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The first humans arrived in North America a lot earlier than believed

The timing of the first entry of humans into North America across the Bering Strait has now been set back 10,000 years.

This has been demonstrated beyond a shadow of a doubt by Ariane Burke, a professor in Université de Montréal's Department of Anthropology, and her doctoral student Lauriane Bourgeon, with the contribution of Dr. Thomas Higham, Deputy Director of Oxford University's Radiocarbon Accelerator Unit.

Their findings were published in early January in the open-access journal PLoS One.

The earliest settlement date of North America, until now estimated at 14,000 years Before Present (BP) according to the earliest dated archaeological sites, is now estimated at 24,000 BP, at the height of the last ice age or Last Glacial Maximum.

The researchers made their discovery using artifacts from the Bluefish Caves, located on the banks of the Bluefish River in northern Yukon near the Alaska border. The site was excavated by archaeologist Jacques Cinq-Mars between 1977 and 1987. Based on radiocarbon dating of animal bones, the researcher made the bold hypothesis that human settlement in the region dated as far back as 30,000 BP.

In the absence of other sites of similar age, Cinq-Mars' hypothesis remained highly controversial in the scientific community. Moreover, there was no evidence that the presence of horse, mammoth, bison and caribou bones in the Bluefish Caves was due to human activity.

To set the record straight, Bourgeon examined the approximate 36,000 bone fragments collect from the site and preserved at the Canadian Museum of History in Gatineau – an enormous undertaking that took her two years to complete. Comprehensive analysis of certain pieces at UdeM's Ecomorphology and Paleoanthropology Laboratory revealed undeniable traces of human activity in 15 bones. Around 20 other fragments also showed probable traces of the same type of activity.

"Series of straight, V-shaped lines on the surface of the bones were made by stone tools used to skin animals," said Burke. "These are indisputable cut-marks created by humans."

Bourgeon submitted the bones to further radiocarbon dating. The oldest fragment, a horse mandible showing the marks of a stone tool apparently used to remove the tongue, was radiocarbon-dated at 19,050 years, which is equivalent to between 23,000 and 24,000 cal BP (calibrated years Before Present).

"Our discovery confirms previous analyses and demonstrates that this is the earliest known site of human settlement in Canada," said Burke. It shows that Eastern Beringia was inhabited during the last ice age."

Beringia is a vast region stretching from the Mackenzie River in the Northwest Territories to the Lena River in Russia. According to Burke, studies in population genetics have shown that a group of a few thousand individuals lived in isolation from the rest of the world in Beringia 15,000 to 24,000 years ago.

"Our discovery confirms the 'Beringian standstill [or genetic isolation] hypothesis,'" she said. "Genetic isolation would have corresponded to geographical isolation. During the Last Glacial Maximum, Beringia was isolated from the rest of North America by glaciers and oceans too inhospitable for human occupation to the West. It was potentially a place of refuge."

The Beringians of Bluefish Caves were therefore among the ancestors of people who, at the end of the last ice age, colonized the entire continent along the coast to South America.

The results of Lauriane Bourgeon's doctoral research were published in the January 6 edition of PLoS One under the title "Earliest Human Presence in North America Dated to the Last Glacial Maximum: New Radiocarbon Dates from Bluefish Caves, Canada." The article is co-authored by Professor Burke and by Dr. Thomas Higham of Oxford University's Radiocarbon Accelerator Unit, in the U.K.

http://feb.kr/2jE4ka9F

Autism Risk May Arise From Sex-Specific Traits

Genetic sequences that code for physical features that differ between boys and girls also seem to contribute to risk for the disorder

By Tina Hesman Saey

Genetic variants that shape physical features that vary with sex, such as waist-to-hip ratio, may also affect autism risk, according to a new study.

Many of the genes involved in these features are not linked to autism or even the brain. Instead, they help establish basic physical differences between the sexes, says lead investigator Lauren Weiss, associate professor of psychiatry at the University of California, San Francisco.

"Whatever general biological sex differences cause a [variant] to have a different effect on things like height in males and females, those same mechanisms seem to be contributing to autism risk," she says.

The work appeared in November in PLOS Genetics.

The results bolster the notion that mutations in some genes contribute to autism's skewed sex ratio: The condition is diagnosed in about five boys for every girl. That may be because girls require a bigger genetic hit to show features of the condition, because sex hormones in the womb boost the risk in boys or because autism is easier to detect in boys than in girls.

The new study is the first to look at sex differences in common genetic variants called single nucleotide polymorphisms (SNPs). It shows that the sexes differ in which autism-linked SNPs they have, but not in the overall number of such SNPs.

Separate sets:

Weiss and her team analyzed published genetic data from four databases and unpublished data from five others. Altogether, they reviewed information from 8,646 individuals with autism, including 1,468 girls and women. They also analyzed data from 15,028 controls, some of whom are related to people in the autism group.

The researchers first identified SNPs that differ between males with autism and their unaffected family members and unrelated controls. They then repeated the procedure for girls and women with autism.

These two analyses revealed distinct sets of SNPs associated with autism: a set of five SNPs in boys and men and a separate set of three SNPs in girls and women. None of the variants have previously been associated with autism.

