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Tiny Ocean Plants Geoengineer Brighter Clouds

Marine life seems to create a reflective sunshade above the Southern Ocean

By David Biello | July 20, 2015

The Southern Ocean has some of the thickest clouds on Earth, made brighter in the summertime by marine microbes living in the waters below, according to new research that combines satellite observations and computer modeling. In fact, bacteria and plankton drifting in the ocean produce particles that get whipped up into atmosphere where they seed cloud droplets, and in turn, the brighter clouds reflect more sunlight away from Earth.

"Life in the ocean increases the brightness of clouds and keeps the climate cooler than it would otherwise be," says Dennis Hartmann, an atmospheric scientist at the University of Washington (U.W.) who helped oversee the research published July 17 in the open-access journal Science Advances. "I was a little surprised how big an effect life has on cloud albedo in pristine areas of the oceans."

The more liquid that is suspended in a cloud, the more reflective it is, and ocean life seems to roughly double the number of water droplets forming in the sky, according to the study. It suggests that the brighter clouds reflect as much as 10 watts per square meter of sunshine in summer, which averages out to a reduction of four watts per square meter reaching the surface annually.

That's about as much sunlight as particulate matter from pollution reflects in other parts of the globe.

The Southern Ocean is a forbidding place, especially to sailors, wracked by persistent winds and extreme weather. A current encircles Antarctica, locking it in deep freeze; it casts off whorls and eddies that swirl and mix vital nutrients in the seawater. When the sunlight shines on the region from spring until fall, an array of photosynthetic life-forms bloom in the water.

"The return of light in the summer ignites an amazing flurry of activity in phytoplankton," says atmospheric scientist Daniel McCoy of U.W., who co-led the research with fellow atmospheric scientist Susannah Burrows of the Pacific Northwest National Laboratory.

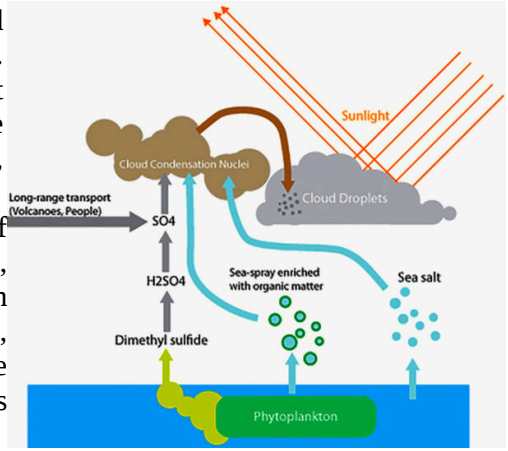
Some bacteria and plankton pump out gases like dimethyl sulfide—the compound responsible for the unique smell of the sea—while also covering the surface of the ocean in an oily profusion of cells. Just like the salt in seawater, wind and waves loft these gases and plant and animal bits into the sky. Those particles—known as aerosols to scientists—become the centers for condensation of vapor in the atmosphere into the droplets that form clouds.

In the Southern Ocean sea salt provides a constant source of cloudiness year-round, but in summer the marine microbes seem to enhance the process.

The more droplets, the brighter a cloud and the better it is at reflecting sunlight. The view from satellites shows that regions with more phytoplankton have clouds with more, smaller droplets, McCoy notes.

Marine blooms increase the number of cloud droplets by as much as 60 percent, and the brighter clouds come just when the most sunlight can be reflected, boosting the effect further. "Life in the ocean directly influences the brightness of clouds," Hartmann says.

clouds-over-southern-ocean-from-space



Courtesy of Daniel McCoy

Some people want to harness this process by spraying more droplets or aerosols into the air to create clouds and mitigate climate change—a geoengineering technique known as marine cloud brightening. But it appears that microbes may have got there first with a form of biological geoengineering.

At the same time, it remains unclear exactly how the biological aerosols change the chemistry of the atmosphere or whether they also boost the extent of cloud cover over the Southern Ocean. Understanding these factors might help elucidate exactly how sensitive Earth's climate might be to rising atmospheric concentrations of greenhouse gases like carbon dioxide, thanks to humanity's pyromania for fossil fuels.

Nor is it clear that phytoplankton bloom or bust in response to cloudiness, forming a potential route for life to regulate climate, which has been suggested by some scientists.

The correlation between marine life and clouds may also prove spurious, Burrows notes, but it is hard to explain the match between blooms and brighter clouds any other way. "Higher cloud droplet numbers occur over phytoplankton blooms, and we currently are unable to explain this phenomenon without invoking an increase in the [aerosols] near these blooms," she says.

"Life brightens the clouds, but we don't know how this might change as the ocean warms up," Hartmann adds. "We don't know if life will produce more or fewer cloud droplets in a warmed Earth." In other words, the tiny geoengineers may help human civilization with the global warming problem—or not.

http://www.eurekalert.org/pub_releases/2015-07/sumc-sss072015.php

Stanford scientists see iron-containing inflammatory cells in Alzheimer's brains

Iron-containing microglia are found in a particular part of the hippocampus

Examining post-mortem tissue from the brains of people with Alzheimer's disease, Stanford University School of Medicine investigators identified what appear to be iron-containing microglia -- specialized scavenger cells that sometimes become inflammatory -- in a particular part of the hippocampus, a key brain structure whose integrity is critical to memory formation. In post-mortem brain tissue from people not diagnosed with Alzheimer's, neither the iron deposits nor the scavenger cells engulfing them were present in that brain region.

The findings, recounted in a study now available online in Neurobiology of Aging, suggest that high-field magnetic resonance imaging, in particular an advanced version called 7T MRI that uses a powerful 7-Tesla magnet, could someday be used to diagnose and monitor Alzheimer's patients earlier than is currently possible.

The findings also add a new suspect to the Alzheimer's disease lineup. A long-held hypothesis holds that the most notorious feature of Alzheimer's disease, amyloid plaques, is the main cause of the disorder. These plaques are extracellular aggregations of a small protein called beta-amyloid that are prominent in diseased patients' brains, as well as in mouse models of the disease. The other most cited key player is tau, another Alzheimer's-associated protein that abnormally aggregates into threadlike tangles inside nerve cells. Surprisingly, in the brain region of interest there was no consistent overlap between the iron-laden microglia and the amyloid plaques or tau.

"Microglia are the brain's immune cells," said Michael Zeineh, MD, PhD, assistant professor of neuroradiology and the study's lead author. In their resting state, they're like police officers in the doughnut shop, sitting down and relaxing, their guns holstered, but keeping their eyes open while placidly munching on whatever cellular debris or stray substances might come their way. If they encounter anything suspicious, though, they whirl into action. Activated microglia are like officers with their guns out and firing, Zeineh said.

Microglia inflamed

The bulk of microglia found in association with iron in the study were in an activated, inflammatory state. Alzheimer's is increasingly understood to involve brain inflammation, and groups led by Stanford researchers such as neurologists Katrin Andreasson, MD, and Tony Wyss-Coray, PhD, and neurobiologist Ben Barres, MD, PhD, have previously fingered microglia as potential suspects in the early inflammatory pathology of the disease. This study adds the new finding that inflamed, iron-associated microglia are present in the hippocampus in Alzheimer's and are observable by 7T MRI, which could advance the scientific community's understanding of the disease.

The researchers noted that this was a preliminary study performed on a small number of human brain specimens, which are generally difficult to obtain. In this case, the specimens were supplied by the study's senior author, Brian Rutt, PhD, professor of radiology.

"Some imaging studies using mouse models of Alzheimer's disease had revealed the presence in these mice's brains of tiny, mysterious black dots that could signal the presence of iron, an element that shows up dark under MRI and, in certain chemical forms, can be highly reactive and inflammation-inducing," Rutt said. These mouse studies had raised the possibility that this iron might be tightly associated with amyloid plaques. Rutt teamed up with Zeineh to scrutinize the human brain specimens for iron particles. "We wanted to see if there was an association of iron with Alzheimer's plaques in humans," Rutt said.

In a series of steps combining 7T MRI, computational analysis and painstaking laboratory staining techniques, the scientists probed slabs of tissue taken from several places within the brains of each of five Alzheimer's and five control brain specimens. "We weren't sure where to look," Rutt said.

These slabs were scanned via 7T MRI, which can provide hair's-thickness resolution in three dimensions. In images from four out of the five Alzheimer's brains -- but in none of the control brains -- the researchers observed black dots in the subiculum, a component of the hippocampus. The hippocampus is known to incur some of the earliest and most severe ravages of Alzheimer's disease.

The Stanford scientists then carefully sectioned the tissue slabs into several hundred ultrathin sections; incubated those sections with stains that pinpoint the location of iron, microglia, amyloid plaques and tau; and analyzed the resulting stain patterns.

Amyloid plaque, tau not consistently near iron

What emerged was evidence that iron, frequently engulfed by microglia, was occupying the same spots in the subiculum of Alzheimer's brains where 7T MRI had found black dots. Those microglia were mostly in an activated state.

As notable was the relative absence of amyloid plaques in these spots. "We didn't consistently find the iron associated with plaques as we were expecting, despite our best efforts to do that," said Rutt. Tau was more often nearby -- but, again, not consistently.

"Amyloid is found all over the brain in Alzheimer's disease, and often in the brains of people who've died with no complaints of memory loss at all," Zeineh

said. "Tau is also found throughout the Alzheimer's brain. This iron-microglia complex, in contrast, really seems concentrated in the subiculum -- and, so far, it's showing up only in brains from Alzheimer's patients."

Zeineh and Rutt said they don't know how the iron gets into brain tissue, or why it accumulates where it does. Micro-injury to small cerebral blood vessels there was one possibility, they speculated.

The researchers cautioned that the stains used in the study wouldn't have been able to visualize soluble clusters of beta-amyloid, now increasingly believed to be the protein's toxic form, as opposed to the aggregated plaques. Soluble amyloid may yet be playing a major if still poorly understood role, they said.

Zeineh, Rutt and Hannes Vogel, MD, a professor of pathology and co-author of the study, plan to explore these findings further in collaboration with Edward Plowey, MD, PhD, assistant professor of pathology. They intend to examine more wide-ranging areas of the brain and to stain for more cell types within larger numbers of post-mortem brain specimens. They also plan to hunt for iron-filled microglia in the brains of living patients during the early stages of neurodegeneration and memory loss that precede the onset of Alzheimer's disease. Their ultimate goal is to translate these imaging findings into clinical tools to help in the fight against dementia.

The study was carried out in collaboration with researchers in Canada and Germany. It was funded by the Radiological Society of North America and by General Electric Healthcare.

http://www.eurekalert.org/pub_releases/2015-07/icl-ffe072015.php

Fossil fuel emissions will complicate radiocarbon dating, warns scientist

Fossil fuel emissions could soon make it impossible for radiocarbon dating to distinguish new materials from artefacts that are hundreds of years old.

Carbon released by burning fossil fuels is diluting radioactive carbon-14 and artificially raising the radiocarbon 'age' of the atmosphere, according to a paper published today (Monday 20 July 2015) in the journal PNAS.

Radiocarbon measurements have a range of uses, from analysing archaeological finds, to detecting fraudulent works of art, to identifying illegal ivory trading, to assessing the regeneration of brain cells in neurological patients.

The new study suggests that some of these current uses will be affected over this century, depending on how much fossil fuel emissions increase or decrease.

"If we reduced fossil fuel emissions, it would be good news for radiocarbon dating," said the study's author, Dr Heather Graven from the Department of Physics and the Grantham Institute - Climate Change and Environment at Imperial College London.

Carbon-14 is a rare, but naturally occurring, radioactive type of carbon that decays over thousands of years. Radiocarbon dating works by measuring how much the fraction of carbon-14 versus non-radioactive carbon in an object has changed and therefore how long the object has been around.

Fossil fuels like coal and oil are so old that they contain no carbon-14. When their emissions mix with the modern atmosphere, they flood it with non-radioactive carbon. In radiocarbon dating terms this makes the atmosphere appear older, which is reflected in the tissues of plants taking in CO2 during photosynthesis, and their products such as cottons.

At the rate fossil fuel emissions are currently increasing, by 2050 a new T-shirt would have the same radiocarbon date as a robe worn by William the Conqueror a thousand years earlier.

If fossil fuel emissions were rapidly curbed, the new t-shirt would only have the same radiocarbon age as something 100 years old, according to the study.

The fraction of carbon-14 in the atmosphere decreased after the Industrial Revolution with the rise of fossil fuel combustion. But in the 1950s and 60s, nuclear weapons testing caused a sharp increase. Since then atmospheric observations show the levels have been dropping, and are now close to the pre-industrial proportions.

The new study indicates that by 2020, the fraction of carbon-14 could drop to such an extent that radiocarbon dating will start to be affected. "We can see from atmospheric observations that radiocarbon levels are steadily decreasing. How low they go depends on changes in our fossil fuel emissions," said Dr Graven.

http://www.eurekalert.org/pub_releases/2015-07/du-gwp072015.php

Gut worms protect babies' brains from inflammation

Mom's parasites could help protect her baby's brain

DURHAM, N.C. - A Duke University study in rats finds that gut worms can protect babies' brains from long-term learning and memory problems caused by newborn infections. Baby rats with tapeworms avoided the brain inflammation that plagued worm-free rats after exposure to immune triggers in adulthood.

What's more, the benefits began early, while still in the womb. Expectant mother rats with tapeworms passed similar protection on to their worm-free pups, the researchers found. The findings could point to new ways to treat or prevent the chronic brain inflammation linked to cognitive disorders like Alzheimer's disease, autism and depression. The study appears online in the journal Brain, Behavior, and Immunity.

Previous studies by Duke neuroscientist Staci Bilbo and colleagues showed that when rats get bacterial infections at a very early age, even elsewhere in the body, immune cells in their brains become hypersensitive to subsequent infections and

pump out a continuous stream of messenger molecules called cytokines that can cause cognitive problems later in life.

But for Bilbo, who is an associate professor of psychology and neuroscience and a member of the Duke Institute for Brain Sciences, something didn't quite add up. Given how frequently bacterial infections strike, it was still unclear why a single infection at the wrong time would send the brain's immune cells into permanent overdrive.

"We have faced bacterial infections throughout our entire evolutionary history, presumably also during the neonatal period," Bilbo said. "It always seemed kind of strange that the immune system would have evolved to overreact like that."

That got Bilbo thinking. "Maybe this isn't how the immune system evolved to work," she said.

According to what scientists call the "Biome Depletion Theory," some autoimmune and inflammation-related diseases may be the result of too few of the life forms that once lived in and on the body -- particularly gut worms -- rather than too many. Tapeworms, roundworms and other wormy companions have inhabited the warm wet folds of animal intestines for more than 100 million years, bathing in a constant supply of food and nutrients.

Over millions of years of co-existence, the theory goes, the immune system learned to tolerate these live-in guests, and eventually adapted to work with worms in mind. The theory is that now, with worms gone from our guts, the body's natural defenses can spiral out of control. "Our bodies are essentially an ecosystem," said Duke immunologist and study co-author William Parker.

Parker and Bilbo decided to see if restoring the internal ecosystem in the gut could bring the brain's immune cells back in balance.

Laboratory rats are ideal for testing the idea, Parker said, because the life of a lab rat is a remarkably clean one. Scientists started breeding strains of rodents for laboratory experiments about 150 years ago. These animals are housed exclusively indoors, where their cages and bedding are regularly disinfected. A series of pumps and fans change the cage air more than a dozen times an hour. They eat processed food and sip treated water, and take deworming drugs and antibiotics to keep them free of parasites and pathogens.

"In a real sense we've done the same things to our lab animals that we've inadvertently done to ourselves," Parker said. The researchers did a first set of experiments comparing worm-free lab rats with rats that were raised on a farm where they were exposed to worms. When they infected the rats with bacteria, they found that the farm-raised rats avoided the damaging overproduction of cytokine proteins linked to cognitive decline later in life.

"We didn't see the same hyper immune response in the brain," Bilbo said.

Next, the researchers studied two groups of rats in the lab. One group consisted of typical lab rats whose guts were worm-free. The other group was identical in diet, housing, exercise and genetics to the first, except they -- and their parents before them -- were deliberately given tapeworms.

Both groups were injected with E. coli bacteria when the rats were newborns. Once the pups reached adulthood they were given a second injection, this time with a chemical from the cell walls of bacteria known to spring the immune system into action. The researchers then monitored changes in the rats' brains and behavior to see how they responded to the one-two punch.

The worm-free rats responded to the second immune challenge with the same harmful outpouring of inflammatory cytokines seen in previous studies. But the wormy rats, and also rats that were worm-free but born to worm-infested parents, responded differently. Notably, the immune cells in their brains were able to respond to the second trigger without going into overdrive. They also didn't develop the same memory problems later in life that their worm-free counterparts did.

Next, the researchers hope to figure out whether before or sometime after birth is the optimal time for treatment. "Pregnancy is such an interesting time for the immune system," Bilbo said. "Maybe that's why it worked so well. We just don't know yet."

Duke researchers Lauren Williamson, Erin McKenney, Zoie Holzknecht, Christine Belliveau, John Rawls and Susan Poulton were also authors of this study. This research received support from the Coalition for SafeMinds.

CITATION: "Got Worms? Perinatal Exposure to Helminths Prevents Persistent Immune Sensitization and Cognitive Dysfunction Induced by Early-Life Infection," L. Williamson et al. Brain, Behavior, and Immunity, July 2015. DOI: 10.1016/j.bbi.2015.07.006

http://www.eurekalert.org/pub_releases/2015-07/asu-aun072015.php

Archaeologists use new methods to explore move from hunting, gathering to farming

One of the enduring mysteries of the human experience is how and why humans moved from hunting and gathering to farming.

From their beginnings humans, like other mammals, depended on wild resources for sustenance. Then between 8,000 and 12,000 years ago, in a transitional event known as the Neolithic Revolution, they began to create and tend domestic ecosystems in various locations around the world, and agriculture was born.

Despite decades of research into this major human advancement, scientists still don't know what propelled it. The recent work of a research team led by Arizona State University postdoc Isaac Ullah narrows the mystery by showing what variables might have affected the transition.

Ullah is an archaeologist in the School of Human Evolution and Social Change in the College of Liberal Arts and Sciences. Most of his research uses dynamical systems theory (DST) and centers on understanding the ways in which human societies changed with the advent of plant and animal domestication.

His latest research project, undertaken with Ian Kuijt of the University of Notre Dame and Jacob Freeman of Utah State University and published this week in the Proceedings of the National Academy of Sciences, combined the field of DST with existing research on the origins of plant and animal domestication.

