in human cells. But until now, none had been successful.
Previous research in Hanna's lab pointed to new methods that could take human cells to the PGC state. That research had focused on the question of how human iPS cells and mouse embryonic cells differ: The mouse embryonic cells are easily kept in their stem cell state in the lab, while human iPS cells that have been reprogrammed - a technique that involves the insertion of four genes - have a strong drive to differentiate, and they often retain traces of "priming." Hanna and his group then created a method for tuning down the genetic pathway for differentiation, thus creating a new type of iPS cell that they dubbed "naïve cells." These naïve cells appeared to rejuvenate iPS cells one step further, closer to the original embryonic state from which they can truly differentiate into any cell type. Since these naïve cells are more similar to their mouse counterparts, Hanna and his group thought they could be coaxed to differentiate into primordial germ cells. Working with naïve human embryonic stem and iPS cells, and applying the techniques that had been successful in the mouse cell experiments, the research team managed to produce cells that, in both cases, appeared to be identical to human PGCs. Together with the lab group of Prof. Azim Surani of Cambridge University, the scientists further tested and refined the method jointly in both labs. By adding a glowing red fluorescent marker to the genes for PGCs, they were able to gauge how many of the cells had been programmed. Their results showed that quite a high rate - up to 40% - had become PGCs; this quantity enables easy analysis.
Hanna points out that PGCs are only the first step in creating human sperm and ova. A number of hurdles remain before labs will be able to complete the chain of events that move an adult cell through the cycle of embryonic stem cell and around to sperm or ova. For one, at some point in the process, these cells must learn to perform the neat trick of dividing their DNA in half before they can become viable reproductive cells. Still, he is confident that those hurdles will one day be overcome, raising the possibility, for example, of enabling women who have undergone chemotherapy or premature menopause to conceive.
In the meantime, the study has already yielded some interesting results that may have significant implications for further research on PGCs and possibly other early embryonic cells. The team managed to trace part of the genetic chain of events that directs a stem cell to differentiate into a primordial germ cell, and they discovered a master gene, Sox17, that regulates the process in humans, but not in mice. Because this gene network is quite different from the one that had been identified in mice, the researchers suspect that more than a few surprises may await scientists who study the process in humans.
Hanna: "Having the ability to create human PGCs in the petri dish will enable us to investigate the process of differentiation on the molecular level. For example, we found that only 'fresh' naïve cells can become PGCs; but after a week in conventional growth conditions they lose this capability once again. We want to know why this is. What is it about human stem cell states that makes them more or less competent? And what exactly drives the process of differentiation once a cell has been reprogrammed to its more naïve state? It is the answers to these basic questions that will, ultimately, advance iPS cell technology to the point of medical use."

This collaborative project was made possible by a grant from BIRAX Britain Israel Research and Academic Exchange Partnership - Regenerative Medicine Initiative.
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http://bit.ly/1t1NaxE

There's More to Frankincense and Myrrh Than Meets the Eye

Frankincense and myrrh have interesting medicinal properties

By Colin Schultz

As per the Biblical tale, as recounted in Matthew 2:1-12, an infant Jesus of Nazareth was visited in Bethlehem on the eve of his birth by Magi bearing gifts of gold, frankincense and myrrh.

To our modern sensibilities, these three gifts don't seem quite even. Gold, then as now, is a highly valuable treasure. But frankincense and myrrh... what even are they?

According to Simon Cotton for Chemistry World, frankincense and myrrh are sap, drawn from the Boswellia sacra and Commiphora trees, respectively.

Frankincense was often burned as an incense, while myrrh made its way into medicine and perfume. In antiquity, writes Cotton, these saps were worth just as much as gold.

But as modern science has shown, these Magi (or wise men or kings, as they've come to be known) may have been onto something with their gifts. More than just aromatic compounds, frankincense and myrrh have interesting medicinal properties.

"From tests on mice, chemists at the University of Florence have found that molecules in myrrh act on the brain's opioid receptors, explaining its painkilling action," says Cotton.

The key active ingredient in frankincense, boswellic acid, meanwhile, "has a structure not dissimilar from some hormones like testosterone."

Boswellic acids have anti-inflammatory and antiarthritic effects, so that they are finding pharmacological use in both East and West. These compounds seem to work by preventing the body from making pro-inflammatory compounds, whilst they also exert antitumor effects in colorectal cancer cells.

Atop its analgesic action, myrrh also seems to have anti-cancer properties.

Of the Magi's three gifts, maybe gold was the least valuable of all?

http://bit.ly/13xGXT6

Imagination, Reality Look Different in the Brain

"Turn off your mind, relax, and float down stream..."