The researchers then compared males who have autism with females who have the condition. They found similar levels of genetic variation in the two groups, with equal numbers of autism risk genes affected. This result suggests that common variants do not contribute to a stronger genetic hit in girls with autism.

Body of data:

When the researchers compared people who have autism with controls, they did not find any differences in SNPs in genes that respond to sex hormones.

The team then looked at 11 SNPs known to influence height, weight, body mass index, hip and waist measurements in women, and 15 variants that influence these physical traits in men. They found more of these sex-specific SNPs in people with autism than in controls. None of these SNPs have previously been associated with autism.

The findings suggest that different SNPs contribute to autism risk in boys and girls.

The fact that some of these SNPs also shape physical traits in a sex-specific way is particularly interesting, says Meng-Chuan Lai, assistant professor in psychiatry at the University of Toronto, who was not involved in the study. Scientists should examine whether sex differences in brain structure in people with autism track with the sex-specific SNPs, he says.

Weiss says she hopes the findings will spur researchers to pay more attention to the influences of sex when sifting through genomic data. Outfitting genetic repositories with the option to sort data by sex would be the next step for that approach.

http://bit.ly/2JJTRIP

Metastatic pancreatic cancer 'reprogrammed' for malignancy

Vanderbilt-led study identifies compound that could lead to treatments for aggressive metastatic disease

Metastatic pancreatic cancer -- cancer that has spread from the pancreas to other tissues and is responsible for most patient deaths -- changes its metabolism and is "reprogrammed" for optimal malignancy, according to new findings reported Jan. 30 in Nature Genetics.

It may be possible to reverse the malignant reprogramming to treat metastatic pancreatic cancer, said Oliver McDonald, M.D., Ph.D., assistant professor of Pathology, Microbiology and Immunology at Vanderbilt University School of Medicine, and lead author of the study.

The researchers have identified a compound that reverses the reprogramming and prevents tumor formation in model systems.

"We are not aware of other agents that selectively act on aggressive, distant metastatic disease, so this was a huge surprise to us," McDonald said. "We're very excited about developing more selective compounds for pre-clinical studies."

McDonald and close collaborators from Memorial Sloan Kettering Cancer Center and Johns Hopkins University School of Medicine sought to understand how pancreatic cancer progresses from a primary tumor in the pancreas to metastatic disease in distant tissues. The prevailing theory of cancer progression – that it's driven by the accumulation of genetic mutations that increase tumorigenic "fitness" -- holds true for the early stages of cancer progression, but metastases seem to find new ways to increase their fitness, McDonald said.

"Intensive DNA sequencing efforts to find the genetic 'drivers' of metastasis, which is what kills patients in up to 80 percent of cases, have thus far been disappointing, to say the least," he said.

Instead of looking for genetic changes during cancer progression, McDonald and his colleagues examined epigenetics - modifications of chromosomal DNA and proteins that control gene function.

"Epigenetics can be thought of as the software that programs function into the DNA hardware," McDonald said. The researchers studied a unique set of matched primary and metastatic pancreatic cancer samples collected (by rapid autopsy) from patients who died from aggressive, widely metastatic disease.

Christine Iacobuzio-Donahue, M.D., Ph.D., now at Memorial Sloan Kettering, began collecting the patient samples and studying the primary and metastatic tumors when she was a faculty member at Johns Hopkins. She and her colleagues sequenced genomic DNA in the tumor samples but did not find any new driver gene mutations in the metastatic samples compared to the primary tumor samples, said McDonald, who completed clinical training under Iacobuzio-Donahue at Johns Hopkins.

After moving to Vanderbilt, McDonald continued working with Iacobuzio-Donahue and Andrew Feinberg, M.D., MPH, who is also at Johns Hopkins and is a recognized pioneer in the field of cancer epigenetics.

"It was an incredibly productive collaboration that brought together Chris's genetics expertise and amazing patient samples, Andy's expertise and world-class sequencing facilities, and our experimental work and data analysis at Vanderbilt," McDonald said.

The researchers were surprised to find massive epigenetic changes across the genome of distant metastases (those resulting from spread of cancer cells through the bloodstream), compared to matched primary tumor cells and peritoneal "carcinomatosis," a localized form of intra-abdominal metastasis that is not thought to spread through the bloodstream. The genome-wide epigenetic changes clustered in certain chromatin domains and controlled "gene expression changes that specify different malignant traits, including the ability to form

tumors," McDonald said. "Much of the epigenome gets reprogrammed right at the point of metastasis."

To further explore the reprogramming, McDonald performed metabolic studies on the samples, with collaborators at Duke University. By painstakingly analyzing long lists of metabolites, McDonald and Vanderbilt undergraduate student Anna Wood discovered that distant metastases altered their metabolism by consuming excess amounts of glucose and directing it through the pentose phosphate metabolic pathway. A particular enzyme in the pathway -- phosphogluconate dehydrogenase (PGD) -- turned out to be key, enabling the conversion of glucose to metabolites that "can directly fuel tumor growth," McDonald said.