For Ullah, DST provided a way around the obstacles researchers have historically faced in defining the origins of domestication: the transition occurred long ago; much of the evidence for the impetus for the transition was not preserved in the archaeological record; and the transition didn't occur everywhere or all at once and seems to have been quite different -- involving different crops and animals -- in the places where it did occur.

Ullah's team approached the ethnographic record of human subsistence from the perspective that human subsistence systems are complex adaptive systems, or systems composed of many interrelated parts that react to and interact with their environment.

"We used ideas generated from observations of other dynamical systems - both in the real world and in computer simulations - to create hypotheses about the way data about human subsistence ought to pattern when subjected to specific statistical analyses," Ullah says.

The main phenomena they hoped to find were 'attractors' and 'repellers.' In DST, an attractor is a combination of variable states that is relatively stable over time, whereas a repeller is a combination of variable states that is not. "In other words," Ullah explains, "DST tells us that there ought to be some combinations of subsistence behaviors and environmental characteristics that are generally stable and some that aren't."

He says that when the researchers initially conducted the analysis, they weren't sure if attractors and repellers would be observable, but from early on, they saw interesting clusterings of societies that suggested the attractor/repeller phenomenon.

What was even more interesting to the team was that they began to see that the clustering was largely controlled by a small number of important variables, such as resource density, mobility and population size.

The team discovered that changes in these variables brought some attractors closer together, created new ones or eliminated others.

That showed them that even though the general possibilities for human subsistence is largely governed by a small number of highly important variables, moving from one subsistence attractor to another is more possible under some socio-environmental conditions than others.

"It is this specific insight that may help to explain why the transition to food production happened in some times and places but not in others, why it happened so differently in all these places and at different times and rates," Ullah states.

http://www.bbc.com/news/health-33569161

Poor sleeping patterns link to cancer

Irregular sleeping patterns have been "unequivocally" shown to lead to cancer in tests on mice, a study suggests.

By James Gallagher Health editor, BBC News website

The report, in Current Biology, lends weight to concerns about the damaging impact of shift work on health. The researchers said women with a family risk of breast cancer should never work shifts, but cautioned that further tests in people were needed. The data also indicated the animals were 20% heavier despite eating the same amount of food.

Studies in people have often suggested a higher risk of diseases such as breast cancer in shift workers and flight attendants. One argument is disrupting the body's internal rhythm - or body clock - increases the risk of disease.

However, the link is uncertain because the type of person who works shifts may also be more likely to develop cancer due to factors such as social class, activity levels or the amount of vitamin D they get.

Mice prone to developing breast cancer had their body clock delayed by 12 hours every week for a year. Normally they had tumours after 50 weeks - but with regular disruption to their sleeping patterns, the tumours appeared eight weeks earlier. The report said: "This is the first study that unequivocally shows a link between chronic light-dark inversions and breast cancer development."

Interpreting the consequences for humans is fraught with difficulty, but the researchers guesstimated the equivalent effect could be an extra 10kg (1st 8lb) of body weight or for at-risk women getting cancer about five years earlier.

'Definitive experimental proof'

"If you had a situation where a family is at risk for breast cancer, I would certainly advise those people not to work as a flight attendant or to do shift work," one of the researchers, Gijsbetus van der Horst, from the Erasmus University Medical Centre, in the Netherlands, said.

Dr Michael Hastings, from the UK's Medical Research Council, told the BBC: "I consider this study to give the definitive experimental proof, in mouse models, that circadian [body clock] disruption can accelerate the development of breast cancer. The general public health message coming out of my area of work is shift work, particularly rotational shift work is a stress and therefore it has

consequences."There are things people should be looking out for - pay more attention to your body weight, pay more attention to inspecting breasts, and employers should offer more in-work health checks. "If we're going to do it, then let's keep an eye on people and inform them."

http://www.eurekalert.org/pub_releases/2015-07/mali-bib072115.php

Biomarkers in blood shown to be highly selective indicators of brain damage

Caused by traumatic brain injury

New Rochelle, NY, - Researchers have shown that the levels of two proteins present in blood and cerebrospinal fluid increase significantly at different time points following traumatic brain injury (TBI), confirming their potential value as biomarkers of trauma-related brain damage.

The researchers linked the changes in circulating UCH-L1 and GFAP proteins in rats to brain tissue damage and neuronal degeneration seen on examination of the rat brains and present their findings in an article published in Journal of Neurotrauma, a peer-reviewed journal from Mary Ann Liebert, Inc., publishers. The article is available free on the Journal of Neurotrauma website.

Xian-jian Huang and coauthors, Shenzhen University 1st Affiliated Hospital (China), University of California at Davis, Banyan Biomarkers, Inc. (Alachua, FL), and University of Messina (Italy), measured the levels of ubiquitin carboxy-terminal hydrolase L1 (UCH-L1), a protein specific to neurons, and glial fibrillary acidic protein (GFAP), a brain-specific protein made mainly by astrocytes in the blood and cerebral spinal fluid of rats that did and did not experience TBI. Measurements taken 2 days before injury and at 3, 6, and 24 hours after TBI showed significant differences in UCH-L1 and GFAP levels at different timepoints in injured versus non-injured animals. The correlation between increased protein levels and direct evidence of brain damage makes these promising biomarkers for assessing brain injury following TBI.

The authors describe their methods and results in the article "Acute Temporal Profiles of Serum Levels of UCH-L1 and GFAP and Relationships to Neuronal and Astroglial Pathology following Traumatic Brain Injury in Rats").

"These studies are important not only from the basic science but also the clinical perspective," says John T. Povlishock, PhD, Editor-in-Chief of Journal of Neurotrauma and Professor, Medical College of Virginia Campus of Virginia Commonwealth University, Richmond. "The studies confirm the importance of GFAP as well as UCH-L1 as biomarkers for the detection of the consequences of TBI, particularly as they relate to neuronal and glial perturbation. The nice coupling of biomarker evaluation and histological examination demonstrates that these biomarkers derive from damaged glial and neuronal elements rather than a generalized cellular upregulation of these proteins. The implications of these studies for future clinical and basic science discovery are profound."

http://www.eurekalert.org/pub_releases/2015-07/nki-spa072115.php

Specific protein as missing link for earliest known change in Alzheimer's pathology

Findings may influence strategies for treatment

NEW YORK - A recent study conducted at Nathan S. Kline Institute for Psychiatric Research (NKI) and NYU Langone Medical Center implicates a new culprit in Alzheimer's disease development. The research reveals that ßCTF -- the precursor of the amyloid beta (Aß) peptide -- acts at the earliest stage of Alzheimer's to initiate a range of abnormalities leading to the loss of groups of neurons critical for memory formation. Results from the study are published online July 21, 2015 in the journal, Molecular Psychiatry, and the article has been selected for an issue cover.

The recent study findings involving βCTF have significant implications for treatment strategies and furthering the course of Alzheimer's drug development. Presently, the most common strategy for treating Alzheimer's disease is targeting the amyloid ß peptide, which has had modest success in clinical trials. Findings from this research suggest that drugs that may reduce βCTF levels as well as beta-amyloid, such as the class of BACE1 inhibitors currently under development, may help slow or stop the progression of Alzheimer's disease.

βCTF is formed during endocytosis, the process by which cells absorb nutrients and sample various materials from the outside environment. It has been known for some time that abnormalities of endocytosis develop very early in Alzheimer's disease, well before clinical symptoms, and that variant forms of genes controlling endocytosis are frequently implicated as risk factors promoting Alzheimer's. Endosomes -- the membranous vesicles mediating endocytosis -- start to swell abnormally in some neurons beginning even in infancy in Down syndrome - a developmental disability that almost invariably leads to early-onset AD. Research indicates that more than 75 percent of those with Down's, aged 65 and older, have Alzheimer's disease.

The NYU Langone - NKI research team led by Ralph Nixon, MD, PhD, professor in the departments of psychiatry and cell biology at NYU Langone School of Medicine and director of the Center for Dementia Research at the Nathan S. Kline Institute for Psychiatric Research found that, in Alzheimer's and Down Syndrome, βCTF forms more rapidly on endosomes triggering a molecular pathway leading to loss of neurons involved with memory. The researchers discovered APPL1, a

protein unrelated to amyloid precursor protein (APP) despite its similar acronym, directly links βCTF to a second protein, rab5, known to activate the molecular chain of events leading to neurodegeneration. Lowering APPL1 levels in cells of individuals with Down syndrome abolished the abnormal endocytosis, indicating the vital role of APPL1 in this molecular cascade. The identification of APPL1 as the missing link in a well-described chain of events associated with very early Alzheimer pathology implies a direct contribution of ßCTF to Alzheimer's disease development. Notably, a recently discovered APP mutation that uniquely lowers, rather than raising, risk for Alzheimer's is believed to act by slowing the formation of ßCTF.

While the current findings do not place any more or less importance to Aß as a culprit and a target for Alzheimer's therapy, they now underscore the importance of ßCTF as a key contributor to disease development. "It will be important to consider the role of βCTF in the design of future therapies for Alzheimer's disease and in the interpretation of current clinical trials of BACE1 inhibitors. BACE1 inhibitor trials have been considered a test of the Aß/amyloid hypothesis but the primary action of these inhibitors is actually to block formation of ßCTF, the precursor of Aß," said Ralph A. Nixon, MD, PhD.

These findings are a result of an eight year investigative track funded by the National Institute on Aging. In addition to Ralph Nixon, Seonil Kim, PhD played the major role in the study, in collaboration with other NKI and NYU researchers Yutaka Sato PhD, Panaiyur S. Mohan, PhD, Corrinne Peterhoff, Anna Pensalfini, MS, PhD, Andrew Rigoglioso, and Ying Jiang, PhD.

http://www.eurekalert.org/pub_releases/2015-07/uom-bvc072115.php

Blood vessels can actually get better with age

Study finds that arteries adapt to oxidative stress caused by aging

Columbia, Mo. -- Although the causes of many age-related diseases remain unknown, oxidative stress is thought to be the main culprit. Oxidative stress has been linked to cardiovascular and neurodegenerative diseases including diabetes, hypertension and age-related cancers. However, researchers at the University of Missouri recently found that aging actually offered significant protection against oxidative stress. These findings suggest that aging may trigger an adaptive response to counteract the effects of oxidative stress on blood vessels.

"Molecules known as reactive oxygen species, or ROS, play an important role in regulating cellular function," said Steven Segal, a professor of medical pharmacology and physiology at the MU School of Medicine and senior author of the study. "However, the overproduction of ROS can help create a condition referred to as oxidative stress, which can alter the function of cells and interfere with their growth and reproduction."

To understand the effects of aging on the function of blood vessels when they are exposed to oxidative stress, Segal's team studied the inner lining, or endothelium, of small resistance arteries. Resistance arteries are important to cardiovascular function because they regulate both the amount of blood flow into tissues and systemic blood pressure.

"We studied the endothelium from resistance arteries of male mice at 4 months and 24 months of age, which correspond to humans in their early 20s and mid-60s," Segal said. "We first studied the endothelium under resting conditions and in the absence of oxidative stress. We then simulated oxidative stress by adding hydrogen peroxide. When oxidative stress was induced for 20 minutes, the endothelial cells of the younger mice had abnormal increases in calcium when compared to the endothelial cells of the older mice. This finding is important because when calcium gets too high, cells can be severely damaged."

When oxidative stress was extended to 60 minutes, Segal's team found that the death of endothelial cells in the younger mice was seven times greater than those from the older mice. These findings indicated that with advancing age, the endothelium had adapted to preserve cellular integrity when confronted with oxidative stress.

"The most surprising thing we found is that the endothelium was much less perturbed by oxidative stress during advanced age when compared to younger age," Segal said. "This finding contrasts with the generally held belief that the functional integrity of the endothelium is compromised as we age. Our study suggests that blood vessels adapt during the aging process to regulate ROS and minimize cell death when subjected to an abrupt increase in oxidative stress. This adaptation helps to ensure that the arteries of older individuals can still do their jobs."

"Although more studies are needed to identify the mechanism by which the endothelium adapts to advanced age, our study provides evidence that the natural tendency of the body is to adapt to oxidative stress during healthy aging," Segal said.

The study, "Advanced Age Protects Microvascular Endothelium from Aberrant Ca2+ Influx and Cell Death Induced by Hydrogen Peroxide," recently was published in The Physiological Society's Journal of Physiology. In addition to Segal, the research team included Matthew Socha, Erika Boerman, Erik Behringer, Rebecca Shaw and Timothy Domeier -- all with the MU School of Medicine's Department of Medical Pharmacology and Physiology. Funding for the study was provided by the National Institutes of Health (R37-HL041026, R01-HL086483, F32-HL107050, F32-HL118836, K99-AG047198 and K01-AG041208). The content of the article is solely the responsibility of the authors and does not necessarily represent the official views of the funding agencies.

http://www.eurekalert.org/pub_releases/2015-07/b-dam072015.php

Doctors and medical students in India should stop wearing white coats

They harbor infection and should be banned, argues doctor

Doctors and medical students in India should stop wearing white coats, argues a doctor in The BMJ this week. Edmond Fernandes, a postgraduate at Yenepoya Medical College in Mangalore, says evidence shows that long sleeved coats spread infection and lead to avoidable harm and cost to patients.

Although long sleeved white coats have traditionally been worn by doctors since the 19th century, we now know that white coats "harbour potential contaminants and contribute considerably to the burden of disease acquired in hospital by spreading infection," writes Fernandes.

He explains that in India, changing areas in hospitals are rare because of space constraints, so white coats are commonly worn by students coming from college and outside the hospital.

They are also often left on chairs, tables, and in corridors.

In many cities in India some junior doctors are also now seen wearing white coats in shopping malls and cinemas too, and then they enter sterile zones in the hospital in the same attire, he adds.

"Given India's tropical climate, common sense indicates that we should discourage wearing white coats that are washed perhaps only every few weeks," he suggests.

He points out that in 2007, the United Kingdom took the landmark decision to ban long sleeved white coats - and that in 2009, the American Medical Association wanted to follow suit and dump the white coats, "but the proposal was dismissed because clinicians wanted to keep their traditional gowns."

Some may argue that white coats are a badge of honour, says Fernandes, "but they are mere symbolism and wearing them does not itself confer status or professionalism." He believes that "dressing presentably and sporting a smile are more important than white coats" and that institutions "should give every medical student and doctor a recognisable name badge to wear."

And he points out that we can do other things to reduce hospital acquired infections, such as better hand washing compliance.

"Every hospital should have a committee to check and respond to hospital acquired infections," he says. "But an easy win would be for India's ministry of health to ban doctors and medical students from wearing white coats, to reduce the harm and cost that results from hospital acquired infections."

http://www.eurekalert.org/pub_releases/2015-07/asfm-ufv071715.php

Universal flu vaccine in the works

New study points to possibility of creating a 'universal' vaccine that can provide broad protection against numerous influenza strains

WASHINGTON, DC - Each year, scientists create an influenza (flu) vaccine that protects against a few specific influenza strains that researchers predict are going to be the most common during that year. Now, a new study shows that scientists may be able to create a 'universal' vaccine that can provide broad protection against numerous influenza strains, including those that could cause future pandemics. The study appears in mBio, the online open-access journal of the American Society for Microbiology.

"The reason researchers change the vaccine every year is that they want to specifically match the vaccine to the particular viruses that are circulating, such as H1N1. If the vaccine is just a little bit different to the target virus, it is not expected to offer much protection," said principal investigator of the study Jeffery Taubenberger, MD, PhD, Chief of Viral Pathogenesis and Evolution Section, Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases (NIAID). "What we have done is design a strategy where you don't have to think about matching the vaccine antigen to the virus at all."

In the new study, researchers at the NIAID used a virus-like particle vaccine cocktail that expressed a handful of different subtypes of a key surface protein of the influenza virus: hemagglutinin H1, H3, H5 and H7. "There are 16 different hemagglutinin subtypes that circulate in birds and are thought to be the basis for current and future influenza pandemics," said Dr. Taubenberger. "The hypothesis was that the presentation of these different viral proteins would stimulate the development of cross-protective immunity that would provide broader protection against multiple subtypes."

The researchers picked the H1 and H3 subtypes because they have been the major cause of human seasonal flu outbreaks since 1918. They chose the H5 and H7 subtypes because they have been the cause of recent bird flu outbreaks and have pandemic potential. This selection also provided a broad representation of hemagglutinins across the phylogenetic tree.

In a series of experiments, the researchers found that 95% of mice vaccinated with the investigational cocktail were protected against a lethal challenge with eight different influenza strains expressing seven different influenza A subtypes, compared to only 5% of mice who received mock vaccinations.

"Almost all of the animals that were vaccinated survived, including mice that were challenged with viruses that expressed hemagglutinin subtypes that were not in the vaccine at all, viruses that expressed H2, H6, H10, and H11," said Dr.

Taubenberger. "What that suggests is that this approach really gives us broad spectrum protection, and could serve as a basis for an effective pre-pandemic vaccine."

Additional experiments showed that the vaccine was durable, effective for at least 6 months, and that it worked well in older mice. This is important given that elderly people are particularly susceptible to severe disease following influenza infection, and current vaccines are less efficacious in the elderly than in younger people.

"These initial findings are very positive and suggest a promising and practical strategy for developing a vaccine with amazing, broad protection," said Dr. Taubenberger.

http://nyti.ms/1MsoFXN

Scientists Trace an Ancient DNA Link Between Amazonians and Australasians

Some people in the Brazilian Amazon are very distant relations of indigenous Australians, New Guineans and other Australasians

By JAMES GORMAN JULY 21, 2015

Some people in the Brazilian Amazon are very distant relations of indigenous Australians, New Guineans and other Australasians, two groups of scientists who conducted detailed genetic analyses reported Tuesday. But the researchers disagree on the source of that ancestry.

The connection is ancient, all agree, and attributable to Eurasian migrants to the Americas who had some Australasian ancestry, the scientists said.

But one group said the evidence is clear that two different populations came from Siberia to settle the Americas 15,000 or more years ago. The other scientific team says there was only one founding population from which all indigenous Americans, except for the Inuit, descended and the Australasian DNA came later, and not through a full-scale migration. For instance, genes could have flowed through a kind of chain of intermarriage and mixing between groups living in the Aleutian Islands and down the Pacific Coast.

Both papers were based on comparisons of patterns in the genomes of many living individuals from different genetic groups and geographic regions, and of ancient skeletons.

David Reich of Harvard, the senior author of a paper published Tuesday in the journal Nature, said the DNA pattern was “surprising and unexpected, and we weren’t really looking for it.”

Pontus Skoglund, a researcher working with Dr. Reich who was investigating data gathered for previous research, found the pattern, or signal, as he described it. He and Dr. Reich and their colleagues used numerous forms of analysis, comparing different groups to see how distant they were genetically, to determine if there was some mistake. But, Dr. Skoglund said, “we can’t make it go away.”