Maybe John Lennon was onto something when he wrote those words for the Beatles' song "Tomorrow Never Knows."

It turns out that that reality and imagination flow in different directions in the brain, researchers say. The visual information from real events that the eyes see flows "up" from the brain's occipital lobe to the parietal lobe, but imagined images flow "down" from the parietal to the occipital.

"There seems to be a lot in our brains and animal brains that is directional — that neural signals move in a particular direction, then stop, and start somewhere else," said Dr. Giulio Tononi, a psychiatry professor and neuroscientist at the University of Wisconsin-Madison and one of the study's co-authors. "I think this is really a new theme that had not been explored."

The finding, published in the November issue of the journal NeuroImage, may lead to a better understanding of how the brain processes short-term memories and how memory is connected to imagination, the researchers said.

By "flow," the scientists are referring to the general direction of electrical signaling of neurons in the brain. This direction is oriented against the various lobes of the brain. [Inside the Brain: A Photo Journey Through Time]

The occipital lobe sits in the lower, back part of the brain. Containing the visual cortex, this lobe's primary function is to process visual information. The parietal lobe lies above the occipital lobe, and its primary function is to integrate sensory information, such as vision, but also touch and sound. In doing so, the parietal lobe assembles elementary building bricks from so-called "lower-order" brain regions to create concepts, said Daniela Dentico, a researcher at the University of Wisconsin-Madison and lead author on the report.

A leading theory in image processing "posits that our visual mental images are not stored somewhere in the brain, but get actively reconstructed," Dentico told Live Science. The brain does this, she said, by reversing the order it uses for visual perception. She described this as the "top-down" direction, which starts from the big concept and moves back toward the smaller elements.

"Our study represents the first direct measure of the prevalence of top-down signal flow during imagery," Dentico added.

To determine the flow of neural firing, the Madison researchers, along with scientists at University of Liege in Belgium, asked study participants who were hooked up to an electroencephalography (EEG) machine to watch videos or to imagine fantastical scenes, such as traveling on a magic bicycle. EEG is an established technique that uses sensors on the scalp to measure underlying

electrical activity. But because the brain isn't "quiet," EEGs tend to reveal the cacophony of brain activity, said Barry Van Veen, a professor of electrical and computer engineering at Madison and senior author on the report.

So, to zero in on the flow of reality and imagination, the researchers created complex statistical modeling requiring high-throughput computing. From this, they could determine, for the first time, the average directional flow of neural firing during the tasks of seeing and imagining.

The researchers could not determine, however, whether imagination originates in the parietal lobe. It may instead flow through the parietal lobe from the frontal lobe, the brain region most associated with human intelligence. This is a topic for further investigation, the researchers said.

http://bit.ly/1AMBsNL

Why There's a Dangling Thing In Our Throats

The riddle of the uvula may not be solved yet, but it looks like we have some ideas

By Marissa Fessenden

Apparently, humans have long looked into the back of their throats, or down others', at the bit of flesh that dangles and hangs there and wondered: What is that thingy for? A paper published in 1992 titled "The riddle of the uvula" references a history of "interesting and contradictory observations."

Science journalist Robbie Gonzalez recently rounded up some of the theories and unknowns surrounding the uvula at io9. He asked Katie Plattner, a speech-language pathologist for the L.A. Unified School District why we have them:

"I think one theory is to increase surface area where a gag reflex can be triggered so nothing too big gets down there without you knowing," she replied. She then added: "I also might have made that up, but [it] seems right."

When I asked Plattner if I could quote her, she said "If you include that I don't really know, sure!" Why would I quote her if she's not really sure? Because it turns out the theory she floats actually stands up. More importantly, though, her qualification of that theory captures a lot of the uncertainty that surrounds the uvula and its function. The gag theory is supported by a review that pins the uvula down as one of five zones that triggers the gag reflex.

The uvula may be important for speech - it's used to make uvular sounds (think of the trilled r's in French), but it could also affect voice quality. At least one study notes that whatever it does isn't noticeable enough to prevent uvular removal from being routine. In northern Morocco, a physician observed that many people had their uvula snipped out, he reported in 1965. He wrote, "The absence of uvula was in all cases imputable to surgical removal during childhood, mostly soon after birth. The operation is performed on infants of both sexes; it is carried out either by the caretaker or sexton of the neighborhood mosque, or by a barber (*mohallem hayyam*). Many barbers in tradition-fast Morocco are still practical surgeons as they were in medieval Europe."