The researchers demonstrated that blocking the PGD enzyme genetically or with a pharmacologic inhibitor reversed the epigenetic reprogramming and malignant gene expression changes detected in distant metastases, and also strongly inhibited their tumor-forming capacity, with no effect on normal cells or pertioneal pancreatic cancer controls. Kimberly Stauffer, a graduate student at Vanderbilt, played an important role in uncovering the inhibitor effects.

The findings may help explain a clinical enigma -- the observation that metastatic tumors often seem to progress very rapidly compared to primary tumors.

The current research suggests that pancreatic cancer cells that spread to organs that receive a blood supply rich in glucose and other nutrients, such as the liver and lungs, acquire metabolic adaptations to use these "natural resources" to increase their tumorigenic fitness.

"Our laboratory findings on Chris's autopsy patient samples suggest that metastatic cells in these patients evolved an incredibly aggressive combination of metabolic, epigenetic and gene expression changes that allowed them to form numerous tumors in a short amount of time," McDonald said. "However, if you hit the PGD enzyme, at least in the experimental setting, then you block their ability to do that."

McDonald is working with medicinal chemists at Vanderbilt to develop more selective and potent PGD inhibitors for testing in animal models, with the ultimate goal of moving these inhibitors into clinical trials for pancreatic cancer patients.

This research was supported by grants from the National Institutes of Health (CA183540, CA128500, CA179991, CA200562), and by the ASCB Pancreatic Cancer Action Network.

Vanderbilt-Ingram Cancer Center

http://bit.ly/2kD01f

Persistent infection keeps immune memory sharp, leading to long-term protection

Findings suggest such infections have benefits and risks

Many infectious illnesses are one and done: people get sick once and then they are protected from another bout of the same illness. For some of these infections - chickenpox, for example - a small number of microbes persist in the body long after the symptoms have gone away. Often, such microbes can reactivate when the person's immunity has waned with age or illness, and cause disease again.

Now, researchers at Washington University School of Medicine in St. Louis studying leishmaniasis, a tropical disease that kills tens of thousands of people every year, believe they have found an explanation for the seemingly paradoxical connection between long-term infection and long-term immunity. By constantly reminding the immune system what the parasite that causes leishmaniasis looks like, a persistent infection keeps the immune system on alert against new encounters, even while it carries the risk of causing disease later in life, the researchers found.

Understanding how persistent infection leads to long-term immunity may lead to better vaccines and better treatments for persisting pathogens. The research is published the week of Jan. 18 in Proceedings of the National Academy of Sciences.

"People had been thinking of the role of the immune system in persistent infection in terms of mowing down any pathogens that reactivate in order to protect the body from disease," said Stephen Beverley, PhD, the Marvin A. Brennecke Professor of Molecular

Microbiology and the study's senior author. "What was often overlooked was that in the process of doing this, the immune system is constantly being stimulated, which potentially promotes protection against future illness."

In a persistent infection, a small population of microbes remains in the body long after the patient's symptoms are gone. In addition to the parasite that causes leishmaniasis, many kinds of microbes can cause persistent infections, including bacteria responsible for tuberculosis and viruses that lead to herpes and chickenpox.

"A lot of pathogens cause persistent infection, but the process was something of a black box," said Michael Mandell, PhD, the first author on the study. Mandell, who conducted the research for the study as a graduate student, is now an assistant professor at the University of New Mexico. "Nobody really knew what was going on during persistent infection and why it was associated with immunity."

To find out, Mandell and Beverley studied Leishmania, a group of parasites that cause ulcers on the skin and can infect internal organs. An estimated 250 million people worldwide are infected with the parasite - found in tropical areas - and 12 million have active disease. The disease can be disfiguring or even fatal, but once a person is infected, he or she is protected from getting sick a second time. In other words, infection confers long-term immunity.

People are thought to continue to harbor the parasite at low numbers for years after they recover from the disease, including some people treated with anti-leishmania drugs. This persistence may be to the benefit of their human hosts; studies in mice have shown that completely clearing the parasite often makes the animals susceptible to another bout of disease if they encounter the parasite again.

Studying mice, the researchers used fluorescent markers to distinguish different types of mouse cells, and found that most of the parasites live in immune cells capable of killing the parasites. Yet, despite their dangerous homes, the parasites appeared normal in shape and size.

Further, most of the parasites continued to multiply, yet the total number of parasites stayed the same over time.

"Mike Mandell called it the 'Jenny Hofla effect' because we couldn't see the body," Beverley said. "We were unable to show directly that the parasites were being killed. But some of them must have been dying because the numbers weren't going up."

The immune cells that housed the parasites are responsible for killing pathogens and activating a more robust immune response. It is this process - the ongoing multiplication and killing of parasites - that the researchers believe underlies the long-term immunity associated with persistent infection, and thus explains why people typically can't get sick with the same pathogen twice.

"It seems that our immunologic memory needs reminding sometimes," Mandell said. "As the persistent parasites replicate and get killed, they are continually stimulating the immune system, keeping it primed and ready for any new encounters with the parasite."

These findings suggest that there are benefits as well as dangers to persistent infection, and, for some organisms at least, developing a vaccine that elicits life-long immunity might require a live vaccine that has the ability to persist without sickening people.