Dr. Reich reported in 2012, based on some of the same evidence, that a group he called the First Americans came from Siberia 15,000 or more years ago, and were the ancestors of most Native Americans on both continents. There was a second and later migration, he said, that gave rise to a group of Indians including the Chipewyan, Apache and Navajo, who speak similar languages. The Inuit are generally agreed to have made a separate, later migration.

Now, based on new evidence and much deeper analysis, he and Dr. Skoglund and colleagues concluded that the first migration, which began 15,000 or more years ago, consisted not only of the group he identified as the First Americans, but of a second group that he calls Population Y. They could have come before, after or around the same time as the First Americans. But Population Y, he writes, “carried ancestry more closely related to indigenous Australians, New Guineans and Andaman Islanders than to any present-day Eurasians or Native Americans.”

Population Y comes from Ypykuéra, a word meaning ancestor in a language spoken by the two Amazonian groups, the Surui and Karitiana, that show the strongest genetic connection to Australasians.

The other paper, published in the journal Science, originated in the lab of Eske Willerslev, a noted detective of ancient DNA at the University of Copenhagen and the Center for Geogenetics. It came to involve 101 authors around the globe over several years of work. The goal, said Maanasa Raghavan, a molecular biologist in Copenhagen who was one of the main scientists on the project, was to bring together genomic, archaeological and other research on modern and ancient peoples of the Americas to come up with a clearer picture of how the continents were populated.

They concluded that Native Americans diverged genetically from Eurasians about 23,000 years ago. They also concluded, in contrast to the Harvard group, that all indigenous Americans except the Inuit came from one founding population.

But they, too, found the trace of Australasian ancestry in some South American natives, although it was not as strong as that reported by Harvard. Dr. Raghavan said the raw evidence in both papers of an Australasian genetic signal was consistent. “What is different is how we think that the signal got here,” she said.

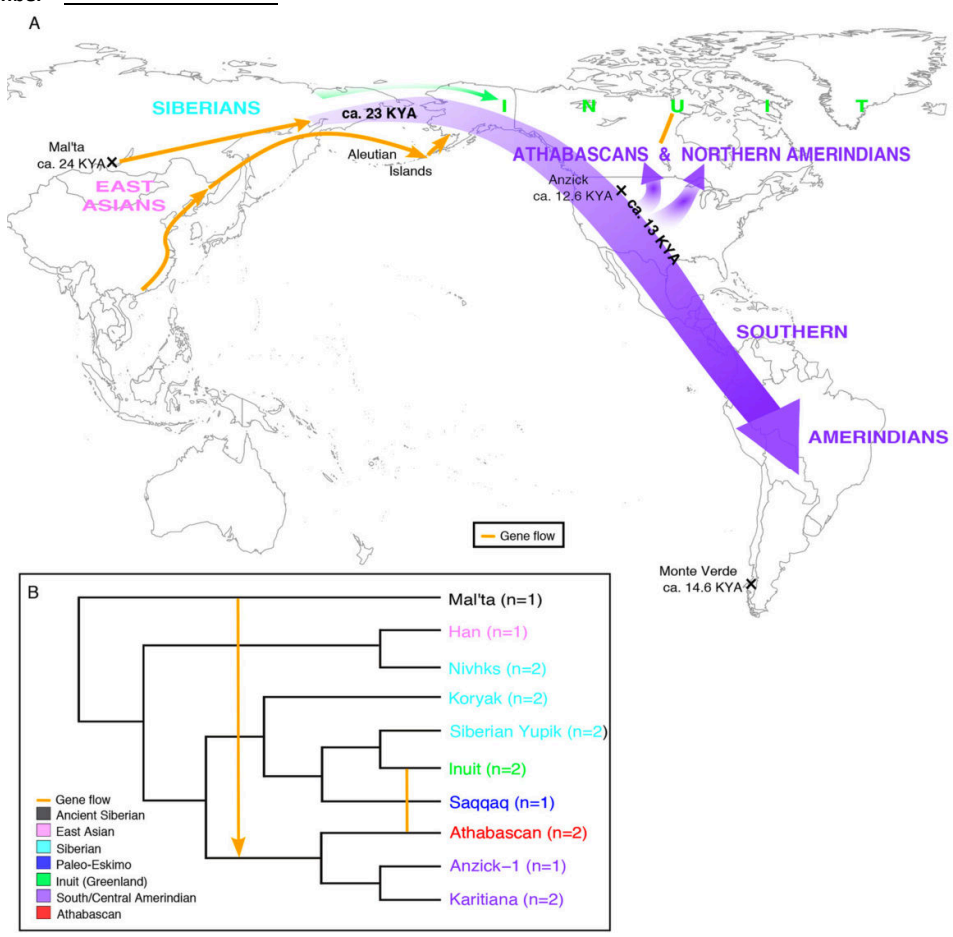
Origins and population history of Native Americans. (A) Our results show that the ancestors of all present-day Native Americans, including Amerindians and Athabascans, derived from a single migration wave into the Americas (purple), separate from the Inuit (green). This migration from East Asia occurred no later than 23 KYA and is in agreement with archaeological evidence from sites such as Monte Verde (50). A split between the northern and southern branches of Native Americans occurred ca. 13 KYA, with the former comprising Athabascans and northern Amerindians and the latter consisting of Amerindians in northern North America and Central and South America including the Anzick-1 individual (5). There is an admixture signal between Inuit and Athabascans and some northern Amerindians (yellow line); however, the gene flow direction is unresolved due to the complexity of the admixture events (28). Additionally, we see a weak signal related to Australo-Melanesians in some Native Americans, which may have been mediated through East Asians and Aleutian Islanders (yellow arrows). Also shown is the Mal'ta gene flow into Native American ancestors some 23 KYA (yellow arrow) (4). It is currently not possible for us to ascertain the exact geographical locations of the depicted events; hence, the positioning of the arrows should not be considered a reflection of these. B. Admixture plot created on the basis of TreeMix results (fig. S5) shows that all Native Americans form a clade, separate from the Inuit, with gene flow between some Native Americans and the North American Arctic. The number of genome-sequenced individuals included in the analysis is shown in brackets.

Neither group supported an existing theory, based on the shape of ancient skulls in South America, that a group called PaleoAmericans, who were very similar to Australasians, came to the Americas before the ancestors of most Native Americans did. The Science paper specifically rejected that idea, saying that both gene studies and a re-examination of the shape of some historical skulls contradicted that theory.

Rasmus Nielsen, a computational geneticist at the University of California, Berkeley, and one of the senior authors of the Science paper, said he saw no conflict in the raw data used in the two studies.

David Meltzer, an anthropologist and archaeologist at Southern Methodist University and another author of the Science paper, said the difference in interpretation between the two groups was "not an irresolvable problem." More analysis of ancient DNA or the discovery of a new skeleton could provide an answer.

Dr. Reich, of Harvard, agreed that the papers were not in disagreement, but said his team had delved far deeper in its analysis of the Australasian trace. "We have overwhelming evidence of two founding populations in the Americas," he said.



https://bitly.com/a/bitlinks/1emYQcL

New mussel-inspired surgical protein glue: Close wounds, open medical possibilities

Light-activated, mussel protein-based bioadhesive works on the same principles as mussels attaching to underwater surfaces

One of the most basic yet important surgical skills to keep a patient alive and intact may be closing wounds. It seems that doctors will now get the job done with more ease thanks to new, nontoxic surgical glue that instantly seals a bleeding wound and helps it heal without a scar or inflammation.

Inspired by nature's wonders, Korean scientists at Pohang University of Science and Technology (POSTECH) have developed a light-activated, mussel protein-

based bioadhesive (LAMBA) that works on the same principles as mussels attaching to underwater surfaces and insects maintaining structural balance and flexibility. The product, called LAMBA, has emerged as a promising candidate for an ideal bioadhesive for its outstanding properties; LAMBA's compatibility with the human body, strong adhesiveness in wet conditions, and convenient handling point to the possibility of myriad medical applications.

Mechanical fasteners like sutures and staples have accounted until recently for a major portion of conventional medical devices that are used to hold body tissues together. The invasive nature of traditional methods, however, has been the biggest drawback causing severe tissue damage, complicated post-treatment management, and scars. Their use is also limited when handling delicate tissues and internal organs, giving rise to a need for alternatives that do not require penetration. Tissue adhesives have been increasingly pursued these days as a more desirable bonding material, but the adhesives currently available in the market likewise have their own limitations. While chemically derived adhesives such as cyanoacrylates are likely to provoke an adverse reaction, biologically derived ones are not strong enough to close wounds like sutures do. A common and critical challenge, moreover, is that most surgical glues do not stick in a wet environment, which is essential for medical applications.

Dr. Hyung Joon Cha, a professor of the Department of Chemical Engineering at POSTECH, and his student, Eun Young Jeon, have developed a new approach that readily overcomes these drawbacks. The new product LAMBA, a focus of their recent publication in Biomaterials, is an upgrade version of previously known mussel-inspired adhesives that copy mussels' ability to fix their body under water. Instead of producing recombinant mussel adhesive proteins (MAPs) by modifying DOPA, a key element for the adhesive property, E.Y. Jeon et al., have created the new tissue adhesive via a photochemical reaction using blue visible light.

E.Y. Jeon et al gained the idea for this more economic, facile, and reliable strategy from dityrosine crosslinks that are often found in dragonfly wings and insect cuticles. When visible light triggers a photo-oxidation reaction in MAPs plentiful of tyrosine, neighboring tyrosine residues are instantly coupled into dityrosine crosslinks, which in turn enhance structural stability and adhesive properties of the new MAPs in the form of hydrogel.

The researchers report in their article published in Biomaterials that animal studies have proved LAMBA's superiority to existing options including sutures and other surgical glues, potentially qualifying for an ideal tissue bonding material. The new adhesive hydrogel not only closes an open wound on a bleeding site within less than 60 seconds, but also effectively facilitates the healing process without inflammation or a scar.

"LAMBA opens numerous doors for medical practices ranging from blocking air leaks and sutureless wound closures of delicate organs or tissues beyond surgeons' reach, to hemostatic agent and drug delivery medium, just to name a few," commented Dr. Cha, a corresponding author of this study.

This work was supported by the Marine Biotechnology program of Ministry of Oceans and Fisheries, Korea. The main author, E.Y.J was supported by Global PH.D Fellowship program funded by the Ministry of Education, Korea.

http://www.eurekalert.org/pub_releases/2015-07/gu-eba072115.php

Elderberry benefits air travelers

The negative health effects of international air travel are well documented but now it seems that the common elderberry can provide some relief

The negative health effects of international air travel are well documented but now it seems that the common elderberry can provide some relief.

Associate Professor Evelin Tiralongo and Dr Shirley Wee from Griffith's Menzies Health Institute Queensland (MHIQ) have completed a clinical trial showing that an elderberry supplement can provide some protection from cold and flu-like symptoms following long-haul flights.

Elderberries are pictured. ニワトコ **Credit: Iprona AG**

Intercontinental air travel can be stressful and affect a passenger's physical and psychological wellbeing. Whilst jet lag and fatigue remain the best known problems, holidaymakers also often experience upper respiratory symptoms.

Presenting their results at the 21st Annual International Integrative Medicine Conference in Melbourne, the research team showed how elderberry appears to reduce the duration and severity of the cold.

The randomised, double-blind placebo controlled clinical trial was conducted with 312 economy class passengers travelling from Australia to an overseas destination. Cold episodes, cold duration and symptoms were recorded in a daily diary and participants also completed surveys before, during and after travel.

"We found that most cold episodes occurred in the placebo group, but the difference between the placebo and active group was not significant. However, the placebo group had a significantly higher number of cold episode days, and the symptom score in the placebo group over these days was also significantly higher," says Associate Professor Tiralongo.

"Complementary medicines are used by two in three Australians, thus increasing the evidence base of these medicines should be at the forefront of our efforts. It's often forgotten that the evidence for various herbal medicines is extract specific," says Associate Professor Tiralongo.

The trial used capsules containing 300mg of a standardised, proprietary membrane-filtered elderberry extract which has shown to be effective in working against respiratory bacteria and influenza viruses.

The Griffith study follows recent European research published in the open access journal Current Therapeutic Research which suggests that a combination of Echinacea herb and root extract supplemented with elderberry can be as effective as the conventional antiviral medicine Tamiflu for the early treatment of influenza.

http://www.eurekalert.org/pub_releases/2015-07/p-cod071615.php

Class of diabetes medication associated with lower incidence of Parkinson's disease

Lower incidence of PD among people using a glitazone drug

A class of drugs used to treat diabetes may be associated with protection against Parkinson's disease (PD), according to research published this week in PLOS Medicine. The study, conducted by Dr. Ruth Brauer, of the London School of Hygiene & Tropical Medicine, found a lower incidence of PD among people using a glitazone drug (either rosiglitazone or pioglitazone) to treat diabetes when compared to people who had used different treatments for diabetes.

The cohort study was conducted using data from the UK Clinical Practice Research Datalink, and compared individuals with diabetes who were exposed to glitazones (44,597 total) with up to five individuals with diabetes who never used glitazones (120,373 in total), matched on age, sex, primary care practice, and diabetes treatment stage. The researchers analyzed the records of these patients from 1999, when glitazones were introduced to treat diabetes, until 2013. During that time, individuals who had used glitazones to treat diabetes were 28% less likely to be diagnosed with PD than individuals with diabetes who never used glitazones. Adjusting for known predictors of PD such as smoking and head injury did not alter this association. When the researchers considered past and current glitazone users separately, they found that the decreased incidence in PD was only observed in individuals currently using a glitazone (a 41% decrease in PD incidence), not those who had previously used glitazone but stopped or switched to another medication, indicating little to no persisting benefit of glitazone use.

These findings are consistent with animal and in vitro studies which suggested that glitazones and other drugs that target peroxisome proliferation-activated receptor gamma (PPARγ) may have neuroprotective effects. It is important to note that these results may not apply to people without diabetes and do not indicate whether glitazones can slow PD progression. Further, it is possible that unknown patient characteristics associated with glitazone use might also be linked to PD, contributing to the appearance of a direct causal connection. In addition, glitazones have been associated with serious side effects.

However, the authors are hopeful that these findings may pave the way towards other treatments that target the same pathway: "Our findings indicate that interventions based on the same mechanisms as PPARγ agonist activity may be fruitful targets for future research in PD."

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Competing Interests: *I have read the journal's policy and the authors of this manuscript have the following competing interests: LS has received research funding from GSK and ID has consulted for, and holds stock in, GSK.*

Citation: *Brauer R, Bhaskaran K, Chaturvedi N, Dexter DT, Smeeth L, Douglas I (2015) Glitazone Treatment and Incidence of Parkinson's Disease among People with Diabetes: A Retrospective Cohort Study. PLoS Med 12(7): e1001854. doi:10.1371/journal.pmed.1001854*

http://www.bbc.com/news/health-33610569

Cell transplant 'regenerates' liver

Transplanting cells into livers has the potential to completely regenerate them, say scientists.

The Medical Research Council team showed severely damaged organs in mice could be restored to near-normal function. They say the findings, published in Nature Cell Biology, could eventually help people stuck on a waiting list for a transplant. Further tests are now taking place with human tissue.

The liver does have a remarkable ability to heal itself. Even if half of the organ is removed, it can grow back. The team, based at the University of Edinburgh, has been investigating the regenerative potential of the liver. Normally, the main type of cell in the liver - hepatocytes - is able to restore the organ.

But one of the researchers, Prof Stuart Forbes, said: "The hepatocytes normally divide beautifully, but eventually they give up that ability to keep dividing, they become senescent, and that is something we see in all forms of severe liver injury."

Regeneration

So the Edinburgh team turned to a closely related group of stem cells from the biliary duct. Injecting these cells into damaged mouse livers led to near compete regeneration. Prof Forbes added: "The big aim would be to develop a clinically applicable cell therapy for patients with severe liver failure where transplantation is not an option."

The team say tissue from livers unsuitable for transplant could be a source of these cells. However, Prof Forbes said liver transplants would remain the main option for patients and encouraged people to join the donor register. Further studies will now focus on repeating the results with human tissue.

Dr Rob Buckle, the director of science programmes at the Medical Research Council, said: "This research has the potential to revolutionise patient care by finding ways of co-opting the body's own resources to repair or replace damaged or diseased tissue."

http://www.bbc.com/news/health-33617141

Early signs that drug 'may delay Alzheimer's decline'

The first details of how a drug could slow the pace of brain decline for patients with early stage Alzheimer's disease have emerged.

By James Gallagher Health editor, BBC News website

Data from pharmaceutical company Eli Lilly suggests its solanezumab drug can cut the rate of the dementia's progression by about a third. The results, presented to a US conference, are being met with cautious optimism.

A new trial is due to report next year and should provide definitive evidence.

The death of brain cells in Alzheimer's is currently unstoppable. Solanezumab may be able to keep them alive. Current medication, such as Aricept, can manage only the symptoms of dementia by helping the dying brain cells function. But solanezumab attacks the deformed proteins, called amyloid, that build up in the brain during Alzheimer's. It is thought the formation of sticky plaques of amyloid between nerve cells leads to damage and eventually brain cell death.

Silver lining

Solanezumab has long been the great hope of dementia research, yet an 18-month trial of the drug seemingly ended in failure in 2012. But when Eli Lilly looked more closely at the data, there were hints it could be working for patients in the earliest stages of the disease. It appeared to slow progression by around 34% during the study. So the company asked just over 1,000 of the patients in the original trial with mild Alzheimer's to take the drug for another two years.

And positive results from this extension of the original trial have now been presented at the Alzheimer's Association International Conference. They show those taking the drugs the longest had the most benefit.

Dr Eric Siemers, from the Lilly Research Laboratories, in Indiana, told the BBC: "It's another piece of evidence that solanezumab does have an effect on the underlying disease pathology. "We think there is a chance that solanezumab will be the first disease-modifying medication to be available."

The company also started a completely separate trial in mild patients in 2012, and these results could prove to be the definitive moment for the drug.

Analysis

Today is not the day to jump up and down proclaiming a breakthrough in slowing the pace of Alzheimer's. The limited data which has been released is the scientific equivalent of a poll before a general election or a trailer ahead of a movie. It provides captivating clues, hints and teases, but nothing definitive.

At the moment there is no medication that can slow down dementia. If such a drug was developed it could transform how the disease is managed. People would still get worse, but they would spend more time in the milder phase of the degenerative disease rather than needing constant care. In a field that has been plagued by repeated disappointment, even a hint of such a drug is an exciting moment. Next year, when further trial results are due, we will know for certain whether solanezumab is the breakthrough everyone hopes it could be.