The "Riddle of the uvula" paper observed that the human uvula is larger and more complex in humans than in baboons. (It wasn't even present in other mammals.) They argue that this supports the idea that the uvula is important for speech, a feature that distinguishes humans. The researchers also noted that uvula can produce a "large quantity of fluid salvia that can be excreted in a short time."

Maggie Ryan Sandford at *Mental Floss* also compiled a list of theories about the uvula and some problems:

That it once helped guide the flow of food and water, and, in humans, was a mere remnant from previous mammals who had to lean down to eat and drink.

That it contributed to "chronic cough." A problem that 19th-century doctors treated with a "simple" "clipping" procedure.

That it contributes to cardiovascular problems like sudden infant death syndrome (SIDS) and sleep apnea.

It could be that all the theories are true to some extent, but as of now the uvula remains a curious structure to ponder.

http://bit.ly/16T1KTw

Renewable energy companies use new clout in statehouses

Earlier this year, Ohio became the first state to freeze a scheduled increase in the amount of electricity that must be generated by wind, solar and other renewable sources.

December 24th, 2014 by Jeffrey Stinson

The move gave advocates of repealing states' mandatory green energy standards a rare victory after defeats the last two years. But the Ohio victory may have been an aberration: Green energy industries have become mainstream businesses with the political clout to match the fossil fuel industry and big electric utilities in many statehouses, and they are using that influence to defend the renewable energy standards in place in 31 states and the District of Columbia.

Green industry is creating jobs, providing lease payments to landowners and taxes for local government in many states. Companies like Siemens and GE are highly invested in green energy. And many state lawmakers don't want to see the economic benefits shrink or disappear.

Wind represents about $118 billion in private investment in the U.S. economy and sustains about 73,000 jobs, according to the American Wind Energy Association. About $17.3 billion a year is invested in new wind farms. The solar industry, meanwhile, employs about 143,000 people and pumps nearly $20 billion a year into the economy, according to the Solar Energy Industries Association.

The economic impact of the fossil fuel industry is much larger, but Tom Plant of the Center for the New Energy Economy at Colorado State University noted that green energy has "become mainstream ... and a pretty significant component of economies of the states."

Nevertheless, Ohio's action gave hope to repeal advocates like John Eick of the American Legislative Exchange Council (ALEC) that other states will follow this coming year and slow or modify the mandates, if not repeal them outright.

"It (Ohio) may have laid the groundwork for other states to move in this direction in the coming year," said Eick, director of the task force on energy, environment and agriculture for ALEC, the free-market think tank of state lawmakers and private industry that drafted model repeal legislation.

State renewable portfolio standards are an outgrowth of the energy deregulation movement of the 1990s and rising concern over the environmental damage caused by greenhouse gases. The standards require utilities to sell an escalating amount of power generated by renewable or alternative fuels, which can vary from wind and solar to biomass, geothermal and hydroelectric. Many states also require greater energy efficiencies as part of the package.

Ohio's 2008 law required utilities to gradually phase in the purchase of renewable, alternative and emerging energy technologies until it comprised 25 percent of their electricity output by 2025. It also mandated a 22 percent reduction in consumption by then.

The mandates are now frozen until 2017 while a legislative commission studies whether the standards contribute to rising electricity rates and should be altered.

How much electricity must come from renewable sources varies across the states. California, for instance, requires 25 percent by 2017 and 33 percent by 2020, according to a database kept by the North Carolina Clean Energy Technology Center in conjunction with the U.S. Department of Energy. Connecticut requires 27 percent by 2020.

In addition to states with mandatory standards, seven - Alaska, Indiana, North Dakota, Oklahoma, South Dakota, Utah and Virginia - have voluntary goals.

Combined with federal and state green energy tax breaks, state renewable portfolios have been instrumental in building the renewable sector. They "drive demand," said Susan Sloan, vice president for state policy at the American Wind Energy Association. Technological advancement is making green energy more economically competitive compared to fossil fuel, especially coal.

Although debate rages on how much the mandates cost ratepayers, a survey in May by the National Renewable Energy Laboratory calculated that from 2010-2012 they drove up rates by 0.9 percent.

Electricity generated by wind power costs $28-$32 a megawatt hour, Colorado State's Plant said, while natural gas is about $45 a megawatt hour and coal generation is $48-$50 per megawatt hour.

Green energy will get another boost from new U.S. Environmental Protection Agency rules that will force old coal-fired plants to cut their carbon emissions by 30 percent. That will make coal generation even more costly, said Steve Kalland, executive director of the North Carolina Clean Energy Technology Center at North Carolina State University.