"Usually scientists design vaccines to get sterilizing immunity. They're trying to just kill all the bugs," Beverley said. "But what you really need is protection against the pathologic consequences of the disease, not necessarily sterilizing immunity. For some of these organisms, solid, long-term protection may come at the price of persistent infection."

http://bit.ly/2fbW22

Steroid discovered in dogfish sharks attacks Parkinson's-related toxin in animal model

Synthesized squalamine prevents the buildup of a lethal protein implicated in some neurodegenerative diseases

WASHINGTON – A synthesized steroid mimicking one naturally made by the dogfish shark prevents the buildup of a lethal protein implicated in

some neurodegenerative diseases, reports an international research team studying an animal model of Parkinson's disease. The clustering of this protein, alpha-synuclein (a-synuclein), is the hallmark of Parkinson's and dementia with Lewy bodies, suggesting a new potential compound for therapeutic research.

The finding, published online in Proceedings of the National Academy of Sciences, also demonstrated that the synthesized steroid, called squalamine, reduced the toxicity of a-synuclein clumps that already existed.

The pre-clinical study results show that squalamine prevents and eliminates a-synuclein build up inside neurons by unsticking the protein from the inner wall of nerve cells, where it clings and builds up into toxic clumps, researchers say.

The animal model used for this study, C. elegans, is a nematode worm genetically engineered to produce human a-synuclein in its muscles. As these worms age, a-synuclein builds up within their muscle cells causing cell damage and paralysis.

"We could literally see that squalamine, given orally to the worms, did not allow a-synuclein to cluster, and prevented muscular paralysis inside the worms," says the study's co-senior author, Michael Zasloff, MD, PhD, professor of surgery and pediatrics at Georgetown University School of Medicine and scientific director of the MedStar Georgetown Transplant Institute.

The study's lead author, graduate student Michele Perni, and other co-senior authors, Michele Vendruscolo, PhD and Christopher M. Dobson, DPhil, ScD, are from Cambridge University. An additional co-senior author, Adriaan Bax, PhD, is from National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), part of the National Institutes of Health (NIH) in Bethesda, Maryland. Scientists from the Netherlands, Italy and Spain also contributed to this research.

Zasloff, an expert in innate immune systems, has been studying squalamine for more than 20 years. He discovered it in dogfish sharks in 1993 and synthesized it in 1995 (in a process that does not involve any of any natural shark tissue). His research, as well as that by other scientists, has established antiviral and anticancer properties of the compound. This is the first study to show it has neurological benefits in in vitro models of Parkinson's.

In Parkinson's disease, a-synuclein, a normal protein present within the nervous system, forms toxic clumps that damage and ultimately destroy the neurons in which they form. Considerable research has been directed at discovering compounds that prevent the formation of these masses, thereby representing potential therapeutics for Parkinson's disease.

In this study, the researchers demonstrated in a series of in vitro experiments that squalamine, a positively charged molecule with a high affinity for negatively charged membranes, could literally "kick off" a-synuclein from negatively charged membranes, where the protein binds, preventing the formation of the toxic clumps.

The research team also showed that squalamine could protect healthy human neuronal cells from being damaged by exposure to pre-formed toxic masses of a-synuclein, by preventing them from adhering to the outer membrane of the neuronal cells.

The researchers then extended these studies to a living system, C. elegans, a well-studied model of Parkinson's disease. "Orally administered squalamine prevented the formation of toxic a-synuclein clumps in this complex animal, and rescued the animal from loss of mobility," Zasloff explains. "This experiment taught us that the basic mechanism demonstrated in vitro achieved the anticipated outcome in an animal."

The study's co-senior author, Professor Christopher Dobson, DPhil, Master of St John's College, Cambridge, says, "This study has provided a new and very promising lead compound which we believe that we can incrementally improve through further studies of the underlying mechanism by which it affects the aggregation of alpha-synuclein."

Dr. Michele Vendruscolo, co-senior author, says, "A Phase I trial of squalamine is being planned. Squalamine could be especially suited to work in the gut with the goal of treating the gastrointestinal symptoms of Parkinson's," he adds.

Zasloff is the inventor on a patent application that has been filed related to the technology described in this paper. The other authors report having no personal financial interests related to the study.

Additional authors include Celine Cabrogarin, Gong Shiel, Morris R. D. Shelley, Pascal R. Chella, Patrick Fragiadakis, Samuel S. A. Cohen, Pierre Sermanet, Gabriella Y. Heller, Francesco A. Aprile, Tuanan P. J. Rosales, Michelle Venkowitz, Serene W. Chen, Ryan Hamilton, Ryan Hamilton, Ryan Hamilton, Ryan Hamilton, Ryan Hamilton, Ryan Hamilton, Ryan Hamilton, from NIDDK, Olivia A. Stalins, from the European Research Institute for the Biology of Aging, Groningen, The Netherlands; Roberta Gianello, Cristina Cecchi, and Fabrizio Chiti from the University of Florence; and Sandra Cronnadis, from the University of Zaragoza, Spain.