Potential breakthrough

Dr Eric Karran, the director of research at Alzheimer's Research UK, told the BBC News website: "If this gets replicated, then I think this is a real breakthrough in Alzheimer's research. "Then, for the first time, the medical community can say we can slow Alzheimer's, which is an incredible step forward. "These data need replicating, this is not proof, but what you can say is it is entirely consistent with a disease-modifying effect. "We've never ever had evidence that we can affect the disease process."

Clare Walton, the research manager at the Alzheimer's Society, told the BBC: "The data hints that the antibodies are having an effect, it is promising and it's better than no effect, but it's inconclusive. "After a decade of no treatments and many drug failures, it's exciting to get promising news, but it doesn't really tell us either way, and we need to wait for the phase-three study, and that is in 18 months."

How much benefit?

In the first stage of the original trial, which ended in failure, half of the patients with Alzheimer's were given solanezumab and half were not. A reanalysis of the cognition scores of the patients with mild Alzheimer's suggested taking the drug had cut the rate of the disease's progression by about 34%. The implication is that the amount of cognitive decline normally seen in 18 months would take 24 months with the drug.

In the extension of the original trial, all of the 1,000-plus mild Alzheimer's patients participating were given solanezumab. So, at the end of the extension, half of them had been taking the drug for three and a half years while the other half had been taking it for two years. The latest data shows those taking solanezumab for the longest time still had better scores of cognitive function. This suggests the course of the disease was being slowed.

If the patients' brains had continued to decline at the normal pace and the drug had been merely helping with symptoms, then all of the patients participating in the extension of the original trial - whether they had been taking solanezumab for three and a half or two years - would have had similar scores of cognitive function.

http://www.bbc.com/news/technology-33609495

Robotic surgery linked to 144 deaths in the US

A study into the safety of surgical robots has linked the machines' use to at least 144 deaths and more than 1,000 injuries over a 14-year period in the US.

The events included broken instruments falling into patients' bodies, electrical sparks causing tissue burns and system errors making surgery take longer than planned. The report notes that the figures represent a small proportion of the total number of robotic procedures. But it calls for fresh safety measures.

"Despite widespread adoption of robotic systems for minimally invasive surgery, a non-negligible number of technical difficulties and complications are still being experienced during procedures," the study states. "Adoption of advanced techniques in design and operation of robotic surgical systems may reduce these preventable incidents in the future." Robotic surgery can reduce the risk of infections and help patients heal more quickly.

The UK's Royal College of Surgeons said it believed the report should be "treated with caution". "The authors note 'little or no information was provided in the adverse incident reports' about the cause of the majority of deaths, meaning they could be related to risks or complications inherent during surgery," said a spokeswoman. "The authors do not compare the level of complications in surgery where robots are not used, nor do they examine the benefits of robotic surgery which are starting to be reported."

More accidents

The work was carried out by researchers at the University of Illinois at Urbana-Champaign, the Massachusetts Institute of Technology and Chicago's Rush University Medical Center. Their paper says 144 deaths, 1,391 injuries and 8,061 device malfunctions were recorded out of a total of more than 1.7 million robotic procedures carried out between January 2000 and December 2013.

This was based on reports submitted by hospitals, patients, device manufacturers and others to the US Food and Drug Administration, and the study notes that the true number could be higher. Its authors say the number of injuries and deaths per procedure has remained relatively constant since 2007. But due to the fact that the use of robotic systems is increasing "exponentially", they add, this means that the number of accidents is increasing every year.

They highlight that when problems do occur, people are several times more likely to die if the surgery involves their heart, lungs, head and/or neck rather than gynaecological and urological procedures.

They acknowledge that the data does not pinpoint why, but suggest it is because the former are more complex types of operations for which robots are less commonly used, so there is less experience and expertise available.

The researchers did not, however, compare accident rates with similar operations in which robots were not used. Their study has not been peer reviewed.

Falling sales

Surgical robotic devices are typically expensive - costing millions of pounds - but offer advantages. They can allow surgeons to use smaller instruments, letting them make smaller and more nimble cuts. That can mean patients recover faster, with less risk of infection and the promise of smaller scars.

In addition, the development of remote surgery means that doctors do not always need to be in the same room as their patients, allowing specialists who are in demand to treat more people.

Despite these benefits, sales of surgical robots declined by 2% in 2013 - the most recent year for which figures have been published by the International Federation of Robotics. That has been linked to some medical experts questioning claims that the cost of using such machines is justified by improved outcomes.

"There is no good data proving that robotic hysterectomy is even as good as - let alone better - than existing, and far less costly, minimally invasive alternatives," the American College of Obstetricians and Gynecologists said in 2013.

"Aggressive direct-to-consumer marketing of the latest medical technologies may mislead the public into believing that they are the best choice."

Others specialists have, however, vouched for such systems' benefits in other procedures. "The Royal Marsden has performed more robotic surgical procedures for prostate cancer than any other hospital in the UK," states the London hospital's website. "We have dramatically improved functional and oncological outcomes for patients undergoing radical prostatectomy [the removal of the prostate gland to treat cancer]."

Broken parts

Although the study links hundreds of injuries and deaths to robotic surgery, in most cases the FDA's logs do not make clear whether the use of the machines was directly responsible. In fact, of the headline figures, only a minority - five of the

deaths and 436 of the injuries - are specifically tied to technical errors that occurred during an operation.

But the authors say there is still reason to be concerned. They list 1,166 cases of broken/burned parts falling into patients' bodies, which contributed to 119 injuries and one death. Uncontrolled movements and spontaneous powering on/off of the machines are said to have caused 52 injuries and two deaths. Electrical sparks, unintended charring and damaged accessory covers are linked to 193 injuries, including the burning of body tissues. And the loss of quality video feeds and/or reports of system error codes are said to have contributed to a further 41 injuries and one death.

The report's authors suggest that one way to tackle such problems would be to give surgical teams more troubleshooting training - including the use of computer simulations that feature technical problems - to help them learn how to restart surgery more quickly after interruptions.

http://www.eurekalert.org/pub_releases/2015-07/uoia-mdd072215.php

Mowing dry detention basins makes mosquito problems worse, team finds

Mowing wetland plants can increase populations of mosquitoes that carry the West Nile virus, researchers report

CHAMPAIGN, Ill. -- A study of the West Nile virus risk associated with "dry" water-detention basins in Central Illinois took an unexpected turn when land managers started mowing the basins. The mowing of wetland plants in basins that failed to drain properly led to a boom in populations of Culex pipiens mosquitoes, which can carry and transmit the deadly virus, researchers report.

A paper describing their findings is in press in the journal Ecological Applications. The team, led by University of Illinois postdoctoral researcher Andrew Mackay, found that mowing down cattails and phragmites, two invasive plants that tend to permeate stormwater basins, adds a lot of plant debris to the water.

"We suspect bacteria quickly colonize the waterborne debris, and mosquito larvae feed on the bacteria," said Illinois entomology professor Brian Allan, a co-author on the study with Mackay, Illinois Natural History Survey entomologist Ephantus Muturi and U. of I. natural resources and environmental sciences professor Michael Ward.

"After aquatic plants were mowed in the basins, we saw a large increase in the number of Culex pipiens mosquito larvae in the basins, which had relatively few before mowing," Mackay said. "And perhaps more importantly, we caught about twice as many adult Culex mosquitoes in traps at basins after these plants were mowed, compared with basins where the aquatic vegetation was left intact."

Mowing phragmites, a tall and sturdy invasive grass, also dispersed a host of bird species that liked to roost in the grass, Allan said.

"We had observed that these phragmites-invaded basins would become colonized by large communal roosts of birds," he said. "And we thought that was important because birds are the natural reservoir hosts of West Nile Virus."

The researchers suspected that a bird roost near a mosquito nursery might increase the West Nile virus risk to people living nearby.

"Instead, we found that the presence of a communal bird roost actually decreased West Nile virus risk," Allan said. "That may be because these wetland roosts include a variety of bird species, many of which are not good reservoirs of the virus. They don't amplify the virus like other bird species more associated with residential areas do - the American robin, for example.

"We measured mosquito abundance, and we measured West Nile virus prevalence in the mosquitoes we collected in this field study, and we were able to show that it's these mowed areas where you actually get the highest West Nile virus risk to people in the surrounding landscape," Allan said.

"You might think you're helping by mowing, but you're creating another problem," Muturi said. "It's all a matter of good planning and coordination to be sure that the kind of activities we do, either for aesthetic or for any other reason, don't increase public health risk."

The INHS is a division of the Prairie Research Institute at the U. of I.

The U. of I. School of Integrative Biology; the Institute for Sustainability, Energy and the Environment; and the Illinois Used Tire Management Fund supported this research.

The paper, "Invasive aquatic macrophytes in urban stormwater habitats and West Nile virus transmission risk," is available online or from the U. of I. News Bureau.

http://www.eurekalert.org/pub_releases/2015-07/wuso-dtd072215.php

Diagnostic test developed for enterovirus D68

Respiratory virus caused severe illness, deaths in children

Researchers at Washington University School of Medicine in St. Louis have developed a diagnostic test to quickly detect enterovirus D68 (EV-D68), a respiratory virus that caused unusually severe illness in children last year. The outbreak caused infections at an unprecedented rate, with over 1,000 confirmed cases and 14 reported deaths nationwide, according to the Centers for Disease Control and Prevention (CDC).

Results published in the August issue of the Journal of Clinical Microbiology demonstrate that the test is extremely effective at identifying various strains of EV-D68 and reduces the amount of time needed to detect the virus.

Earlier procedures for identifying enterovirus strains involved sequencing a region of the virus's genome, which is too cumbersome to perform on large numbers of

patients. The new test can be completed in a few hours, while previous techniques took several days to process. The researchers also said the new test is more specific than commercially available diagnostic tests for enterovirus.

"Commercial tests for respiratory viral infections typically don't distinguish between rhinoviruses, which cause the common cold, and enteroviruses, and within each of those groups there are many different types," said senior author Gregory A. Storch, MD, the Ruth L. Siteman Professor of Pediatrics.

"Having a tool to identify which cases of respiratory illness are actually EV-D68 is an advantage for public health," he added. "These kinds of tests help treatment decisions because it is important to know that the patient doesn't have influenza or another disease that might require a specific treatment. It's also important in a hospital for preventing infections because doctors take patients with one particular virus and keep them apart from patients infected with other infectious agents."

Since details of the test's technique are published, other labs can use it as a template to create their own tests in the event of another outbreak.

Rhinoviruses and enteroviruses are common causes of respiratory illnesses. Clinicians often don't test patients for suspected infections with either virus especially when their illnesses are mild. But after last year's deadly outbreak of the D68 strain, a team of researchers led by Storch and Todd N. Wylie, instructor in pediatrics, started working to develop a test that could target EV-D68.

There are a number of variants of enterovirus D68 that are closely related genetically. But slight variations in their genomes complicate researchers' ability to detect all variants with one test. Because of this variation, earlier EV-D68 tests will miss some of the EV-D68 variants.

The researchers isolated tiny segments of viral DNA sequences that are common to every D68 subtype but no other virus. Wylie developed a computer program that allows large numbers of DNA sequences to be compared simultaneously. No other lab previously had used public databases in this manner to collect the nucleotide sequences of every variant of D68 available and compare them to the sequences of all other viruses for which sequence data is available. Wylie's technique made it possible to create a test that identifies every known subtype of enterovirus D68 while excluding all other known viruses.

The new test is more effective than others for EV-D68, including one developed by the CDC in October that was deployed quickly in response to the public health crisis. To assess the new test, the researchers examined known panels of rhinoviruses and enteroviruses. Of the viruses they looked at, the new test did not miss any known samples of EV-D68, and it did not falsely identify EV-D68 in samples that were known to be other viruses. The test retained its accuracy even with tiny amounts of the virus.

"There are a range of D68 viruses, and our assay was designed to detect them all," Storch said. "We received many samples of enterovirus from other hospitals and ran the test blindly on all of them. In the viruses we looked at, the test worked 100 percent of the time. It only detected EV-D68 strains, and it did detect all of them; the test didn't detect any of the other enteroviruses or rhinoviruses."

The researchers described some possible limitations of the new test. There are many viruses with sequences that are not available and could not be considered in designing the test. In addition, the researchers said that EV-D68 potentially could mutate in the future so that the assay might fail to detect it.

The new test was made possible by earlier work by Kristine M. Wylie, PhD, assistant professor of pediatrics, and colleagues in the Department of Pediatrics and at the university's McDonnell Genome Institute, who sequenced the genome of EV-D68 that circulated in St. Louis last year. One future area of research is to investigate whether Wylie's technique can serve as a template to create more tests for other viruses whose genomes have been sequenced.

This work was supported by the National Institute of Allergy and Infectious Diseases of the National Institutes of Health (NIH), grant numbers R01AI097213 and U01AI077810, and by institutional funds.

Wylie TN, Wylie KM, Buller RS, Cannella M, Storch GA. Development and evaluation of an enterovirus D68 real-time reverse transcriptase polymerase chain reaction (RT-PCR) assay. The Journal of Clinical Microbiology. Online June 10, 2015.

http://www.eurekalert.org/pub_releases/2015-07/bu-rip072215.php

Researchers identify plant cultivation in a 23,000-year-old site in the Galilee

Earliest-known example of plant cultivation in the Levant is 11,000 years before earliest-known agriculture

The Middle East is called the "Cradle of Civilization" because it is where our hunter-gatherer ancestors first established sedentary farming communities. Recently, the traditional dating of humans' first agricultural attempt was shaken up by the discovery of the earliest-known example of plant cultivation in the Levant, 11,000 years earlier than previously accepted.

The team of archaeologists, botanists, and ecologists from Bar-Ilan University, Haifa University, Tel Aviv University, and Harvard University published their work in the scientific journal Plos One on July 22, 2015. The team's conclusions rest on three inter-connected findings, says the study's lead researcher, Prof. Ehud Weiss of Bar-Ilan University's Martin (Szusz) Department of Land of Israel Studies and Archaeology. First is the higher-than-usual presence at the site of domestic-type, rather than wild-type, wheat and barley dispersal units. Second, the researchers noted a high concentration of proto-weeds - plants of the type known

to flourish in fields planted with domesticated crops. Finally, analysis of the tools found at the site revealed blades used for cutting and harvesting cereal plants.

First author is Dr. Ainit Snir, part of whose doctoral research - conducted in Prof. Weiss' lab - is included in the present study.

An Agricultural "Time Capsule" Hidden Under the Sea

The researchers' discovery was made at Ohalo II, a 23,000-year-old camp site of a community of hunter-gatherers that lived on the shore of the Sea of Galilee, Israel. The site is located 9 kilometers (5.5 miles) south of the modern city of Tiberias, and was discovered in 1989 when the level of the lake plummeted. The site was then excavated for six seasons by Prof. Dani Nadel from the Zinman Institute of Archaeology, the University of Haifa. Excavations at Ohalo II exposed six brush hut dwellings, a human grave, copious and well-preserved remains of both animal and plant foods, beads from the Mediterranean Sea, as well as evidence of flint tool manufacture and use. According to Weiss, the study represents the earliest example of small-scale cultivation found anywhere in the world.

"The plant remains from the site were unusually well-preserved because of being charred and then covered by sediment and water which sealed them in low-oxygen conditions," Weiss explains. "Due to this, it was possible to recover an extensive amount of information on the site and its inhabitants - which made this a uniquely preserved site, and therefore one of the best archaeological examples worldwide of hunter-gatherers' way of life. Here we see evidence of repeated sowing and harvesting of later domesticated cereals."

From Plant Gathering to Flour Production

In the Ohalo II dwellings was a particularly rich assemblage of some 150,000 plant remains, showing that the site's residents gathered over 140 different plant species from the surrounding environment. Among these, Weiss's team identified edible cereals - such as wild emmer, wild barley, and wild oats. These cereals were mixed with 13 species of "proto-weeds" - ancient ancestors of the current weeds known to flourish in cultivated, single-crop fields - indicating that they grew and were subsequently unintentionally gathered together.

A grinding slab set firmly on a brush hut floor, a stone tool from which microscopic cereal starch granules were extracted, as well as a unique distribution pattern of seeds around this tool, provided additional, unequivocal evidence that cereal grains were brought into the hut and processed into flour. This flour was probably used to make dough, maybe by baking it on an installation of flat stones, found just outside one of the shelters.

Plants' Statistics Show Genetic Change Linked with Cultivation

Examination of the cereals found at the site shows an unusual percentage of domesticated-type, rather than wild-type, ear morphology. As Weiss explains, this change in the plant population is characteristic of a genetic mutation triggered when wild-type plants are sown repeatedly in cultivated fields.

"The ears of cereals like wheat and barley - in their wild form - are built from separate units that break off and are easily dispersed, allowing the seeds to reach the ground, germinate, and grow into a new plant without any human intervention," he says. "When humans cultivate these grains over a number of successive seasons, however, a change occurs. They develop a rough scar that locks the seed dispersal units together. Such plants cannot sow themselves. This is the hallmark of domesticated, rather than wild-type plants."

As part of Snir's thesis, Weiss and Snir undertook field tests around Israel, establishing that stands of wild-type barley are characterized by a low level of this rough-scar appearance - about 10% of the total population. The study of Ohalo II's plant remains, however, revealed a greatly-increased incidence of 36% mutated domestic-type disarticulation units - proving that planned cereal sowing and harvesting in this ancient community had been underway for years.

Tools for Harvesting

Another intriguing finding relates to a number of sickle blades - harvesting tools composed of sharp flint implements inserted in wood or bone handles - found at the site; these are among the oldest of their kind ever found.

"We found several sickle blades at Ohalo II, and the study under the microscope of the gloss along their cutting edge indicates that they were used for harvesting cereals just before their complete ripening," says Prof. Dani Nadel. "Analysis showed the presence of silicon, transferred from the wheat and barley plants at the time of cutting. This is another indication that the presence of a high percentage of domestic-type cereals was not random, but rather is a sign of the long-term cultivation practices of the site's residents."

Weeds and Planted Fields

When studying the plants found at Ohalo II, the researchers were surprised to find a large number of plants similar to weeds previously seen only 11,000 years later than Ohalo II, at the traditional date for the beginning of agriculture. Does this indicate that agriculture indeed began much earlier than historians, archaeologists and botanists have traditionally believed? Weiss says that the isolated example on the shores of the Sea of Galilee is an insufficient basis for such a claim.