Battles over the portfolios heated up two years ago after ALEC drafted model legislation to roll back or repeal them because, Eick said, "government entities shouldn't distort the energy markets with mandated quotas favoring one source of power over another." Eick's task force, which drafted the legislation, now comprises 130 state legislators from across the country and about 50 companies, including fossil fuel producers and investor-owned utilities.

Although the task force has no renewable energy companies as members, Eick said, ALEC isn't anti-green energy. The group doesn't favor any one source of energy, he said. Nor is it opposed to voluntary renewable goals. But, he said, the markets shouldn't be distorted, and electric customers shouldn't have affordability and reliability put at risk by government intervention.

Twenty-six bills to roll back or repeal the mandates were introduced in 2013, after Republicans won a large number of legislative seats in the 2012 election, according to tracking by Colorado State's Center for the New Energy Economy. None passed. Just 14 were introduced this year. Only Ohio rolled back in a significant way.

Ohio's freeze, approved by the Republican legislature, was signed by Republican Gov. John Kasich in June after he first threatened to veto a rollback.

In addition to ALEC, the utility First Energy and the Ohio Chamber of Commerce pushed for the rollback. On the other side, the Ohio Manufacturers Association and companies like Whirlpool and Honeywell opposed the rollback. Wind energy accounts for about 5,000 jobs in the state, the Environmental Defense Fund said.

Wind, solar and other alternative sources are increasingly popular with the public compared to fossil fuels, polls indicate. And many companies want to demonstrate they're in favor of environmentally friendly policies.

If there's any state that could follow Ohio's lead this coming year, Eick said, it is Kansas, where standards mandate 20 percent renewables by 2020.

Efforts to first repeal and then phase out the standards were defeated in the House this year after winning overwhelming Senate approval. The repeal's sponsor, Republican Rep. Dennis Hedke, said it's too early to say whether he will push again. But the climate would appear ripe, especially now that Republican Gov.

Sam Brownback says he supports phasing out the standards after previously remaining silent on the issue.

Kansas is home to energy giant Koch Industries, which backs ALEC and Americans for Prosperity, whose state chapter favors repeal. The state is the nation's 10th biggest producer of oil and home to the Hugoton natural gas fields. And, according to the Energy Information Administration, 61 percent of the state's electricity comes from coal plants.

However, wind energy has a foothold in the state and accounts for about 19 percent of electricity generation. The industry has made an $8 billion capital investment in the state, accounting for some 13,000 jobs, said Karin Brownlee, spokesperson for Kansans for Wind Energy. Siemens manufactures housing for its wind turbines there.

Wind has become "a big cash crop" for Kansas farmers, she said, with wind farms providing about $16 million a year in lease payments to landowners. Local government receives about $10 million a year in lieu of tax payments from them, which is good revenue for rural counties.

Brownlee, a former Republican state senator and former ALEC member, said she doesn't know whether another repeal effort will erupt when lawmakers convene in January. But, she said, the industry now is a "huge" part of the Kansas economy and is prepared with the facts for any fight.

http://s.nikkei.com/1tgnZNy

Methane hydrate extracted in Sea of Japan off northern Japan

successful extraction of methane hydrate from the seabed in the Sea of Japan off northern Japan

TOKYO (Kyodo) -- The Japanese government has succeeded in extracting samples of a next-generation resource called methane hydrate from the seabed in the Sea of Japan off northern Japan, the Agency for Natural Resources and Energy said Thursday.

Researchers conducted drilling surveys and were able to obtain samples of the "fiery ice" under the ocean floor off Niigata Prefecture north of Tokyo as well as the two northern Japan prefectures of Akita and Yamagata. The samples collected were up to 1 meter thick. The agency, an arm of the Ministry of Economy, Trade and Industry, will continue its three-year survey through fiscal 2015 on methane hydrate -- an ice-like substance consisting of methane and water -- and aims to assess the amount of reserves in the Sea of Japan.

The agency also said geological structures suggesting possible reserves of the natural gas were found at 746 locations in Japanese coastal waters as a result of the latest sonic survey covering some 20,000 square kilometers. That comes on top of the 225 locations found during the last fiscal year that ended in March.

It is estimated that methane hydrate worth 100 years of supply of natural gas exists in Japanese coastal waters, and resource-poor Japan has been keen on exploring the reserves.

But technology to extract methane hydrate has yet to be fully developed. There are also concerns about environmental damage that could be caused by the process of extraction, partially because methane is a greenhouse gas.

http://bit.ly/146cKM9

When the doctor's away, the patient is more likely to survive

Deaths due to heart problems drop during major cardiology gatherings.

by John Timmer - Dec 26 2014, 11:30pm TST

"Don't get sick on a weekend." That advice is also part of a title of a research paper that evaluates the fates of patients who go through the emergency room on a weekend. These patients are more likely to die. It's just one of a number of studies that suggests patients who enter the hospital while the staffing is lower or the staff more relaxed end up with worse results.