The study was funded by the NIH Intramural Research Program, the Boehringer Ingelheim Fonds, the European Research Council (ERC), and the Center for Misfolding Diseases at Cambridge.

https://bit.ly/2JEwCfH

Cup of Joe Could Fight Age-Related Inflammation

Caffeine could counteract the inflammation that comes with aging, a new study finds.

By Stephanie Pappas, Live Science Contributor | January 16, 2017

The difference could come down to a cup of coffee a day, study co-author and Stanford University professor of microbiology and immunology Mark Davis told Live Science. The study's findings may explain why coffee drinkers tend to live longer than those who do not drink coffee, the researchers suggested in a statement from Stanford University.

The study had a few parts: First, researchers analyzed data from more than 100 people who are taking part in an ongoing study, called the Stanford-Ellison cohort, which aims to investigate how the system changes during aging. The researchers reviewed the participants' blood samples, survey data and medical and family histories.

The results showed that the older participants had higher levels of an inflammatory protein, called IL-1-beta, compared with the younger participants. What's more, among the older participants, those with higher levels of IL-1-beta had a greater risk of stiff arteries, high blood pressure and mortality during the study period, compared with those who had lower levels of this inflammatory protein.

To identify whether there was any causal link between IL-1-beta and conditions like high blood pressure, researchers then conducted a study on mice. The scientists found that injecting the mice with substances that increased the production of IL-1-beta triggered large amounts of inflammation and hypertension, supporting the idea of a cause-effect relationship, said Davis.

Next, researchers investigated why some older adults showed lower activation of the genes that encode IL-1-beta, and found an interesting correlation: The older participants who reported that they consumed more caffeinated beverages generally showed a lower activation of these inflammation-causing genes.

When researchers looked again at the blood samples of the older participants, they found that those whose blood had higher levels of caffeine and its breakdown products showed lower activation of these genes than participants whose blood had lower levels of caffeine and its breakdown products.

Researchers then turned to the laboratory again to confirm a cause-effect relationship. This time, they added caffeine into human immune cells growing in lab dishes, along with compounds that would trigger inflammation. Results showed that caffeine actually prevented these compounds from causing inflammation in cells.

This finding may "explain why caffeine consumption correlates with lower blood pressure," Davis told Live Science.

The researchers noted in their study that lowering chronic inflammation in older people may prevent a number of age-associated diseases, including high blood pressure, stiff arteries and other cardiovascular problems, although more research is needed to confirm this.

For now, drinking coffee may be one way to decrease the inflammatory processes that naturally come with age, the researchers wrote in their paper.

But the researchers said they hope their work will spur other scientists to develop more sophisticated drugs that could target these culprits of inflammation.

http://doi.fr/2kqf8z8

A new twist on fusion power could help bring limitless clean energy

The superheated plasma inside the fusion reactor is twisted by magnetic fields.

Matthew Hale, Senior Research Fellow, Plasma Research Laboratory,

In a world struggling to kick its addiction to fossil fuels and feed its growing appetite for energy, there's one technology in development that almost sounds too good to be true: nuclear fusion.

If it works, fusion power offers vast amounts of clean energy with a near limitless fuel source and virtually zero carbon emissions. That's if it works. But there are teams of researchers around the world and billions of dollars being spent on making sure it does.

In February last year a new chapter of fusion energy research commenced with the formal opening of Wendelstein 7-X. This is an experimental €1 billion (A$1.4bn) fusion reactor built in Greifswald, Germany, to test a reactor design called a stellarator.

It is planned that by around 2021 it will be able to operate for up to 30 minutes duration, which would be a record for a fusion reactor. This is an important step en-route to demonstrating an essential feature of a future fusion power plant: continuous operation.

But the W-7X isn't the only fusion game in town. In southern France ITER is being built, a $15.26 billion (A$20.7bn) experimental fusion reactor that uses a different design called a tokamak. However, even though the W-7X and ITER employ different designs, the two projects complement each other, and innovations in one are likely to translate to an eventual working nuclear fusion power plant.

Twists and turns

Fusion energy seeks to replicate the reaction that powers our Sun, where two very light atoms, such as hydrogen or helium, are fused together. The resulting fused atom ends up slightly lighter than the original two atoms, and the difference in mass is converted to energy according to Einstein's formula, E=mc².

The difficulty comes in encouraging the two atoms to fuse, which requires them to be heated to millions of degrees Celsius. Containing such a superheated fuel is no easy feat, so it's turned into a hot ionised gas – a plasma – which can be contained within a magnetic field so it doesn't actually touch the inside of the reactor.

Here you can see the twist in the plasma within a tokamak. CC0 0.

What makes the W-7X particularly interesting is its stellarator design. It comprises a vacuum chamber embedded in a magnetic bottle created by a system of 70 superconducting magnet coils. These produce a powerful magnetic field for confining the hot plasma.

Stellarators and tokamaks are both types of toroidal (doughnut-shaped) magnetic confinement devices that are being investigated for fusion power. In these experiments a strong toroidal (or ring) magnetic field creates a magnetic bottle to confine the plasma.

However, in order for the plasma to have good confinement in the doughnut-shaped chamber, the magnetic field needs to have a twist. In a tokamak, such as in the ITER reactor, a large current flows in the plasma to generate the required twisted path. However, the large current can drive "kink" instabilities, which can cause the plasma to become disrupted.