"From what we see at Ohalo II, it is clear that cultivation occurred at this surprisingly early point in time, but we have no evidence that it continued in the region," Weiss says. "This is why we term our findings to be evidence of trial cultivation only. Moreover, since weeds are defined by botanists as plants that developed in response to human agriculture, we call the plants that share characteristics with weeds 'proto-weeds'."

A Trial that Preceded Later-Adopted Practice

Prof. Marcelo Sternberg, a co-author of the paper who is an ecologist at the Department of Molecular Biology and Ecology of Plants at Tel Aviv University, claims that the findings are exceptional. "We are witnessing the earliest trial of cultivation combined with land-use changes that led to the appearance of the earliest weeds. The findings are a clear indication of early human disturbance of the natural ecosystem."

Weiss agrees, adding that the current study provides reason to rethink our ancestors' abilities. "Even prior to full-scale cultivation, humans clearly had some basic knowledge of agriculture and even more importantly, exhibited foresight and planning," Weiss says. "The current research results from this site, situated in the cradle of ancient civilizations, show our ancestors were cleverer and more skilled than we had assumed. Although full-scale agriculture did not develop until much later, the attempt had already begun."

Paper co-author Prof. Ofer Bar-Yosef, a prehistorian from Harvard University's Department of Anthropology, notes that "the history of the evolution of technology is littered with new inventions that were either not accepted by their society or simply failed. An historical example is Leonardo da Vinci, who, in his notebooks, designed several flying machines during the early 15th century. Even though da Vinci was on the right track, we had to wait until the 19th century before the Wright brothers got their first plane off the ground."

http://www.eurekalert.org/pub_releases/2015-07/cmc-uli072215.php

Using low-dose irradiation, researchers can now edit human genes

Effectiveness of gene editing in human stem cells improves tenfold using new technique

LOS ANGELES - For the first time, researchers have employed a gene-editing technique involving low-dose irradiation to edit the genome of patient stem cells, according to a study published in the journal Stem Cells Translational Medicine. This method, developed by researchers in the Cedars-Sinai Board of Governors Regenerative Medicine Institute, is 10 times more effective than techniques currently in use.

"This novel technique allows for far more efficient gene editing of stem cells and will increase the speed of new discoveries in the field," said co-senior author Clive Svendsen, PhD, director of the Board of Governors Regenerative Medicine Institute.

The irradiation method could prove effective in learning more about diseases such as spinal muscular atrophy, muscular dystrophy and Huntington's disease. Gene editing allows scientists to correct disease causing mutations and, theoretically, cure the disease in the petri dish. Additionally, gene-editing technology allows scientists to create disease mutations in normal cells, thus modeling human disease.

When using this form of gene editing, Cedars-Sinai scientists can more efficiently insert reporter genes that glow when a stem cell turns into a specific cell of the body. For example, stem cells would turn green when converted into a heart cell and red when turned into a neuron.

"The combination of low-dose irradiation and correct gene copy will accelerate our ability to model human disease using stem cells from patients with many different disorders," said co-senior author Vaithilingaraja Arumugaswami, MVSc, PhD, director of the Pancreas and Liver Program in the Cedars-Sinai Board of Governors Regenerative Medicine Institute.

Over the past few years, the field of creating human diseases in the dish using stem cells has expanded rapidly. This work allows scientists to test novel drugs on human cells that carry disease-causing genes.

"Radiation, which is normally considered harmful, has proven beneficial in gene editing," said Svendsen. This new technique will help us establish far more accurate models and accelerate the discovery process.".

Additional Cedars-Sinai scientists involved in the study include lead project scientist Seigo Hatada, PhD; Aparna Subramanian, PhD; Berhan Mandefro; Songyang Ren, MD, PhD; Ho Won Kim, PhD; Jay (Jie) Tang, PhD; Vincent Funari, PhD; Robert Baloh, MD, PhD; and Dhruv Sareen, PhD.

Funding for this novel work was supported by a Cedars-Sinai programmatic award and the ALS Association.

Citation: Stem Cells Translational Medicine: 2015 July: Low dose irradiation enhances gene targeting in human pluripotent stem cells.

http://www.eurekalert.org/pub_releases/2015-07/uoc-mto072115.php

Musical tastes offer a window into how you think

Do you like your jazz to be Norah Jones or Ornette Coleman, your classical music to be Bach or Stravinsky, or your rock to be Coldplay or Slayer? The answer could give an insight into the way you think, say researchers from the University of Cambridge.

In a study published today in the journal PLOS ONE, a team of psychologists show that your thinking style - whether you are an 'empathizer' who likes to focus on and respond to the emotions of others, or a 'systemizer' who likes to analyse rules and patterns in the world--is a predictor of the type of music you like.

Music is a prominent feature of everyday life and nearly everywhere we go. It's easy for us to know what types of music we like and don't like. When shuffling songs on an iPod, it takes us only a few seconds to decide whether to listen or skip to the next track. However, little is known about what determines our taste in music.

Researchers over the past decade have argued that musical preferences reflect explicit characteristics such as age and personality. For example, people who are open to new experiences tend to prefer music from the blues, jazz, classical, and folk genres, and people who are extraverted and 'agreeable' tend to prefer music from the pop, soundtrack, religious, soul, funk, electronic, and dance genres.

Now a team of scientists, led by PhD student David Greenberg, has looked at how our 'cognitive style' influences our musical choices. This is measured by looking at whether an individual scores highly on 'empathy' (our ability to recognize and react to the thoughts and feelings of others) or on 'systemizing' (our interest in understanding the rules underpinning systems such as the weather, music, or car engines) - or whether we have a balance of both.

"Although people's music choices fluctuates over time, we've discovered a person's empathy levels and thinking style predicts what kind of music they like," said David Greenberg from the Department of Psychology. "In fact, their cognitive style - whether they're strong on empathy or strong on systems - can be a better predictor of what music they like than their personality."

The researchers conducted multiple studies with over 4,000 participants, who were recruited mainly through the myPersonality Facebook app. The app asked Facebook users to take a selection of psychology-based questionnaires, the results of which they could place on their profiles for other users to see. At a later date, they were asked to listen to and rate 50 musical pieces. The researchers used library examples of musical stimuli from 26 genres and subgenres, to minimise the chances that participants would have any personal or cultural association with the piece of music.

People who scored high on empathy tended to prefer mellow music (from R&B, soft rock, and adult contemporary genres), unpretentious music (from country, folk, and singer/songwriter genres) and contemporary music (from electronica, Latin, acid jazz, and Euro pop). They disliked intense music, such as punk and heavy metal. In contrast, people who scored high on systemizing favoured intense music, but disliked mellow and unpretentious musical styles.

The results proved consistent even within specified genres: empathizers preferred mellow, unpretentious jazz, while systemizers preferred intense, sophisticated (complex and avant-garde) jazz.

The researchers then looked more in-depth and found those who scored high on empathy preferred music that had low energy (gentle, reflective, sensual, and warm elements), or negative emotions (sad and depressing characteristics), or emotional depth (poetic, relaxing, and thoughtful features). Those who scored high on systemizing preferred music that had high energy (strong, tense, and thrilling elements), or positive emotions (animated and fun features), and which also featured a high degree of cerebral depth and complexity.

David Greenberg, a trained jazz saxophonist, says the research could have implications for the music industry. "A lot of money is put into algorithms to choose what music you may want to listen to, for example on Spotify and Apple Music. By knowing an individual's thinking style, such services might in future be able to fine tune their music recommendations to an individual."

Dr Jason Rentfrow, the senior author on the study says: "This line of research highlights how music is a mirror of the self. Music is an expression of who we are emotionally, socially, and cognitively."

Professor Simon Baron-Cohen, a member of the team, added; "This new study is a fascinating extension to the 'empathizing-systemizing' theory of psychological individual differences. It took a talented PhD student and musician to even think to pose this question. The research may help us understand those at the extremes, such as people with autism, who are strong systemizers."

Based on their findings, the following are songs that the researchers believe are likely to fit particular styles:

High on empathy

Hallelujah* - *Jeff Buckley
Come away with me* - *Norah Jones
All of me* - *Billie Holliday
Crazy little thing called love* - *Queen

High on systemizing

Concerto in C* - *Antonio Vivaldi
Etude Opus 65 No 3* -- *Alexander Scriabin
God save the Queen* - *The Sex Pistols
Enter the Sandman* - *Metallica

Funders

David Greenberg was funded by the Cambridge Commonwealth, European and International Trust and the Autism Research Trust during the period of this work.

Reference

Greenberg, D. M., Baron-Cohen, S., Stillwell, D. J., Kosinski, M., & Rentfrow, P. J. (2015). Musical preferences are linked to cognitive styles. PLOS ONE; 22 July 2015 http://dx.plos.org/10.1371/journal.pone.0131151

http://www.eurekalert.org/pub_releases/2015-07/uon-nmf072215.php

New material forges the way for 'stem cell factories'

First fully synthetic substrate with potential to grow billions of stem cells

If you experience a major heart attack the damage could cost you around five billion heart cells. Future stem cell treatments will require this number and more to ensure those cells are replaced and improve your chances of survival.

Experts at The University of Nottingham have discovered the first fully synthetic substrate with potential to grow billions of stem cells. The research, published in the academic journal Advanced Materials, could forge the way for the creation of 'stem cell factories' - the mass production of human embryonic (pluripotent) stem cells.

The £2.3m research project, 'Discovery of a Novel Polymer for Human Pluripotent Stem Cell Expansion and Multilineage Differentiation', was led by Morgan Alexander, Professor of Biomedical Surfaces in the School of Pharmacy and Chris Denning, Professor of Stem Cell Biology in the School of Medicine and funded by the Engineering and Physical Sciences Research Council (EPSRC). The material could provide an off-the-shelf product for clinical use in the treatment of the heart, liver and brain. To find out more watch the video.

Professor Alexander, Director of the Interface and Surface Analysis Centre, and his team have been searching for polymers on which human pluripotent stem cells can be grown and differentiated in vast numbers - billions at a time.

Professor Alexander said: "The possibilities for regenerative medicine are still being researched in the form of clinical trials. What we are doing here is paving the way for the manufacture of stem cells in large numbers when those therapies are proved to be safe and effective."

Billions of stem cells are needed as trials move into second phase

Using a high throughput materials discovery approach the research team has found this man-made material, free from possible contamination and batch variability.

Professor Denning, whose field is in cardiac stem cell research, said: "The field of regenerative medicine has snowballed in the last five years and over the coming five years a lot more patients will be receiving stem cell treatments. Clinical trials are still in the very early stages. However, with this kind of product, if we can get it commercialised and validated by the regulators it could be helping patients in two to three years."

Conditions of the heart, liver and brain are all under investigation as possible new stem cell treatments. People are already receiving stem cells derived eye cells for eye disorders.

These new materials have shown great promise in the laboratory. The research team now needs a commercial partner to test this lab based discovery on an industrial scale.

For more information go to:
http://onlinelibrary.wiley.com/doi/10.1002/adma.201501351/abstract

http://bit.ly/1GPaxQl

Semen has controlling power over female genes and behaviour

Semen says turn those genes on

THERE'S more to semen than sperm. In many animals, seminal fluid alters both the bodies and sometimes even the behaviour of females. Human semen, too, triggers changes in the uterus, and might have wider effects on women, aimed at just one goal.

"It's all about maximising the chances of the male reproducing," says Sarah Robertson of the University of Adelaide in Australia. The effects are most striking in fruit flies: seminal fluid can make the females eat more, lay more eggs and be less receptive to other males.

Now a team led by Tracey Chapman at the University of East Anglia in Norwich, UK, has found that male fruit flies selectively alter the chemical make-up of their seminal fluid. In the presence of rivals, the males produce more seminal proteins. "It came as a real surprise," says Chapman. "It's a sophisticated response to the social and sexual situation."

Some of their findings were presented at the Society for Molecular Biology and Evolution conference in Vienna, Austria, last week, including their discovery that one of these proteins is a "master regulator" of genes. Females exposed to it show a wide range of changes in gene expression.

Chapman thinks this kind of seminal signalling is widespread in the animal world. The semen of people, pigs and mice affects the female reproductive tract, and the question is whether it can also produce behavioural responses in female mammals similar to those seen in fruit flies.

There have been claims that semen can do everything from making women sleepy after sex to strengthening the emotional bond with their partner. One 2002 study, based on a survey of 300 students, even found that women whose partners did not use condoms scored lower on a measure of depression.

If that effect is real, depression in some people might be treatable with artificial-semen suppositories. Gordon Gallup of the State University of New York at Albany, who carried out the study, says a PhD student of his has replicated the finding in a survey of 1000 women, but the results were never published.

In flies, seminal proteins can directly affect behaviour because they enter the circulatory system, travelling throughout the body to the brain. "They rapidly get to many places in the female," Chapman says.

From the female's perspective, seminal signalling is usually nothing sinister. According to Chapman, it's an efficient way of getting a female's body ready to produce offspring as soon as possible.

It's not clear whether any components of human semen get into the bloodstream, but it could be possible, particularly for small molecules like hormones, says Robertson. She has shown that seminal fluid induces expression of a range of genes in the cervix, including ones that affect the immune system, ovulation, the receptivity of the uterus lining to an embryo, and even the growth of the embryo itself.

As for seminal signalling, she thinks it's more likely to be indirect, with semen causing the cervix to produce molecules that influence the rest of the body. Her team is studying the effect of three microRNAs – RNA fragments that affect gene expression – released by the cervix in response to semen.

Whatever the mechanism, both Chapman and Robertson say it's plausible that semen could have effects on women well beyond their reproductive tract.

http://www.bbc.com/news/health-33621109

The drug to slow Alzheimer's?

Talk to anyone affected by Alzheimer's and the need for a drug to slow the progression of the disease is clear.

By James Gallagher Health editor, BBC News website

Jeremy Cox is looking after his wife Roz who has been diagnosed with dementia.

"I think one of the things you've got to be aware of is, without a drug like this, what the situation is, it's desperate. "You just go on into the final stage of being spoon-fed baby-food in a care home."

The statistics back up the human stories too. Alzheimer's Research UK estimates that a treatment that could slow dementia progression by 25% would halve the number of people who reach the most debilitating severe form of the disease.

At the moment there is simply nothing to stop that happening. Current medication, such as Aricept, can manage only the symptoms of dementia by helping the dying brain cells function.

First hint

The modern history of dementia research has been unbelievably bleak. More than 100 trials in the past three decades have produced just a handful of drugs that manage symptoms and nothing to stop the death of the brain. Compare that to the phenomenal progress that had been made in heart disease or cancer.

That is why even the slightest hint of progress - for those in the mildest stage of Alzheimer's - is creating excitement. Provisional data suggests that the drug solanezumab may slow the progression of the disease by 34%.

The implication is that the amount of cognitive decline normally seen in 18 months would take 24 months with the drug - allowing patients to spend longer in the mild phase of the degenerative disease. Be in no doubt that it would be a hugely significant moment if such as drug was available.

However, today is not the day to jump up and down proclaiming a breakthrough in slowing the pace of Alzheimer's. The limited data which has been released is the scientific equivalent of a poll before a general election or a trailer ahead of a movie. It provides captivating clues, hints and teases, but nothing definitive.

Next year, when further trial results are due, we will know for certain whether solanezumab is the breakthrough everyone hopes it could be. But even if everything goes perfectly it could take years to reach patients as the drug is licensed and approved. It means people with Alzheimer's today are unlikely to benefit as the effect is seen only in those with the mildest stage of the disease.

http://bit.ly/1JFOehS

New Alzheimer's drugs: What do they do and could they be a cure?

Which drugs are the most exciting?

A flurry of exciting results from new drugs for Alzheimer's disease have been announced this week at the US Alzheimer's Association International Conference in Washington DC. New Scientist looks at the most promising treatments, and asks whether any of them could be a cure.

Which drugs are the most exciting?

Although other drugs are showing huge promise, an antibody called solanezumab has attracted the most attention. It flopped in an 18 month trial, but when this test was extended for a further two years, it was found that the brains and memories of people with Alzheimer's who were taking the drug deteriorated more slowly.

However, these latest results have been greeted with caution, triggering a fall instead of a rise in the company's share price, because the improvements in people taking the drug were only slight, and the full details of solanezumab's impact on Alzheimer's symptoms won't be known until another large trial is completed next year.

So why the fuss about solanezumab?

Those who started taking solanezumab earlier did better. In the earlier trial, half the participants took a placebo, but for the extension, all people were switched to solanezumab. The new results show that those who started on the drug right at the beginning outperformed those who had originally received the placebo.

"The placebo group never caught up, so the earlier you start treatment, the more effective it can be," says Maria Carrillo, chief science officer at the US Alzheimer's Association. But she notes that even in those who started taking solanezumab earlier, brain function still declined. "It's not the final word in treatment," she says.

Does solanezumab work in a special way?

Not really. Solanezumab is an antibody that targets beta amyloid, a brain protein that can coagulate into plaques that kill brain cells. While solanezumab can mop up this protein before it forms plaques, another antibody called aducanumab can destroy these beta amyloid plaques once they have formed.

Results presented in March by Biogen of Cambridge, Massachusetts showed that aducanumab also slows cognitive decline in people. But the data they presented this week in Washington DC shows there are side effects like brain swelling, headaches and small haemorrhages that have led some people to stop taking it. However, the company says it is confident it can address these problems and is preparing for a much larger five-year study.

What other drugs are out there?

One of the dark horses that emerged this week is a drug called azeliragon. Rather than attacking plaque, it reduces brain inflammation, a factor now firmly linked with development of Alzheimer's.

After 18 months on this drug, people rated their own symptoms as declining significantly less than those taking a placebo. "It's difficult to say how that would translate to a patient at home, but these are significant changes," says Carrillo.

The US Food and Drug Administration has been so impressed by results with this drug that it granted azeliragon "fast-track" status to a much larger trial in 800 patients which began in April, organised by vTv Therapeutics of High Point, North Carolina.

Another particularly promising drug, named NPT088 might treat not just Alzheimer's, but several brain disorders that involve types of plaque, including Parkinson's and Creutzfeldt-Jakob disease.

Unlike any other drug, it targets a common structural motif shared by different protein plaques, meaning that as well as breaking down beta amyloid plaques, it targets the other type of plaque found in Alzheimer's disease, which is made out of tau protein. NeuroPhage Pharmaceuticals of Cambridge, Massachusetts, has reported that this drug prevents memory loss in mice, and has announced plans to apply for permission to trial it in people.

Are we finally nearing a cure for Alzheimer's?

Set against the more than 100 trials for Alzheimer's drugs that have failed so far, all these latest developments represent real progress. But no single treatment is ever likely to halt or reverse the symptoms of the disease because it is caused by several factors, including genetics, lifestyle, poor diet or lack of exercise. Chronic inflammatory diseases such as diabetes and obesity are also a factor.