But the precise cause of this enhanced weekend mortality has been hard to determine; is it the reduced staff, a more leisurely approach to care, or some other factor? To try to get at the cause, some researchers obtained records of heart patients who had a critical event during a time when hospitals were at full staff, but heart specialists were likely to be out of town. Unexpectedly, they found that the patients did significantly better when the relevant specialists were unavailable.

The study relied on medicare records to track patients that were admitted to a hospital with a serious heart condition: acute myocardial infarction, heart failure, or cardiac arrest. The key measure was simply whether the patient was still alive 30 days later.

That may sound simple, but the rest of the analysis was remarkably sophisticated. To figure out when heart specialists were most likely to be present at hospitals, they selected two large cardiology meetings: the American Heart Association and the American College of Cardiology, both of which attract over 10,000 participants. Patients admitted during the meetings were compared with groups admitted three weeks before and after. Reasoning that researchers are more likely to attend these meetings, they analyzed teaching hospitals separately from regular ones.

As additional controls, they checked a number of additional meetings for oncology, gastroenterology, and orthopedics specialists. They also looked at the impacts of additional critical injuries, like gastrointestinal bleeding and hip fractures, as well as non-critical cardiac problems.

In total, there were tens of thousands of patients involved. And the trends were clear. At teaching hospitals, the rate of death after heart failure was 24.8 percent

on non-meeting days. While the cardiologists were out of town, it dropped to 17 percent. A similar trend was apparent with cardiac arrests, where death rates fell from 68.6 percent to 59 percent while cardiology meetings were happening. There was no significant difference with acute myocardial infarction patients.

So, having specialists in town appeared to make matters worse for patients—the exact opposite of the hypothesis the researchers set out to examine. The various controls suggested the effect was robust, and it persisted after adjusting for other potential influences, like age and sex.

In a press release accompanying the report, one of its authors, Anupam Jena, said "That's a tremendous reduction in mortality, better than most of the medical interventions that exist to treat these conditions." What could possibly be causing it? The authors consider three possibilities. First, there's something involved with the changes in cardiology staffing that occur when specialists go out of town that actually increases care. The second is that there are fewer people having outpatient or same-day procedures, given that doctors wouldn't schedule these when they knew they'd be absent. This would allow the remaining physicians to better focus care on the serious cases.

The final possibility that they consider is that the doctors that remain behind are more cautious about the care they give, avoiding aggressive procedures such as the use of angioplasty or stents to re-open clogged heart vessels. This would be consistent with the lack of effect in acute myocardial infarction patients, where this procedure is used less often.

Although their analysis can't distinguish among these possibilities, it's clear that this effect warrants further attention. Both because it's possible that the long-term survival evens out thanks to more aggressive treatment, and because we might find out that we've been acting a bit too aggressively.

JAMA Internal Medicine, 2014. DOI: 10.1001/jamainternmed.2014.6781 .

http://bit.ly/1zIp8yO

An Ultrasonic Scalpel for Brain Surgery

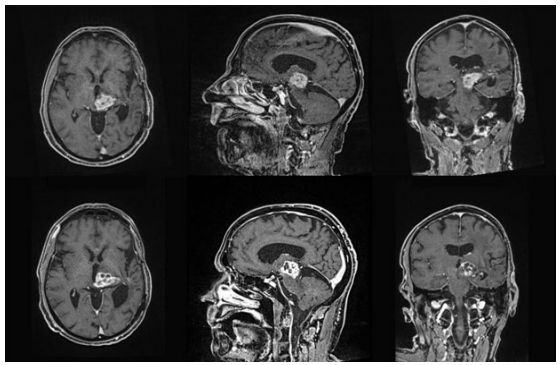
Focused ultrasound lets surgeons treat brain diseases without opening the skull

What if you could slice through the brain without removing any of the skull?

By Neel V. Patel

Brain surgery is fraught with huge risks and uncertainty. Parts of the skull (and sometimes most of it) need to be removed, a lengthy and harrowing procedure that could expose the brain to infection and almost always results in significant postoperative pain. Once the surgeon makes the first incision, the smallest error could have devastating consequences—seizure, loss of sensory or motor function, stroke, or even coma. But what if you could slice through the brain without removing any of the skull—create incisions inside the brain from the outside?