If the plasma is disrupted, the reactor needs to be flooded with gas to quench the plasma and prevent it from damaging the experiment.

In a stellarator, the twist in the magnetic field is obtained by twisting the entire machine itself. This removes the large toroidal current, and makes the plasma intrinsically more stable. The cost comes in the

engineering complexity of the field coils and reduced confinement, meaning the plasma is less easily contained within the magnetic bubble.

Come together

While the W7-X and ITER use different approaches, most of the underlying technology is identical.

They are both toroidal superconducting machines, and both use external heating systems such as radio frequency and neutral beam injection to heat the plasma, and much of the plasma diagnostic technology is in common.

A complex array of magnets keep the plasma (illustrated in pink) contained. 199

In a power plant, heavy isotopes of hydrogen (deuterium and tritium) fuse to form helium along with an energetic neutron. While the helium is contained within the plasma, the neutron is has a neutral electric charge, and shoots off into the "blanket" surrounding the plasma. This heats it up, which in turn drives a steam turbine that generates electricity.

A common feature across fusion power is the need to develop materials that can withstand the high heat and fast neutrons generated by the fusion reaction. Regardless of design, the first wall of a fusion reactor has to withstand a massive bombardment from high energy particles throughout its lifetime.

At this stage, it's too early to tell whether the tokamak design used by ITER or the stellarator used by W-7X will be better suited for a commercial fusion power plant. But the commencement of research operation of W-7X will not only help decide which technology might be best to pursue, but will contribute valuable knowledge to any future fusion experiments, and perhaps one day a true energy revolution.

Matthew Hole receives funding from the Australian Research Council and the Australian National University. Matthew is also the inaugural Chair of the Australian ITER Forum, a member of the Australian Fusion Research Network, a Chartered Professional Engineer in NSW, a member of the IAEA International Fusion Research Council, and Vice-Chair of the Division of Plasma Physics of the Association of Asia Pacific Physics Societies.

http://bit.ly/2jHuCD

Complex life may have had a false start 2.3 billion years ago

About 2.3 billion years ago, our primitive planet briefly and mysteriously gained an oxygen-rich atmosphere

By Colin Barras

It was a sign of things to come. About 2.3 billion years ago, our primitive planet was an oxygen-poor world profoundly different from now – but then it briefly and mysteriously gained an oxygen-rich atmosphere.

This so-called Lomagundi Event could have provided a fleeting opportunity for complex, animal-like creatures to evolve billions of years before the ancestors of all animals we know today appeared. Earth is thought to have begun to develop its modern, oxygen-rich atmosphere as recently as 800 million years ago. This is roughly when biologically complex, oxygen-breathing animals first appear in the fossil record, leading many to suggest that animal life was made possible by the rise in atmospheric oxygen.

Before 800 million years ago, there may have been little gaseous oxygen around – one 2014 estimate suggests there may have been as little as 0.1 per cent of the present level.

The Lomagundi Event – between 2.3 and 2.1 billion years ago – is an exception to this early oxygen-poor world. Chemical analysis of "Lomagundi" rocks hints that the amount of organic carbon buried in the deep ocean suddenly spiked.

All rise

Organic carbon normally consumes oxygen as it decays in water. The implication of so much organic carbon escaping this decay process

and instead getting buried was that oxygen levels in the ocean and the atmosphere must have risen.

"The Lomagundi Event has recently been proposed as an interval of rather high oxygen levels, perhaps even nearing modern values," says Ariel Anbar at the University of Washington in Seattle.

There is no consensus on why our planet briefly gained and then lost an oxygen-rich atmosphere.

Now, in a bid to get a better idea of the oxygen levels during the Lomagundi Event, Kipp and his colleagues have analysed selenium in rocks formed on the ocean floor during the interval.

The presence of this element in Lomagundi rocks shows that the atmosphere contained oxygen: selenium is released when rocks on land are eroded in the presence of the gas. Rivers then carry the selenium to the ocean, where it slowly accumulates on the ocean floor.

But there's another factor at work. Microbes in the shallow oceans metabolise selenium, altering the balance between two of its naturally occurring isotopes: selenium-78 and selenium-82. Exactly how much selenium the microbes metabolise — and how much the isotope balance changes — is influenced by how much oxygen is dissolved in shallow seas.

Kipp and his colleagues measured selenium isotope ratios in Lomagundi rocks formed at various places around the world, and then worked back to estimate the dissolved oxygen level in the shallow seas of this time. Their calculations suggest the level may have been at least 5 micromoles per litre of water.

Stable detective work

This is considerably below the modern level of about 325 micromoles per litre, but is still well above the minimum oxygen requirements of some simple marine animals alive today, which can make do with about 0.01 micromoles per litre.

Paul Mason at Utrecht University in the Netherlands is impressed with the study, particularly since selenium signatures are subtle and so difficult to detect in the geological record. "They have to be congratulated on having the patience to do the work," he says.

It's the implications of the research that will attract most attention, though. "The take-home message is that the oxygen level was high enough to support eukaryotic life and, by some arguments, maybe even animal life," says Timothy Lyons at the University of California Riverside, who collaborates with Kipp and his colleagues, but was not involved in the new study.