Ultimately, using several drugs together might prove to be the most effective, as is done in cancer and HIV treatment.

"Alzheimer's is very complicated and combination, multi-target therapies are much more likely to show promise modifying the disease process," says Peter Roberts of the University of Bristol, UK. "Aside from our pharmacological strategies, our 'epidemic' of dementia really needs much more investment in prevention, rather than treatment, by identifying risk factors for developing the disease."

http://bit.ly/1I2ymGe

Myth of pristine Amazon rainforest busted as old cities reappear

The first Europeans to penetrate the Amazon rainforests reported cities, roads and fertile fields along the banks of its major rivers.

"There was one town that stretched for 15 miles without any space from house to house, which was a marvellous thing to behold," wrote Gaspar de Carvajal, chronicler of explorer and conquistador Francisco de Orellana in 1542. "The land is as fertile and as normal in appearance as our Spain."

Such tales were long dismissed as fantasies, not least because teeming cities were never seen or talked about again. But it now seems the chroniclers were right all along. It is our modern vision of a pristine rainforest wilderness that turns out to be the dream.

What is today one of the largest tracts of rainforest in the world was, until little more than 500 years ago, a landscape dominated by human activity, according to a review of the evidence by Charles Clement of Brazil's National Institute of Amazonian Research in Manaus, and his colleagues.

After Europeans showed up, the inhabitants were decimated by disease and superior weaponry, and retreated into the bush, while the jungle reclaimed their fields and plazas. But, thanks to a combination of deforestation and remote sensing, what's left of their civilisation is now re-emerging.

They reveal an anthropogenically modified Amazonia before the European conquest. "Few if any pristine landscapes remained in 1492," says Clement. "Many present Amazon forests, while seemingly natural, are domesticated."

Amazon domesticity

The evidence for this radical rethink has been stacking up for some time. Archaeologists have uncovered dense urban centres that would have been home to up to 10,000 inhabitants along riverbanks, with fields and cultivated orchards of Brazil nuts, palm and fruit trees stretching for tens of kilometres. Remote sensing has revealed extensive earthworks, including cities, causeways, canals, graveyards and huge areas of ridged fields that kept crops like manioc, maize and squash clear of floods and frosts.

Meanwhile, agriculturalists have discovered that many forest soils have been mulched and composted with waste. These fertile "dark earths", or *terra preta*,

may cover 150,000 square kilometres, much of it now reclaimed by rainforests. Before the arrival of Europeans, the region's population may have reached 50 million.

The remains date back 3000 years or more, say the authors, who include geographer William Denevan of the University of Wisconsin-Madison, and anthropologist Michael Heckenberger of the University of Florida at Gainesville – both pioneers of the idea that the Amazon has long been modified by humans.

Not everyone agrees. Dolores Piperno of the Smithsonian Tropical Research Institute in Panama recently argued that "recent investigations of soils in parts of the western Amazon… found little vegetation disturbance".

Clement and his co-authors agree that "the idea of a domesticated Amazonia… contrasts strongly with reports of empty forests, which continue to captivate scientific and popular media".

But the idea of a domesticated Amazon complements research in other rainforest regions, including the Congo basin and South-East Asia, that also suggest that much of what seems pristine is actually regrowth after dense human occupation.

Erle Ellis of the University of Maryland, Baltimore, says such evidence suggests that we should be dating the start of the Anthropocene – the era of human domination of the planet – to thousands of years ago rather than in the middle of 20th century.

Journal reference: Proceedings of the Royal Society B, DOI: 10.1098/rspb.2015.0813

http://www.eurekalert.org/pub_releases/2015-07/vt-soj072315.php

Small oxygen jump helped enable early animals take first breaths

Discovery in rocks shows extent that atmospheric oxygen helped give rise to complex life

If oxygen was a driver of the early evolution of animals, only a slight bump in oxygen levels facilitated it, according to a multi-institutional research team that includes a Virginia Tech geoscientist.

The discovery, published Wednesday in the journal Nature, calls into question the long held theory that a dramatic change in oxygen levels might have been responsible for the appearance of complicated life forms like whales, sharks, and squids evolving from less complicated life forms, such as microorganisms, algae, and sponges.

The researchers discovered oxygen levels rose in the water and atmosphere, but at lower levels than was thought necessary to trigger life changes.

"We suggest that about 635 million to 542 million years ago, the Earth passed some low, but critical, threshold in oxygenation for animals," said Benjamin Gill, an assistant professor of geoscience in the College of Science. "That threshold was in the range of a 10 to 40 percent increase, and was the second time in Earth's history that oxygen levels significantly rose."

The scientists estimated oxygen levels by analyzing iron found in shale rock, which was once mud on ancient seafloors. The location and amounts of iron in the rock gave important clues about ancient ocean water chemistries over time.

Rock data from across the world were collected by the research team, analyzed, compiled, and statistically modeled.

Many organisms on Earth, including animals, need oxygen to produce energy and perform other life functions.

"Going forward we will need much more precise constraints on the magnitude of oxygenation and the physiological requirements of early animals to continue testing the impact of oxygenation on Cambrian animal life," said Erik Sperling, an assistant professor of geological and environmental sciences at Stanford University, and first author on the paper.

http://www.eurekalert.org/pub_releases/2015-07/tl-tln072215.php

The Lancet: New studies show that 2 classes of inexpensive generic drugs can reduce breast cancer deaths

aromatase inhibitors and bisphosphonates, can improve survival prospects for postmenopausal women with early breast cancer

Two new studies, both published in The Lancet, suggest that two different classes of drugs, aromatase inhibitors (AIs) and bisphosphonates, can each improve survival prospects for postmenopausal women with early breast cancer. Moreover, the researchers suggest that the two types of drug can be used together, increasing the benefits while also decreasing some side-effects.

Most women are post-menopausal when they develop breast cancer, and breast cancer is usually found early, when surgery can remove all detectable disease, but might leave dangerous undetected micrometastases (small secondary tumours). About 80% of breast cancers are hormone sensitive (ER-positive), which means that they can be stimulated by the body's own hormones, such as oestrogen. Endocrine treatments, which act to stop hormones stimulating cancer cells, can help protect against breast cancer recurrence.

The Early Breast Cancer Trialists' Collaborative Group (EBCTCG) is a worldwide collaboration set up 30 years ago by researchers at the University of Oxford, Oxford, UK to bring together every few years all the evidence from randomised trials on the treatment of early breast cancer. The two reports published today [Friday 24 July, 2015] provide the best evidence yet for the effects of AIs and bisphosphonates on postmenopausal women with early breast cancer.

The first study brings together evidence from 30 000 postmenopausal women in 9 randomised trials, showing that 5 years of treatment with the newer endocrine therapy (ie, an AI) produces somewhat better survival than five years of standard endocrine therapy (tamoxifen). Compared to tamoxifen, taking AIs for five years further reduced the likelihood of the cancer recurring by about a third (30%), and the risk of dying from breast cancer by around 15% throughout the decade after beginning treatment. The researchers estimate that, compared to no endocrine treatment, the risk of dying from breast cancer for women who took AIs would be reduced by around 40% in the decade after beginning treatment.

According to the AI study's lead author, Professor Mitch Dowsett of The Royal Marsden and The Institute of Cancer Research, London, UK, "Our global collaboration has revealed that the risk of postmenopausal women with the most common form of breast cancer dying of their disease is reduced by 40 per cent by taking five years of an aromatase inhibitor - a significantly greater protection than that offered by tamoxifen. The impact of aromatase inhibitors is particularly remarkable given how specific these drugs are - removing only the tiny amount of oestrogen that remains in the circulation of women after the menopause - and given the extraordinary molecular differences between ER-positive tumours. But aromatase inhibitor treatment is not free of side-effects, and it's important to ensure that women with significant side-effects are supported to try to continue to take treatment and fully benefit from it."

The second study brings together evidence from another 20 000 women in 26 randomised trials, showing that 2-5 years of treatment with a class of drugs called bisphosphonates, which are usually used to treat osteoporosis, reduces the risk of breast cancer recurring in post-menopausal women, and also significantly extends survival. However, bisphosphonate treatment appears to have little effect in premenopausal women.

The most common site for breast cancers to spread to is bone. Tumour cells released from the primary breast cancer can remain dormant in the bone for years before spreading to other parts of the body. Bisphosphonates alter the bone microenvironment, which could make it less favourable for cancer cells and so reduce the risk of cancer recurrence in the bone and in other organs. Taken separately, previous clinical trials of bisphosphonates in early breast cancer have shown mixed results, but taking all their results together, a clearer picture emerges. The meta-analysis included individual patient data on 18 766 women in 26 randomised trials, comparing between two and five years of bisphosphonates versus no bisphosphonate. In the overall study population, the only clear benefit of bisphosphonates was a 17% reduction in recurrence of cancer in the bone. However, among postmenopausal women, bisphosphonate treatment produced a larger reduction in bone recurrence of 28% and also reduced the risk of dying from breast cancer by 18% during the first decade after diagnosis [2].

This benefit appeared to be irrespective of the type of bisphosphonate, treatment duration, how big the tumour was, whether it had spread to the lymph nodes, or whether or not it was oestrogen-receptor (ER) positive. However, bisphosphonate treatment did not reduce the risk of new breast cancers developing in the opposite breast.

According to the bisphosphonate study's lead author Professor Robert Coleman, from the University of Sheffield, UK, "Currently, bisphosphonates are mainly used to reduce bone loss and fractures in postmenopausal women and to reduce bone complications in advanced cancer patients. Our results show that adjuvant bisphosphonates in postmenopausal women prevent around a quarter of bone recurrences and one in six of all breast cancer deaths in the first decade of treatment. These simple, well tolerated treatments should now be considered for routine use in the treatment of early breast cancer in women with either a natural or medically induced menopause to both extend survival and reduce the adverse effects of cancer treatments such as the aromatase inhibitors on bone health." [1]

Professor Richard Gray, from the University of Oxford, UK, who was the lead statistician for both studies, comments that, "These studies provide really good evidence that both of these inexpensive, generic drugs can help to reduce breast cancer mortality in postmenopausal women. About two-thirds of all women with breast cancer are postmenopausal with hormone-sensitive tumours, so could potentially benefit from both drugs. The drugs are complementary, because the main side effect of aromatase inhibitors is an increase in bone loss and fractures, while bisphosphonates reduce bone loss and fractures as well as improving survival."

Both studies were funded by Cancer Research UK and the UK Medical Research Council.

[1] Quotes direct from authors and cannot be found in text of Article.

[2] The absolute reduction in the risk of death from breast cancer at 10 years was 3·3% with the use of bisphosphonates (10-year risk 14·7% vs 18·0% for women who did not receive bisphosphonates).

http://www.eurekalert.org/pub_releases/2015-07/p-ww071615.php

Why West Nile virus is more dangerous in the elderly

West Nile virus (WNV) is particularly dangerous in older people, who account for a large number of severe cases and deaths caused by the virus.

WNV infection turns serious when the virus crosses the blood-brain-barrier and wreaks havoc among nerve cells in the brain. A study published on July 23rd in PLOS Pathogens suggests that several critical components of the early immune

response to the virus are impaired in elderly individuals, and that this can explain their vulnerability.

Michael Diamond, from Washington University in Saint Louis, USA, and colleagues analyzed and compared the immune response to WNV infection in four-month-old (the equivalent of young adults) and 18-month-old (elderly) mice. The older mice were more than three times as likely to die after WNV infection. When the researchers measured the amount of virus present in different tissues, they found that, in addition to more virus in their blood and spleens, the older mice had 20-fold higher virus levels in their brains--which likely causes the excess deaths.

Following transmission by mosquitoes, the early specific (also called adaptive) immune response to WNV is thought to be dominated by antibodies, and, consistent with this, the researchers found that older mice had less potent WNV-specific antibody responses during the early phase of infection. They also had weaker long-term antibody memory responses.

Antibody responses are initiated in lymph nodes close to the site of initial infection (so-called draining lymph nodes, or DLNs), where antigen-presenting cells, helper T cells, and antibody-producing B cells migrate to and interact to form so-called "germinal centers" and produce a highly specific antibody response. In the older mice, the researchers found, germinal center formation was delayed, consistent with the blunted early antibody response.

Analyzing the DLNs in more details, they found that fewer helper T cells were present, suggesting that these cells from older mice are less capable of "trafficking" to the lymph nodes. Experiments in which the researchers transplanted helper T cells from young adults or older mice into young adult recipients and then followed them by live microscopy (the paper contains several movies of these experiments) showed that this was due to reduced migratory ability of the helper T cells themselves.

Besides the reduced numbers of helper T cells in the DLNs, the researchers also found that the lymph node environment in older mice contained lower levels of immune stimulators (so-called chemokines) and therefore was less capable of attracting other immune cells necessary for germinal center formation.

While the observed differences of the individual steps were mostly modest, mathematical modeling suggested that even small delays in the trafficking of these immune cells will lead to reduced initiation of a WNV-specific antibody-response during the early stages after infection. This can lead to substantially higher early viral loads, which in turn can increase the chance of the infection spreading to the brain and worsen clinical outcome.

The authors conclude that their study "identifies a series of key early defects associated with immune responses in old animals." Regarding the mechanisms, they say "the delayed antibody and germinal center cell responses are due to trafficking defects, which are compounded by lower levels of chemokines in the lymph node after infection. Ultimately, this leads to blunted adaptive immune responses, higher viral titers, and increased death after West Nile virus infection."

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http://www.eurekalert.org/pub_releases/2015-07/aaft-wko072015.php

What killed off the megafauna?

Strong case for climate change as the key driver of megafaunal extinctions

This news release is available in Japanese.

Rapid phases of warming climate played a greater role in the extinction of megafauna in the Late Pleistocene than did human activity, a new study shows. The study helps to inform the debate about what killed off megafaunal species (or animals over 100 pounds) during the last glacial period - a subject that is highly debated, with some scientists pointing to human hunting and land alteration, and others to climate change.

Progress on the debate has been hindered by reliance on fossil evidence in lieu of studies of ancient DNA, which could shed more light on the timing of major animal population changes, like migration or extinction events. Here, to parse out the roles for human activity or changing climate in the Late Pleistocene megafaunal extinction, Alan Cooper and colleagues used a combination of ancient DNA and detailed paleoclimate data.

They evaluated DNA from megafaunal species, looking back over more than 50,000 years of DNA records for extinction events. The researchers compared information on megafauna extinctions to records of severe climate events in the

Late Pleistocene obtained through Greenland ice cores and other sources. They report a close relationship between Pleistocene megafaunal extinction events and the rapid warming events at the start of so-called interstadial periods (or regularly recurring warm phases). They note that the unique megafauna population structures that resulted from climate change events could have been more susceptible to human impact. Their analysis ultimately strengthens the case for climate change as the key driver of megafaunal extinctions, with human impacts playing a subsidiary role.

The reports by Alan Cooper et al. and Maanasa Raghavan et al. are related to a special package from Science's News department on ancient DNA, the study of which has already lead to breathtaking finds -- including the entire genomes of Neandertals and other kinds of ancient humans. Until recently, extracting and studying ancient DNA was so difficult that it was limited to just a few sophisticated labs. Now, writes Science Deputy News Editor Elizabeth Culotta, techniques for studying ancient DNA are more accessible, being applied widely and broadly to explore an array of questions, and catapulting paleogenetics into a golden era.

In one article in this package, contributing correspondent Ann Gibbons explores how studies of ancient genetic material are prompting scientists to rethink long-held views of human prehistory; for example, ancient DNA has led to the discovery of new types of ancient humans and revealed interbreeding between our ancestors and our archaic cousins.

Writing from Mexico City, news writer Lizzie Wade highlights the push to acquire ancient DNA from hot and humid locales, where much of the world's biodiversity evolved. Samples from such regions (instead of from frigid ones, a more typical source of ancient DNA) could solve myriad controversies, like the origins of the large animals that once dominated South America and Australia.

Looking to the future of the field, news writer Robert Service explores how researchers are using ancient protein, which has some advantages over ancient DNA, to uncover the diets and lifestyles of past cultures and diagnose infection in ancient specimens. The full package includes six articles written by various members of Science's News department and edited by Culotta.

Article #24: "Abrupt warming events drove Late Pleistocene Holarctic megafaunal turnover," by A. Cooper; B.W. Brook; C.J.A. Bradshaw at University of Adelaide in Adelaide, SA, Australia; C. Turney at University of New South Wales in Sydney, NSW, Australia; K.A. Hughen at Woods Hole Oceanographic Institution in Woods Hole, MA; B.W. Brook at University of Tasmania in Hobart, TAS, Australia; H.G. McDonald at National Parks Service in Fort Collins, CO.

http://www.eurekalert.org/pub_releases/2015-07/cu-sis072315.php

Scientists identify schizophrenia's 'Rosetta Stone' gene

Breakthrough reveals gene's influence in a vulnerable period of the brain's development

Scientists have identified a critical function of what they believe to be schizophrenia's "Rosetta Stone" gene that could hold the key to decoding the function of all genes involved in the disease. The breakthrough has revealed a vulnerable period in the early stages of the brain's development that researchers hope can be targeted for future efforts in reversing schizophrenia.

In a paper published today in the journal Science, neuroscientists from Cardiff University describe having uncovered the previously unknown influence of a gene in ensuring healthy brain development.

The gene is known as 'disrupted in schizophrenia-1' (DISC-1). Past studies have shown that when mutated, the gene is a high risk factor for mental illness including schizophrenia, major clinical depression and bipolar disorder.

The aim of this latest study was to determine whether DISC-1's interactions with other proteins, early on in the brain's development, had a bearing on the brain's ability to adapt its structure and function (also known as 'plasticity') later on in adulthood. Many genes responsible for the creation of synaptic proteins have previously shown to be strongly linked to schizophrenia and other brain disorders, but until now the reasons have not been understood.

The team, led by Professor Kevin Fox from Cardiff University's School of Biosciences, found that in order for healthy development of the brain's synapses to take place, the DISC-1 gene first needs to bind with two other molecules known as 'Lis' and 'Nudel'.

Their experiments in mice revealed that by preventing DISC-1 from binding with these molecules - using a protein-releasing drug called Tamoxifen at an early stage of the brain's development - it would lack plasticity once it grows to its adult state, preventing cells (cortical neurons) in the brain's largest region from being able to form synapses. The ability to form coherent thoughts and to properly perceive the world is damaged as a consequence of this.

Preventing DISC-1 from binding with 'Lis' and 'Nudel' molecules, when the brain was fully formed, showed no effect on its plasticity. However, the researchers were able to pinpoint a seven-day window early on in the brain's development - one week after birth - where failure to bind had an irreversible effect on the brain's plasticity later on in life.