Through "transcranial focused ultrasound," physicians can now use high frequencies of ultrasound (typically from 650 to 710 kilohertz) to create discrete lesions in brain tissue without making direct physical cuts. Patients and doctors alike hope this could be a transformative tool for treating many different psychiatric and neurological disorders more easily and more effectively.



Images: Focused Ultrasound Foundation *Before and After:* ***Doctors focused ultrasound inside the brain of a patient with essential tremor, creating lesions [bottom row] deep within the brain [black spots inside white mass at center].***

In fact, focused ultrasound has already proved successful in treating patients with the condition known as essential tremor. Neurosurgeons at the University of Virginia's Focused Ultrasound Center, in Charlottesville, saw tremor rates fall by more than 50 percent in patients treated with ultrasound. The surgeons are currently finishing the final round of patient testing before seeking U.S. government approval as a prescribed treatment of essential tremor. The same clinical team is conducting trials for Parkinson's patients as well.

"Ultrasound is able to move through obstructions, like the skull," explains psychiatrist Alexander Bystritsky, of the University of California, Los Angeles. "It's noninvasive. You simply have to focus ultrasound much like you would focus a light."

Actually, it's a bit more complicated than that. Ordinarily ultrasound passes through body tissue without any effect. But in the same way that rays of sunlight can be focused by a magnifying glass to start a fire, beams of ultrasound can be focused to converge on a specific target, raising temperatures and destroying tissue. This three-dimensional "thermal ablation" can be anywhere between 1 and 15 millimeters in diameter, and it can be localized to a specific target deep in the brain without affecting the surrounding tissue. It's like performing surgery with a heat ray instead of a knife, but with the same precision and deep brain penetration—all without opening up a person's skull.

The initial problem with the transcranial delivery of ultrasound was that sound waves would lose focus and reflect off the skull. A section of skull would have to be removed to deliver the ultrasound deep into the brain. If part of the skull had to

be removed, there was little real advantage to ultrasound over conventional surgery.

Robert Dallapiazza, a neurosurgeon at the University of Virginia, says phased-array systems developed decades later meant that "instead of just two or three ultrasound sources, they started using 200, 500, and 1,000 elements, all focused into one discrete spot." Combined with a machine-mediated correction algorithm that informs doctors how the energy would be reflected or how its transmission would be altered through the skull, there is now no need to carve a window into the cranium.

Of the multitude of ultrasound applications being researched, one of the most promising is the treatment of motor disorders. Dallapiazza and his colleagues are currently researching the surgical applications of MRI-guided high-frequency focused ultrasound. A patient enters an MRI machine, and an ultrasound transducer is placed over his head. The whole setup, Dallapiazza says, "kind of looks like a hair dryer at a beauty salon." Within the MRI, low-energy ultrasound is applied to verify the machine's alignment with the targeted brain region. Amplitudes are then increased to generate a more intense beam that results in lesions. Patients are awake during the entire procedure and can usually feel the effects instantaneously. "They can really tell when they got the treatment," he says. "People are really brimming with excitement over focused ultrasound, because it doesn't require awake surgery or electrode implantation for potentially the rest of their lives."

Dallapiazza is optimistic about using ultrasound as a way to treat other diseases too, such as cancer. In March 2014, neurosurgeons at the University Children's - Hospital Zurich used ultrasound to treat a brain tumor for the first time. Much more research will be needed to assess the efficacy of such a procedure, but the surgery was a milestone nonetheless. "In the next five or 10 years, I think there are going to be a lot of breakthroughs of what we're actually able to do with this technology," says Dallapiazza. "It's an exciting time."

http://www.bbc.com/news/health-30551093

Whooping cough proteins evolving 'unusually' fast

Whooping cough may be evolving to outsmart the currently used vaccine, say researchers.

By Emma Wilkinson Health reporter, BBC News

Analysis of strains from 2012 shows the parts of the pertussis bacterium that the vaccine primes the immune system to recognise are changing. It may have "serious consequences" in future outbreaks, UK researchers state in the Journal of Infectious Diseases. But experts stressed the vaccine remains highly effective in protecting the most vulnerable young babies.

There has been a global resurgence of whooping cough in recent years.

In 2012, there were almost 10,000 confirmed cases in England and Wales - a dramatic increase from the last "peak" of 900 cases in 2008. The outbreak led to 14 deaths in babies under three months of age - the group who are most vulnerable to infection. Rising figures prompted health officials to recommend vaccination of pregnant women so immunity could be passed to their newborns - a strategy that a recent study showed was working well.

Evolving strains

But there has been much debate among experts about whether the introduction of a new vaccine in 2004 has been a factor in rising rates of whooping cough.