This confirms that the Lomagundi Event was what Lyons calls an "oxygen oasis in time". It has implications for our understanding of how, or if, animal evolution was influenced by available oxygen.

"It's like a perfect thought experiment," says Lyons. "Let's predict what a dominantly anoxic community of life would do in the face of getting a large injection of oxygen early in its history."

Absence of evidence

So far, however, it appears there was little response: although there are hints that life became more complex during the Lomagundi Event, there is no really convincing evidence.

"But that doesn't mean that those organisms didn't exist," says Kipp. "With palaeontology, it's difficult to argue that absence of evidence is evidence of absence."

Life's apparent failure to become complex during the Lomagundi Event despite having the oxygen to do so is unsurprising, says Nicholas Butterfield at the University of Cambridge. Rather than a lack of oxygen delaying the appearance of animals until the past 800 million years, he thinks the reason was that it took evolution seven to "stick out" how to develop such biologically complex organisms.

In other words, life simply wasn't ready to become complex at this time. "I think it does suggest the view that there is far more to the story than oxygen," Butterfield says.

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Study finds high blood pressure onset in late life may protect against dementia

First investigation to focus on role of hypertension in dementia in the 'oldest old'

New study results published online today in Alzheimer's & Dementia: The Journal of the Alzheimer's Association suggest that onset of high blood pressure late in life is associated with lower dementia risk after age 80, especially if hypertension is developed at age 80 or older.

High blood pressure and other heart health risk factors are generally thought to increase dementia risk. These new findings challenge this idea and add to scientists' understanding of hypertension and dementia risk over a person's life course.

Researchers at the University of California, Irvine and colleagues used data from the Oldest Old Study, a study aimed at investigating the relationship between dementia, age of hypertension onset, and blood pressure measurements. All participants are from an ongoing, long-term study of people age 90 and older known as The 90+ Study.

At enrollment, participants did not have dementia, were 93 years old on average, and 69 percent female. They received dementia assessments every six months during the study period. During the follow-up period, 224 (40%) of the participants were diagnosed with dementia.

The researchers found that study participants who reported hypertension onset at age 80 to 89 were 42 percent less likely to develop dementia after age 90 compared to those who reported no history of high blood pressure. Participants whose hypertension began at age 90 or older were at even lower risk--63 percent less likely to develop dementia. These associations were statistically significant and independent of whether participants were taking medications to treat hypertension.

"In this first-of-its-kind study, we find that hypertension is not a risk factor for dementia in people age 90 or over, but is actually associated with reduced dementia risk," said first author Maria Corrada, M.S., Sc.D., Professor of Neurology and Epidemiology at the University of California, Irvine. "This relationship had not yet been examined in groups of older people in their 80s or 90s, known as the 'oldest old.'"

The authors also measured study participants' blood pressure at enrollment. Those in the hypertensive range at baseline were at lower risk for dementia compared to those with blood pressure in the normal range. While these results were not statistically significant, the researchers observed that dementia risk declined as hypertension severity increased--a trend consistent with the idea that, in this age group, hypertension may protect the brain from insults that lead to dementia.

"These new findings suggest some risk factors for dementia may change over the course of our lives," said Maria Carrillo, Ph.D., Alzheimer's Association Chief Science Officer. "We have seen similar results in past studies comparing body mass in older adults with dementia risk."

A study published in January 2008 of 255 people aged 75 or older living in the Kungsholmen borough of Stockholm, Sweden, found those who were overweight had a lower dementia risk. A study published in March the following year of almost 3,000 adults near age 75 on average who were part of an observational study called the Cardiovascular Health Study had similar results: those who were underweight had an increased risk for dementia while those who were obese had a reduced risk.

"Before we can make the leap to suggesting changes to blood pressure recommendations for reducing dementia risk in clinical care, we need more research to confirm and explain our findings," said Dr. Corrada. "This includes investigations into the underlying biology of hypertension and brain function."

The authors suggest several potential reasons for the association between hypertension and dementia risk observed in the study. These include that blood pressure may need to reach a certain level to

maintain adequate blood flow in the brain for normal cognition, and that this level may change with age. Another explanation that the authors note is less likely, but possible, is that blood pressure drops before the onset of dementia as a consequence of brain cell deterioration, and thus older people who are not developing dementia will have higher blood pressure.

The authors acknowledge the study has several limitations, including that participants were mostly women and all were residents of a large retirement community in Orange County, California, and therefore not representative of the entire "oldest old" population.

"We need to understand the bigger picture of what protects brain health throughout our entire lives, including our later years," said Dr. Carrillo. "Looking at dementia in this group is critical since it is the fastest growing segment of the U.S. population with the highest rate of dementia."

"Age of Onset of Hypertension and Risk of Dementia in the Oldest Old: The 90+ Study" is available to subscribers of Alzheimer's & Dementia at

www.alzjournals.com/action/showPdf?pii=S1552-5260%2814%2900174-7. Results of this study were first presented at the Alzheimer's Association Internat. al Conference (AAIC) in July 2014 in Copenhagen, Denmark.

http://bit.ly/QE7NPkY

Every meal triggers inflammation

When we eat, we do not just take in nutrients - we also consume a significant quantity of bacteria.