"We believe that DISC-1 is schizophrenia's Rosetta Stone gene and could hold the master key to help us unlock our understanding of the role played by all risk genes involved in the disease," said Professor Fox.

"The potential of what we now know about this gene is immense. We have identified a critical period during brain development that directs us to test whether other schizophrenia risk genes affecting different regions of the brain create their malfunction during their own critical period.

"The challenge ahead lies in finding a way of treating people during this critical period or in finding ways of reversing the problem during adulthood by returning plasticity to the brain. This, we hope, could one day help to prevent the manifestation or recurrence of schizophrenia symptoms altogether."

Professor Jeremy Hall, an academic mental health clinician and director of Cardiff University's Neuroscience and Mental Health Research Institute, said:

"This paper provides strong experimental evidence that subtle changes early on in life can lead to much bigger effects in adulthood. This helps explain how early life events can increase the risk of adult mental health disorders like schizophrenia."

Schizophrenia affects around 1% of the global population and an estimated 635,000 people in the UK will at some stage in their lives be affected by the condition. The projected cost of schizophrenia to society is around £11.8 billion a year.

The symptoms of schizophrenia can be extremely disruptive, and have a large impact on a person's ability to carry out everyday tasks, such as going to work, maintaining relationships and caring for themselves or others.

The research was funded by the Medical Research Council (MRC) and the National Institute of Health (NIH).

http://www.eurekalert.org/pub_releases/2015-07/aaft-ffs072015.php

Four-legged fossil suggests snakes evolved from burrowing ancestors

The discovery of a four-legged fossil of a snake hints that this suborder may have evolved from burrowing, rather than marine, ancestors.

This news release is available in Japanese.

The unique four-legged specimen, found in Brazil's Crato Formation, provides us with more insight into how these creatures transitioned into the sleek, slithering reptiles that we are familiar with - and often fearful of - today.

By analyzing both the genetics and the morphological features of this species compared to other known snake species, and giving different weight to each factor in four separate analyses, the authors determined that the four-legged creature is in fact an ancestor of modern-day snakes.

The newly discovered species Tetrapodophis amplectus, which lived during the Early Cretaceous 146 to 100 million years ago, maintains many classic snake features, such as a short snout, long braincase, elongated body, scales, fanged teeth and a flexible jaw to swallow large prey. It also maintains the typical vertebrae structure seen in modern-day snakes that allows for the extreme flexibility required to constrict prey. The main, glaring difference is Tetrapodophis's four limbs, which do not appear to have been used for locomotion. Rather, the shorter exterior digits and lengthened second digit suggest that the limbs were used for grasping, the authors say, either to seize prey or to clasp during mating. The authors also note that the specimen lacks the long, laterally compressed tail typically found in aquatic animals, further suggesting that snakes did not evolve from marine ancestors.

Tetrapodophis amplectus hands are pictured. **Dave Martill, University of Portsmouth**

Thus, this intriguing fossil hints at how snakes eventually slithered their way into the modern world. A Perspective by Susan Evans discusses the fossil in more detail.

Article #14: "A four-legged snake from the Early Cretaceous of Gondwana," by D.M. Martill at University of Portsmouth in Portsmouth, UK; H. Tischlinger in Stammham, Germany; N.R. Longrich at University of Bath in Bath, UK.

http://bit.ly/1S3PIwd

Root Beer Is For Adults Again

This is not your soda fountain's root beer

By Helen Thompson smithsonian.com

In recent years, root beer has been relegated to birthday parties and floats, but it's not just for kids anymore. Hard root beer combines the bubbling sweetness of a classic soda and the alcoholic kick of a more traditional brew, Kyle Stock reports for *Bloomberg Business*. That's a recipe that breweries small and large are betting on.

The new "it" brew isn't all that new though. Root beer traces its roots to colonial times. "It was a popular drink in the 18th century before falling out of favor and virtually disappearing," Samantha Christmann notes for *Buffalo News*.

The modern trend seems to have started with Small Town Brewery in Wauconda, Illinois. They've been producing Not Your Father's Root Beer since 2013, Stock notes. It's brewed like regular beer with root beer-like seasoning—sassafras root, vanilla and other spices.

The popularity of the product garnered the attention of larger operations that could take it nationwide, writes Stock. Earlier this year, Small Town Brewery inked a distribution deal with Pabst and sold the beer to a group of investors outright.

Small Town has two more root beer varieties in the works, they say. Chicago's Berghoff Beer recently released Rowdy Root Beer. Sprecher Brewing in Glendale, Wisconsin, makes an alcoholic version of their popular root beer soda. Coney Island Beer, a subsidiary of Boston Brewing (maker of Sam Adams), also produces a hard root beer, points out Ethan Lascity for *International Business Times*.

Craft brewing is a crowded business, but the appeal of hard root beer makes a lot of sense, explains Stock. For those who don't like stronger, hoppier brews, it's a sweet alternative. It also mixes well with some liquors (and probably ice cream). But, more than anything, everybody knows what root beer tastes like, so the hard version could conjure both a sense of novelty and nostalgia.

http://www.bbc.com/news/science-environment-33641648

'Earth 2.0' found in Nasa Kepler telescope haul

A haul of planets from Nasa's Kepler telescope includes a world sharing many characteristics with Earth.

By Paul Rincon Science editor, BBC News website

Kepler-452b orbits at a very similar distance from its star, though its radius is 60% larger. Mission scientists said they believed it was the most Earth-like planet yet. Such worlds are of interest to astronomers because they might be small and cool enough to host liquid water on their surface - and might therefore be hospitable to life.

Nasa's science chief John Grunsfeld called the new world "Earth 2.0" and the "closest so far" to our home. It is around 1,400 light years away from Earth.

John Jenkins, Kepler data analysis lead at Nasa's Ames Research Center in California, added: "It's a real privilege to deliver this news to you today. There's a new kid on the block that's just moved in next door." The new world joins other exoplanets such as Kepler-186f that are similar in many ways to Earth.

Determining which is most Earth-like depends on the properties one considers. Kepler-186f, announced in 2014, is smaller than the new planet, but orbits a red dwarf star that is significantly cooler than our own.

Kepler-452b, however, orbits a parent star which belongs to the same class as the Sun: it is just 4% more massive and 10% brighter. Kepler-452b takes 385 days to complete a full circuit of this star, so its orbital period is 5% longer than Earth's.

The mass of Kepler-452b cannot be measured yet, so astronomers have to rely on models to estimate a range of possible masses, with the most likely being five times that of Earth. If it is rocky, the world would likely still have active volcanism and its gravity could be roughly twice that on our own planet.

The new world is included in a haul of 500 new possible planets sighted by the Kepler space telescope around distant stars.

Twelve of the new candidates are less than twice Earth's diameter, orbiting in the so-called habitable zone around their star. This zone refers to a range of distances at which the energy radiated by the star would permit water to exist as a liquid on the planet's surface if certain other conditions are also met.

Of these 500 candidates, Kepler-452b is the first to be confirmed as a planet.

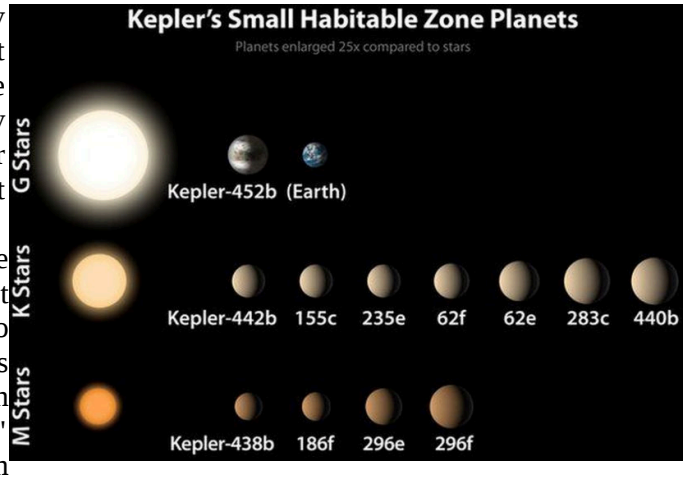
Dr Suzanne Aigrain, from the University of Oxford, who was not involved with the study, told BBC News: "I do believe the properties described for Kepler-452b are the most Earth-like I've come across for a confirmed planet to date.

"What seems even more significant to me is the number of planets in the habitable zone of their host stars with radii below two Earth radii; 12 is quite a few compared to the pre-existing Kepler planet catalogue.

"It bodes well for their attempts to provide a more robust measure of the incidence of Earth-like planets, which is the top-level goal of the Kepler mission."

While similar in size and brightness to the Sun, Kepler-452b's host star is 1.5 billion years older than ours. Scientists working on the mission therefore believe it could point to a possible future for the Earth. "If Kepler-452b is indeed a rocky planet, its location vis-a-vis its star could mean that it is just entering a runaway greenhouse phase of its climate history," explained Dr Doug Caldwell, a Seti Institute scientist working on the Kepler mission.

"The increasing energy from its aging sun might be heating the surface and evaporating any oceans. The water vapour would be lost from the planet forever."



"Kepler-452b could be experiencing now what the Earth will undergo more than a billion years from now, as the Sun ages and grows brighter."

Dr Don Pollacco, from Warwick University, UK, who was not involved with the latest analysis, told the BBC: "Kepler data allows you to estimate the relative size of a planet to its host star, so if you know the size of the host, hey presto, you know the size of the planet. "However, to go further - i.e. is it rocky? - involves measuring the mass of the planets and this is much more difficult to do as the stars are too far away for these measurements (which are incredibly difficult) to make.

"So in reality they have no idea what this planet is made of: It could be rock but it could be a small gassy ball or something more exotic maybe."

Dr Chris Watson, from Queen's University Belfast, UK, commented: "Other Kepler habitable zone planets may well be more Earth-like in this respect. For example, Kepler-186f is approximately 1.17 Earth radii, and Kepler-438b is approximately 1.12 Earth radii. "In fact, at 1.6 Earth radii, this would place Kepler-452b in a category of planet called a 'Super-Earth' - our Solar System does not actually have any planet of this type within it! Super-Earths are hugely interesting for this reason, but one might then say, well, is it really 'Earth-like' given all this?"

He added: "When we look at the type of star Kepler-452b orbits, then it seems to be a star not too dissimilar to our Sun... The other Kepler habitable zone planets that have been discovered so far tend to be orbiting M-dwarfs - stars far cooler than our Sun, and therefore the planets need to orbit much closer to receive the same levels of heating. "So it may be a *potentially* rocky super-Earth in an Earth-like orbit (in terms of host star and orbital distance). It's this combination of the host star and orbit that set it apart in my opinion."

The findings have been accepted for publication in The Astronomical Journal.

http://nyti.ms/1Ms5BrL

Fat Sense: Scientists Show We Have a Distinct Taste for Fat

Move over sweet and salty: Researchers say we have a distinct and basic taste for fat, too.

WASHINGTON - But it's nowhere near as delicious as it sounds.

They propose expanding our taste palate to include fat along with sweet, salty, bitter, sour and relative newcomer umami.

A research team at Purdue University tested look-alike mixtures with different tastes. More than half of the 28 special tasters could distinguish fatty acids from the other tastes, according to a study published in the journal Chemical Senses.

Past research showed fat had a distinct feel in the mouth, but scientists removed texture and smell clues and people could still tell the difference.

"The fatty acid part of taste is very unpleasant," study author Richard Mattes, a Purdue nutrition science professor, said Thursday. "I haven't met anybody who likes it alone. You usually get a gag reflex."

Stinky cheese has high levels of the fat taste and so does food that goes rancid, Mattes said. Yet we like it because it mixes well and brings out the best of other flavors, just like the bitter in coffee or chocolate, he added.

To qualify as a basic taste, a flavor has to have unique chemical signature, have specific receptors in our bodies for the taste, and people have to distinguish it from other tastes. Scientists had found the chemical signature and two specific receptors for fat, but showing that people could distinguish it was the sticky point.

Initially Mattes found that people couldn't quite tell fat tastes when given a broad array of flavors. But when just given yucky tastes — bitter, umami, sour — they could find the fat.

The team started out with 54 people, but concentrated on the results from 28 who were better tasters in general.

Mattes and colleagues proposed calling the taste "oleogustus" (Oh-leo-GUS'-tus) after Latin for fat taste. There is no single scientific authority that names senses.

Robin Dando, a Cornell University food scientist who wasn't part of the research, praised the study as "a pretty strong piece of evidence" for a basic fat taste, but didn't like the suggested name — preferring to just call it fat.

Journal Chemical Senses: *http://chemse.oxfordjournals.org/*

http://www.eurekalert.org/pub_releases/2015-07/b-cf-tfs072415.php

Toxin from salmonid fish has potential to treat cancer

Researchers from the University of Freiburg decode molecular mechanism of fish pathogen

Pathogenic bacteria develop killer machines that work very specifically and highly efficiently. Scientists from the University of Freiburg have solved the molecular mechanism of a fish toxin that could be used in the future as a medication to treat cancer. The scientists have now published their research in the journal Nature Communications.

The Yersinia species of pathogens can cause the bubonic plague and serious gastrointestinal infections in humans. The pharmacologist Dr. Thomas Jank and his fellow researchers in the research group led by Prof. Dr. Dr. Klaus Aktories at the University of Freiburg studied a pathogen of the Yersinia family (Yersinia ruckeri). This pathogen causes redmouth disease in Salmonidae, which includes salmon and trout, resulting in large financial losses in the fish industry. The research group was able to identify a toxin injection machine in the Y. ruckeri genome. The structure of this machine resembles that of viruses that normally attack bacteria. The group demonstrated that the toxin Afp18 in this injection machine is an enzyme that deactivates the switch protein RhoA. RhoA is responsible for many vital processes in the cells of humans and fish. For example, it controls the building up and breaking down of actin filaments. These filaments are not only necessary for cell division, but also for the spreading of tumour metastases in the body.

In close collaboration with the developmental biologist Prof. Dr. Wolfgang Driever, also from the University of Freiburg, the research group injected the toxin Afp18 into zebra fish embryos. The result was that cell division was blocked,

and the fish embryos did not develop. The toxin caused the actin filaments in the fish cells to collapse. This is because the Afp18 attaches a sugar molecule, an N-acetylglucosamine, onto the amino acid tyrosine in RhoA. According to the scientists, this is a very unusual reaction in nature. The team was able to shed light on this mechanism at the atomic level through the X-ray analysis of Afp18-modified RhoA crystals. For this, they collaborated with Prof. Dr. Daan von Aalten from the University of Dundee, Scotland. Rho-regulatory proteins are involved in the growth of cancer, especially metastasis. For this reason, the researchers from the University of Freiburg believe that this fish toxin has great therapeutic potential in cancer treatment.

Thomas Jank and Klaus Aktories are researchers at the Institute of Experimental and Clinical Pharmacology and Toxicology at the University of Freiburg. Wolfgang Driever is the head of the Department of Developmental Biology of the Institute of Biology I, also at the University of Freiburg. Both Aktories and Driever are members of the cluster of excellence BIOSS Centre for Biological Signalling Studies.

Original publication:

Thomas Jank, Stephanie Eckerle*, Marcus Steinemann*, Christoph Trillhaase, Marianne Schimpl, Sebastian Wiese, Daan M.F. van Aalten, Wolfgang Driever & Klaus Aktories, "Tyrosine glycosylation of Rho by Yersinia toxin impairs blastomere cell behaviour in zebrafish embryos." Nature Communications 2015.*

http://bit.ly/1DGRJCH

Brain-Eating Amoebas May Kill You With Help from Your Own Immune System

The amoeba's presence in the brain triggers swelling that may do more harm than good

By Helen Thompson

In the grand scheme of things that can kill you, Naegleria fowleri sounds pretty frightening. When it finds itself in a swimmer's nose, this freshwater amoeba wiggles its way up the olfactory nerve to the brain. There, it starts destroying brain tissue. But, as Laura Sanders reports for Science News, the brain eating might not actually be the thing that kills you when you get an N. fowleri infection.

Stomach acid is deadly to the amoeba, so the nose is the its only a shot at a successful colonization of its host. Upon entering the brain N. fowleri sets off alarm signals in the body's immune system, explains Sanders. This triggers inflammation, which is what causes the brain to swell, and this may pave the way for the pathogen's destruction. The first signs of infection seem fairly innocuous — headache, nausea and fever — but more severe symptoms follow, including hallucinations, seizures and brain swelling.

And it's that immune reaction and brain swelling that might actually be the real killer here. In fact, Abdul Mannan Baig, a physiologist at Aga Khan University in Pakistan, reported in the journal Acta Tropica that the amoeba takes hours longer to destroy brain cells in the absence of immune cells, writes Sanders.

Here's what Baig thinks is going on: The swelling disrupts the blood brain barrier — the system that lets things in and out of the brain — and actually causes brain damage. At the same time, the amoeba releases enzymes and toxins that make that brain damage worse and ultimately irreversible.

Cases of N. fowleri are rare but predominantly fatal. In 2013, a 12-year-old girl became the first survivor in decades. Doctors approached her case with a focus on reducing brain swelling, and if Baig is right, that could explain why it worked.

http://bit.ly/1CZehUF

Leading climate scientist: Future is bleaker than we thought

Highly speculative. Full of conjecture. Based on flimsy evidence. Not supported by mainstream science. Not peer reviewed. Not suitable for basing policy on.

It sounds like climate scientists are talking about the claims of climate deniers. But this time they are talking about a 23 July discussion paper by James Hansen, the most famous and respected climate scientist on the planet.

In it, Hansen starts by arguing that the ice melting on and around Greenland and Antarctica will cause rises in sea level that are much faster than mainstream predictions, meaning that we are likely to see several metres of sea level rise this century. It is an argument he has been making for a long time: for instance in his 2007 feature for *New Scientist*.

Even more startling are the consequences that Hansen thinks will result from this rapid melt. Because fresh water is less dense than saltwater, the cold, fresh meltwater will pool around the coasts of Greenland and Antarctica.

Water blanket

Around Antarctica, this surface layer will act as a blanket, floating on top of warmer, saltier water and preventing it from losing heat to the air. Instead, this heat will go into melting the underside of ice shelves and glaciers. Hansen argues that the growth in sea ice around Antarctica is a sign that this is starting to happen already, with freshening surface water forming sea ice more readily.

This freshwater layer will also shut down the ocean currents that carry heat from the tropics to the poles, so the tropics will warm fast while high latitudes cool down because of the cold surface waters. This resulting temperature difference, Hansen claims, will power superstorms of a size and fury unlike anything we have ever seen.