One issue is that immunity from the newer acellular vaccine - which contains specific proteins from the surface of the bacteria - does not seem to last as long as the previous whole cell version, leaving teenagers and adults lacking protection.

In the latest study, researchers analysed the genes coding for the proteins on the surface of the pertussis bacterium responsible for the UK outbreak.

They found proteins being targeted by the vaccine were mutating at a faster rate than other surface proteins not included in the vaccine. Potentially it means the bacteria is changing quickly to get around immune system's defences put in place with immunisation.

What is whooping cough?

It is also known as pertussis and is caused by a species of bacteria, Bordetella pertussis It mostly affects infants, who are at highest risk of complications and even death. The earliest signs are similar to a common cold, then develop into a cough and can even result in pneumonia Babies may turn blue while coughing due to a lack of oxygen.

The cough tends to come in short bursts followed by desperate gasps for air (the whooping noise) Adults can be infected - but the infection often goes unrecognised.

But the researchers are still trying to work out what the changes mean in reality - for example do the mutations boost the ability of the bacteria to cause infection.

"We wanted to look at strains from the UK to see if there was anything sudden that had occurred that had led to these really large outbreaks," said study leader Dr Andrew Preston from the University of Bath.

Vaccine effectiveness

The "million dollar question" he said was what, if anything, could be done to improve the vaccine - which is still the best defence we have - and prevent future outbreaks. Options to consider include adding more or different proteins to the vaccine, adding novel adjuvants - chemicals which boost the immune response, or even revisiting the old-style whole cell vaccine, he said. "Pertussis has a cyclical

nature and other big question is are we going to see another increase in late 2015," he added

Prof Adam Finn, a paediatric immunology expert at the University of Bristol said the importance - or not - of the subtle changes found in the study was as yet unclear. "But the control of pertussis is a significant worry," he added.

Only 60% of pregnant women have had the pertussis vaccine and we should be doing more to raise awareness of its benefits, he said.

"There is very good new evidence that vaccinating pregnant women protects their babies. And the group we really want to protect is newborn babies," he said.

http://nyti.ms/1AYVmVK

In a New Approach to Fighting Disease, Helpful Genetic Mutations Are Sought

Doug Whitney has a gene mutation that causes early onset Alzheimer's disease. He may also have one that protects him.

By GINA KOLATA DEC. 28, 2014

Doug Whitney should have died years ago. The 65-year-old resident of Port Orchard, Wash., has a devastating gene mutation that — according to the medical literature — causes early onset Alzheimer's disease in everyone who inherits it.

The mutation killed Mr. Whitney's mother and nine of her 13 siblings, and it killed Mr. Whitney's older brother. Every one of them began showing symptoms when they were in their 40s. Most died by their mid-50s. In the next generation, six cousins died of early onset Alzheimer's, and two others are in the final stages of the disease. One of his cousin's children also has Alzheimer's.

But Mr. Whitney has somehow escaped that fate. His memory is intact, and he has no signs of Alzheimer's disease. Researchers want to find out why. They suspect he has another gene mutation that somehow protects him from the horrific Alzheimer's gene mutation, or that at least substantially delays the disease's onset.

So Mr. Whitney has become Exhibit A in a new direction in genetics research. After years of looking for mutations that cause diseases, investigators are now searching for those that prevent them.

By understanding how protective mutations work, they hope to develop drugs that mimic them and protect everyone.

The new approach is turning genetics research on its head, said Eric E. Schadt, director of the Icahn Institute, a medical research institute at Mount Sinai in New York.

"Instead of trying to fix things that are broken let's look at people where things are broken but nature finds way around it," he explained.

In recent years, a few astounding protective gene mutations have been discovered, pretty much by accident. One prevents H.I.V. from entering cells and another enormously reduces the amount of LDL cholesterol, the dangerous kind, that people make. Both led to drugs. The AIDS drug is a mainstay of treatment, and the cholesterol drug is in the final stages of testing.

Researchers, using systematic searches of genetic databases, also found alterations in some genes that partially protect from diseases like heart disease, osteoporosis, Type 2 diabetes and Alzheimer's.

But now some are starting a more ambitious project — a search for mutations that provide complete protection.

It may sound obvious — why not look for people who have a genetic resistance to a disease? After all, everyone knows families that never seem to get common diseases like cancer or heart disease or osteoporosis. Genes might well be involved.

But the trick is to figure out if disease resistance is from a good gene mutation or a good environment or simply good luck, defying the odds when a disease is likely but not inevitable.

And if there is a good gene mutation involved, searching for it among the 20,000 human genes can be daunting. It is easier to find mutations that cause diseases — those appear to be many times more common.