Such superstorms occurred towards the end of the last interglacial period 120,000 years ago, the paper claims. It details several lines of evidence suggesting that the islands of the Bahamas were frequently pounded by massive waves at this time. For instance, there are wave-formed ridges many kilometres long on the

islands, and wave deposits up to 40 metres above current sea level, including massive boulders weighing thousands of tonnes.

Most terrifying of all, Hansen thinks that all of this could happen with just a 2 °C rise in temperature – the supposedly safe limit.

The consequences, of course, would be catastrophic. "It is not difficult to imagine that conflicts arising from forced migrations and economic collapse might make the planet ungovernable, threatening the fabric of civilisation," the paper states.

Far from conclusive

These claims certainly do not reflect the views of most of climate scientists, and the various lines of evidence presented in the paper are far from conclusive. Here's the take-home message, however: we cannot be sure that Hansen is wrong.

When it comes to sea level, just about every glaciologist now agrees that we are heading for massive sea level rises of at least 5 metres. The only contentious issue is how fast this will happen.

The speed question cannot be definitively resolved by studying how fast ice sheets melted in past interglacial periods because the planet has never warmed as fast as it is now. Nor can it be settled by ice models because we have no way of confirming whether they are right about the rate of melting.

There is also wide agreement that large-scale melting of Greenland's ice will shut down ocean circulation. Again, the main contentious issue is how soon it might happen – and there are hints that it is already happening.

There is certainly no agreement about the superstorms that Hansen predicts. But his argument is based on simple physics: winter storms are driven by the temperature difference between the poles and tropics, so if this difference temporarily increases due to massive ice melt, there will be a period of stronger storms.

The fact is that many of the consequences of rising greenhouse gases are extremely difficult to predict. We can be pretty confident about how much the planet will warm and how much the sea level will rise because there is plenty of evidence from the past, but beyond this there are huge gaps in our knowledge. How will plants respond? How many species will go extinct? How will food production be affected? These questions are almost impossible to answer, not least because the answers depend on us.

Unpleasant surprises

There have already been surprises. There is growing evidence, for instance, that much of the extreme weather around the planet in recent years is a result of changes in the behaviour of the jet stream as the poles warm. No one predicted this.

Indeed, the "official" projections of climate scientists have turned out to be too conservative time and time again. Antarctic melting is already a century ahead of schedule. Estimates of sea level rise by the Intergovernmental Panel on Climate Change are going up with every report. Hansen, by contrast, has a history of making predictions that turn out to be bang on the money.

That does not mean he is right again. But the mere possibility that he might be should make us all pause for thought. We are still gambling that we can get away with continuing business as usual without reaping the consequences in our lifetimes. It's a high-stakes gamble that could go horrifically wrong.

http://nyti.ms/1SILqOx

When the Cat Comes Back, With Prey

You can't pick and choose a cat's prey

By JAN HOFFMANJ ULY 24, 2015

Jennifer L. McDonald is an ecologist by profession and a cat person by avocation. Some years ago, Tiggy, her ginger-and-white shorthair, would bring home freshly killed mice and shrews for her consideration.

Dr. McDonald, now an associate research fellow at the Center for Ecology and Conservation at the University of Exeter in England, was curious about the impact of pet cats like Tiggy on wildlife. Fewer mice might be nice. But cats, natural hunters, pounce on birds and rabbits, too.

"You can't pick and choose a cat's prey," Dr. McDonald said. If owners realized how much prey their pets killed, she wondered, would they be willing to contain their cats to protect wildlife?

She and her associates studied the question. The answer, published recently in the journal Ecology and Evolution was unequivocal and emphatic.

No.

In recent years, debates about the predatory effect of cats on wildlife, particularly endangered songbirds, have only intensified. But most public opinion surveys have focused on the management of feral cats, which make up the majority of domestic feline marauders, particularly in the United States.

Dr. McDonald surveyed owners in two British villages about cats they allowed to roam outdoors. Owners were asked to predict the amount of prey taken by their cats and document the actual killings. Owners in one village were then asked whether they believed pet cats had an ecological impact.

Researchers also asked owners about their willingness to keep cats indoors during prime hunting time, from dusk to dawn. The idea was flatly rejected, with some owners providing unsolicited commentary: "My cat chooses for herself whether to stay in or go out," one wrote.

Pointing to "a dissociation between actual and perceived predatory behavior," the researchers concluded that "the cat owners in this study reject the proposition that cats are a threat to wildlife."

Sara J. Ash, a professor of ecology and conservation biology at the University of the Cumberlands in Williamsburg, Ky., said that the results highlighted the deep divide between cat owners, who see their individual animals as doing what comes naturally, and ecologists, who view cats as a predatory, nonnative species.

"These owners think, 'My cat only kills two mice a day,' " Dr. Ash said. "But they don't think about the high density of well-fed cats throughout their neighborhood."

The study's cat owners were generally able to predict whether their pets would bring home prey, but they fared poorly at estimating how much. Among 43 cats tabulated in the Cornwall village of Mawnan Smith, the average monthly catch ranged from none to 10. Over four months, the cats delivered a total of 325 animals: Nearly 60 percent were rodents, and 27 percent were birds. (According to researchers, 6.2 percent were unidentifiable.)

Although Mawnan Smith and another village in the study, Thornhill, in Scotland, are in rural settings, these owners' reactions corresponded with those of urban cat owners in Britain. In a 2012 study, they said overwhelmingly that they did not believe cats depleted certain bird populations.

John Bradshaw, a professor of anthrozoology at the University of Bristol in England, pointed out that the owners in this latest study counted only the prey their cats had brought home, and did not know how many creatures the cats might have left elsewhere — scenarios vividly illustrated in a 2013 University of Georgia study by researchers who attached "kitty cams" to 55 pet cats. Those cats left behind nearly half the prey they had killed.

But Dr. Bradshaw, the author of "Cat Sense," questioned whether cats were really having an ecological impact. "No doubt pet cats kill lots of little animals, but are they doing long-term harm in the United States and Britain?" said Dr. Bradshaw, who feels that the evidence is "flimsy."

Some researchers argue that while cats do have an impact on endangered species, notably on oceanic islands with few indigenous predators, the danger they pose in Europe and North America is hardly as significant as housing development, drought or pollution.

Noting that the biodiversity threat was insufficient to persuade owners to keep their cats indoors, Dr. McDonald and her colleagues suggested a different tactic: emphasizing the deadly hazards to pets that wander at will from, road traffic, for example, and larger predators. Increasingly, in the United States, that has meant coyotes.

According to a new study in The Journal of Mammalogy, cats, and possibly some owners, are getting the memo. American wildlife researchers investigated whether cats, which they assumed hunted mainly in residential areas, were also foraging in parks, where biodiversity is richer. Or were cats avoiding those areas because of coyotes?

With nearly 500 volunteers, researchers placed cameras in 32 parks and one urban area in six states, recording cat and coyote traffic. They found that many coyotes, but very few cats, stalked those protected public lands.

That was even true of Rock Creek Park in Washington, D.C., which is surrounded by residences and likely thousands of pet cats. Yet in six months, researchers caught coyotes on camera 125 times in the park, but photographed a cat only once.

Perhaps wary owners were keeping their cats indoors. "And maybe cats smelled coyote urine, and it struck primal fear into their little pet hearts, so they're staying away," said Roland Kays, the lead author and a research associate professor of wildlife and forestry at North Carolina State University.

But cats and coyotes did overlap in what researchers described as "small urban forests" — smatterings of woodland along greenways in suburban and exurban neighborhoods where coyotes are encroaching.

Studies have shown that such encounters may not end well. "Letting the cat out is not only a risk to the birds but to the cat," Dr. Kays said.

http://bit.ly/1LJjv9O

Scientists Make the First New Lager Yeasts in Centuries

Watch Out, Sam Adams

By Peter Andrey Smith | Jul 14, 2015

Lagers are boring. When you pop a can of lager beer, you taste the product of closely related strains of Saccharomyces pastorianus. Their genetic variety pales in comparison to the small but diverse group of yeasts used for making ale and wine, which pump out vastly different metabolic by-products and a wide range of flavors. In fact, lagers have looked and tasted much the same for hundreds of years because breeding strains with new brewing characteristics and flavors has proved difficult; the hybrids were effectively sterile. But that is about to change.

This good news harks back to the 15th-century origins of lagers. S. pastorianus appears to have been bred after an accidental cross of two other yeasts in a cool, dark cave in Bavaria when monks began "lagering," or storing beer. In the 1980s scientists determined the identity of one original parent: Saccharomyces cerevisiae, which is the mother of all yeasts used in baking and brewing. The other remained unknown until 2011, when Diego Libkind, an Argentine microbiologist, identified Saccharomyces eubayanus in the forests of Patagonia as the missing link. Wild S. eubayanus was not well adapted for industrial brewing,

but its discovery opened up the possibility of developing new yeast crosses. “Once eubayanus was discovered, things suddenly became very interesting,” says Brian Gibson, who studies brewing yeasts at the VTT Technical Research Center of Finland in Espoo.

Processed Food

Lager lovers can now officially raise a toast because Gibson and his colleagues recently logged the success of re-creating the ancient fling between S. cerevisiae and S. eubayanus. “You can now produce lager yeasts that are very different from one another,” Gibson says. All the resulting hybrids outperformed their parents, producing alcohol faster and at higher concentrations and turning out tastier products, as documented in a paper published in the Journal of Industrial Microbiology & Biotechnology. In particular, they made 4-vinylguaiacol, which resulted in flavors more characteristic of Belgian wheat beers. “The beers have a clovey aroma,” Gibson says. “It's actually quite nice but maybe something we don't always want. The idea is to have a whole range of strains, and you just pick and choose.” The hunt has now turned to finding new yeast unions that gobble up sugar more effectively, potentially creating lower-calorie beers.

Gibson notes that building up a wide variety of flavorful strains of lagers should be relatively easy, which bodes well for the as yet undisclosed breweries that are adopting the new fermenters. Lager, according to one 2012 estimate, makes up more than three quarters of the U.S. beer market.

http://bit.ly/1OHDwMd

The First Malaria Vaccine Could Be Released Soon

The vaccine isn’t as effective as hoped however, and needs several more approvals

By Marissa Fessenden

Scientists and public health officials have made great strides against malaria, but the mosquito-borne disease still kills one child living in Africa every minute, according to the World Health Organization. So news that the first malaria vaccine in the world just passed a major regulatory hurdle should be greeted with excitement. Unfortunately, some controversy tempers the announcement.

The vaccine, called RTS,S or Mosquirix, was developed by the pharmaceutical company GlaxoSmithKline and supported by some funding from the Bill and Melinda Gates Foundation. This week, the European equivalent of America’s Food and Drug Administration, the European Medicines Agency (EMA) recommended the vaccine as safe and effective to use for at-risk babies in Africa, reports Kate Kelland for Reuters.

Next, the WHO will decide whether to also give their recommendation on where and when it should be used. Any country hoping to use it would then be able to decide if they want to give the OK.

But those steps could be complicated by the fact that Mosquirix isn’t as effective as expected. For Science, Leslie Roberts reports:

In a large phase III trial, it reduced episodes of malaria by about one-third in young children in sub-Saharan Africa. That’s well under the 50% efficacy expected at the beginning of the trial, and a far cry from the 95% efficacy vaccine makers dream of, leaving scientists and policymakers asking: How good is good enough?

In addition, the vaccine needs to be administered in three doses to babies. And over time, the vaccine grows less effective and needs a booster, reports Loren Grush for The Verge. She writes, "Some scientists are concerned that the potential costs associated with such a complex and somewhat ineffective vaccine may outweigh the benefits."

Still, the danger of malaria is great enough that even mediocre vaccine could help. The EMA decided that the vaccine’s benefits outweigh the risks. Mosquirix is farther ahead in the process than any other vaccines, and GlaxoSmithKline is already working on a second generation version.

“With every vaccine of course you hope for 100% protection,” a GlaxoSmithKline scientist, Moncef Slaoui, who has worked on the vaccine for the past 30 years, tells Roberts. "If your child has three cases of severe malaria a year instead of six, it will change their lives," he says.

If the process goes smoothly for the vaccine, the first doses could be delivered to babies in 2017.

http://www.bbc.com/news/health-33544778

How speaking up can save lives

Surgeons rely on the whole team to be watchful for errors during an operation Bosses in all fields can make mistakes.

And while junior staff may always feel uncomfortable pointing them out, in some areas failing to do so could cost lives.

Aviation and medicine are two professions where the hierarchy that exists can make it particularly difficult for those lower down the pecking order to speak out.

One of the ways airlines are trying to reduce potentially fatal errors occurring is to use psychological techniques to break down that hierarchical structure and encourage people at all levels to highlight if something is about to go wrong - and medicine is starting to follow suit.

The aviation industry has embraced what's known as a "just" culture, where reporting errors is encouraged to prevent mistakes turning into tragedies.

This approach followed disasters like that in Tenerife where on 27 March 1977 when 583 people died after two planes collided on the ground and burst into flames. There was nothing technically wrong with either plane, and the main reason behind the crash was found to be the "authority gradient" in the cockpit of one plane. The captain had overruled the co-pilot who thought they hadn't been cleared for take-off.

Finding it hard to speak up in front of senior colleagues - even when it's a matter of life or death - is something that can get in the way of openly pointing out errors. Even with teams who work very closely, like the crew on an aeroplane, junior staff have been known to keep quiet in an emergency rather than question the actions of a pilot.

Dismissed

Surgical teams now hope to learn from years of research in aviation psychology which have made crashes a rarity. Matt Lindley flies jumbo jets and trains doctors in safety. He recalls a case where a surgeon was preparing to operate on a child's hand.

A junior member of staff noticed they were about to operate on the wrong hand - but her fears were dismissed. She tried again. He said: "It's quite unusual, a lot of people just back down after the first time you're not acknowledged. She was told quite bluntly to be quiet."

The team finally realised they'd operated on the wrong hand about 10 minutes into the procedure. Afterwards the junior doctor said she felt guilty - but also that she didn't have the skills to make herself heard. The high-pressured environment in hospitals means everyone needs to be alert for errors

Mr Lindley says she should have been assertive - and used certain "trigger words". "I am concerned. I am uncomfortable. This is unsafe. Or we need to stop. And I think no matter what position you are in the pecking order, to ignore those four trigger words would be very very difficult." Most doctors say they've had a "light bulb moment" when they finish the course that he runs on these techniques.

"Many say: why am I doing this course when I've been a doctor for 25 years - I should have done this on day one!" In 2012/2013 in England there were nearly 300 "never events" - incidents which can cause serious harm or death and are wholly preventable.

'All human'

Measures do exist. The WHO's Operating Checklist provides prompts at each stage of an operation for staff to carry out important checks - including basic checks like asking a patient to confirm their date of birth.

Rhona Flin, professor of applied psychology at Aberdeen University, has spent years analysing how human error can lead to disaster.

She says: "People often think their own industries are very different. Actually if you're a psychologist who's worked in different industrial settings it all looks pretty much the same to me.

A training session in a Boeing 777 slight simulator, the trainer, left, monitors the pilot's every move "They're all humans working in these technical environments. They're affected by the same kind of emotions and social factors."

Prof Flin says deference to authority can get in the way of open, honest reporting of errors and that at the time of the Tenerife disaster psychologists who observed crews training in flight simulators were alarmed by what they saw.

"Captains were briefed in advance to take some bad decisions or feign incapacity - to measure how long it would take for co-pilots would take to speak up..one psychologist monitoring their responses commented 'Co-pilots would rather die than contradict a captain'."

Simulators are also used to monitor the responses of doctors in training.

Dr Peter Jaye, an emergency medicine consultant who runs realistic simulations at St Thomas's hospital in London, says they always have to balance ensuring doctors are learning with giving them a realistic level of stress.

The team watch the mannequin too: "We're watching what he does and see how well the mannequin responds as well," says Dr Jaye, "because the mannequin can't tell us what's being done to it."

Dr Jaye and his team know they have to pitch the level of the high-pressure scenario just right - including one using a heart/lung machine known as ECMO. "We put the candidate under a lot of stress because this machine, if it goes wrong, you can die in seconds. As he takes action we respond by changing the physiology of the 'patient'."

Mr Frank Cross is a vascular surgeon who works in London. He remembers vividly a mistake he made 30 years ago - leaving a swab behind in a patient's body during an operation on her bowel. When the patient came back complaining of a lump in her abdomen a few months later the swab was detected and removed.

He says it's always better to own up, "You need to be open and honest if you make a mistake, and show that you are sorry."

http://www.eurekalert.org/pub_releases/2015-07/uoe-smo072215.php

Sleep makes our memories more accessible, study shows

Sleeping not only protects memories from being forgotten, it also makes them easier to access

Sleeping not only protects memories from being forgotten, it also makes them easier to access, according to new research from the University of Exeter and the Basque Centre for Cognition, Brain and Language. The findings suggest that after

sleep we are more likely to recall facts which we could not remember while still awake.

In two situations where subjects forgot information over the course of 12 hours of wakefulness, a night's sleep was shown to promote access to memory traces that had initially been too weak to be retrieved.

The research, published today in the journal Cortex, tracked memories for novel, made-up words learnt either prior to a night's sleep, or an equivalent period of wakefulness. Subjects were asked to recall words immediately after exposure, and then again after the period of sleep or wakefulness.

The key distinction was between those word memories which participants could remember at both the immediate test and the 12-hour retest, and those not remembered at test, but eventually remembered at retest.

The researcher found that, compared to daytime wakefulness, sleep helped rescue unrecalled memories more than it prevented memory loss.

Nicolas Dumay of the University of Exeter explains: "Sleep almost doubles our chances of remembering previously unrecalled material. The post-sleep boost in memory accessibility may indicate that some memories are sharpened overnight. This supports the notion that, while asleep, we actively rehearse information flagged as important. More research is needed into the functional significance of this rehearsal and whether, for instance, it allows memories to be accessible in a wider range of contexts, hence making them more useful."

The beneficial impact of sleep on memory is well established, and the act of sleeping is known to help us remember the things that we did, or heard, the previous day. The idea that memories could also be sharpened and made more vivid and accessible overnight, however, is yet to be fully explored.

Dr Dumay believes the memory boost comes from the hippocampus, an inner structure of the temporal lobe, unzipping recently encoded episodes and replaying them to regions of the brain originally involved in their capture - this would lead the subject to effectively re-experience the major events of the day.

Nicolas Dumay is an experimental psychologist at the University of Exeter and an honorary Staff Scientist at the Basque Centre for Cognition, Brain and Language (BCBL), in Spain.

'Sleep not just protects memories against forgetting, it also makes them more accessible' is published in the journal Cortex.