It is only now, with fast and inexpensive methods of sequencing DNA and with massive and ever-growing databases of study subjects whose genomes have been sequenced, that it has become possible to seriously contemplate a search for rarer good genes.

The unprecedented effort has barely begun. One attempt, being led by Dr. Schadt and Dr. Stephen H. Friend, director of Sage Bionetworks, a nonprofit research organization based in Seattle, began because the two scientists had become frustrated with the failures of drug development.

Dr. Friend had worked in academia — M.I.T. and Harvard — then founded a biotechnology company, Rosetta Inpharmatics, and later helped run the cancer drug discovery effort at Merck. He began each new position feeling optimistic. More and more was being discovered about disease-causing genes. "I thought we should be able to develop drugs," he said.

But all too often disease-causing mutations destroy or disable genes, and drugs would have to restore what was lost, which can be difficult.

So Dr. Friend and Dr. Schadt decided to flip it around and search for a good gene mutation that counteracts the bad and — in an easier process — mimic that with a drug.

They gave their plan a name, The Resilience Project, and decided to search databases that held genetic and clinical information, looking for healthy people with mutations for fatal diseases that strike early in life. If the people had lived far

past the age when the disease should have appeared, they assumed they might have a lucky good gene mutation that blocked the bad.

Now, a year later, "we are in this interesting place between excited and frustrated," Dr. Friend says. They analyzed data from over 500,000 people and found 20 who seem to be protected from a fatal disease. But because of privacy issues there were no names attached to the data.

Four of the subjects are in China. Dr. Schadt and Dr. Friend are trying to find a way to contact them, but "it is very difficult," Dr. Schadt said.

Dr. Friend and Dr. Schadt are now looking at other databases that might make it easier to contact subjects, but also decided they need to try different approaches. One will be to simply ask healthy people to let them sequence their DNA, putting out the word that they are looking for volunteers, perhaps hundreds of thousands of them. People who agreed would be contacted only if they appeared to be protected from a fatal disease.

Another is to contact researchers studying extended families with a severe genetic disease to see if they came across anyone who seemed protected. That approach appeared to be a long shot — the number of people in such studies is limited, and if there had been anyone who was protected, wouldn't the researchers have noticed and published their story?

But when they contacted researchers at Washington University, who were studying families with a gene, presenilin, that causes early Alzheimer's, they discovered Doug Whitney.

He certainly is unusual, researchers agree. He could, of course, still get Alzheimer's, but it would have been substantially delayed.

Mr. Whitney had been waiting for Alzheimer's symptoms, starting when he turned 40. He knew he had a 50-50 chance of inheriting the Alzheimer's mutation. But year after year went by and nothing happened.

In 2011, he joined a study at Washington University in St. Louis, led by Dr. Randall Bateman, that recruited people from families with an early onset Alzheimer's gene mutation. Mr. Whitney had finally concluded he did not have the gene mutation — he was 61, after all, and his memory and thinking were fine. On May 31, 2011, his 62nd birthday, he decided to have the genetic test. The result came back the next month. He had the gene.

But now everything has changed again. Dr. Bateman is studying Mr. Whitney. So too is Dr. Thomas Bird, a neurogeneticist at the University of Washington. Dr. Friend and Dr. Schadt have contacted him too.

Mr. Whitney is happy to help. He has just retired and when people ask what he will do now, he has a new reply: "My job is to help them figure out Alzheimer's. I will do what I can to make it happen."

http://bit.ly/1xtuDkZ

Tokyo man in his 30s being tested for Ebola

Health Ministry officials in Japan said Monday that a Japanese man was being tested for the deadly Ebola virus after he developed a fever with a temperature of over 38 degrees.

TOKYO - The man was in the West African country of Sierra Leone which has been hard hit by the Ebola epidemic. He is currently being tested at the National Center for Global Health and Medicine in Tokyo's Shinjuku Ward.

NTV reported that he returned to Japan on Dec 23 after spending eight days in Sierra Leone.

Health Ministry official Kensuke Nakajima told a news conference that the man had been advised to stay home after he arrived back in Japan, NTV reported.

The man told ministry officials that to the best of his knowledge, he did not come into contact with any Ebola sufferers in Sierra Leone but said that he did touch a bag containing the body of an Ebola victim prior to a funeral on Dec 17.

Nakajima declined to say why the man was in Sierra Leone or what his job is, but said he was not a health care worker, NTV reported.

Health ministry officials said the virus may not have been detected when the man arrived back in Japan on Dec 23 due to its potentially immature state.