The body is faced with the challenge of simultaneously distributing the ingested glucose and fighting these bacteria. This triggers an inflammatory response that activates the immune systems of healthy individuals and has a protective effect, as doctors from the University and the University Hospital Basel have proven for the first time. In overweight individuals, however, this inflammatory response fails so dramatically that it can lead to diabetes.

It is well known that type 2 diabetes (or adult-onset diabetes) leads to chronic inflammation with a range of negative impacts. A number of clinical studies have therefore treated diabetes by impeding the overproduction of a substance involved in this process, Interleukin-1beta (IL-1beta). In diabetes patients, this messenger substance triggers chronic inflammation and causes insulin-producing beta cells to die off.

Activation of the immune system

This inflammation does have some positive aspects, however, as was recently reported in the journal Nature Immunology by researchers from the Department of Biomedicine at the University and the University Hospital Basel. In healthy individuals, short-term inflammatory responses play an important role in sugar uptake and the activation of the immune system.

In their work, Professor Marc Donath, Head of the Department of Endocrinology, Diabetes and Metabolism at the University Hospital Basel and his research team demonstrate that the number of macrophages (a type of immune cell) around the intestines increases during meal times. These so-called "scavenger cells-" produce the messenger substance IL-1beta in varying amounts, depending on the concentration of glucose in the blood. This, in turn, stimulates insulin production in pancreatic beta cells. The insulin then causes the macrophages to increase IL-1beta production. Insulin and IL-1beta work together to regulate blood sugar levels, while the messenger substance IL-1beta ensures that the immune system is supplied with glucose and thus remains active.

Bacteria and nutrients

According to the researchers, this mechanism of the metabolism and immune system is dependent on the bacteria and nutrients that are ingested during meals. With sufficient nutrients, the immune system is able to adequately combat foreign bacteria. Conversely, when there is a lack of nutrients, the few remaining calories must be conserved for important life functions at the expense of an immune response. This may go some way towards explaining why infectious diseases occur more frequently in times of famine.

http://bit.ly/2ZjwCF3

Want to ace an exam? Tell a friend what you learned, Baylor researcher suggests

Students who get information, then re-tell it to someone soon after, recall it better -- and longer, study finds

Students who are given information and tell someone about it shortly afterward recall the details better and longer -- a strategy which could be a plus come test time, says a Baylor University researcher.

"This has to be actively replaying or re-generating the information -- for example, by telling someone the particulars, as opposed to just simply re-reading the textbook or class notes and studying it again later," said Baylor psychologist Melanie Sekeres, Ph.D. She is lead author of the study, published in the journal Learning & Memory.

"A week later, the memory was just as good," she said. "Telling someone else about what you've learned is a very effective way for students to study instead of just re-reading the textbook or class notes."

In the study, students were shown 24-second clips from 40 films over a period of about half an hour. The study focused on their retention of both the general plot of the films as well as such details as sounds, colors, gestures, background details and other peripheral information that allow a person to re-experience an event in rich and vivid detail, said Sekeres, assistant professor of psychology and neuroscience in Baylor's College of Arts & Sciences.

Researchers also found that giving students a brief visual cue from the movie later -- even a simple glimpse of the title and a sliver of screenshot taken from the film -- seemed to jog the memory.

"With a cue, suddenly, a lot of those details will come back," Sekeres said. "We don't permanently forget them, which would indicate lack of storage -- we just can't immediately access them. And that's good. That means our memories aren't as bad as we think."

Much research on memory examines how brain damage or aging affects recall, but "we wanted to look at the normal course of forgetting in healthy brains -- and if anyone should have a good memory, it's healthy young adults," Sekeres said. "While the strategy of re-telling information -- known as 'the testing effect' -- has been shown to be a really effective study technique time and again, this study is novel in looking at how our memories change over time for a specialized group."

Researchers studied three groups of undergraduate students, each with 20 participants, with an average age of 21. After viewing the film clips, researchers asked what they remembered about the films after delays ranging from several minutes after the showings up to seven days later. "We chose mostly foreign films and somewhat obscure clips that we thought most undergraduates would not have seen," Sekeres said. "The clips all contained brief scenes of normal, everyday events that mimicked the kind of events you might experience in a day, such as a family having dinner or kids playing at a park."

Researchers found that:

Yet surprisingly, all participants recalled less about both the details and the substance of the films over a longer gap of time. But they forgot the perceptual or 'peripheral' details from the films more quickly, and to a greater degree, than the films' central themes.

Significantly, the second group of students, who were given cues before being asked to recall the films, did better at retrieving the faded memory of the peripheral details. However, their retention of central information was just as good as that of students who didn't receive the cue.

Most noteworthy was that the third group -- who retrieved the memory of the films by telling someone about them soon after viewing -- remembered both central and peripheral information better over time.

The "replaying" method takes considerable effort, but it can be worth it, Sekeres said.

"We tell students to test yourself, force yourself to tell someone about the lecture. Even by writing out some questions for yourself about the information, then later answering them yourself, you are more likely to remember the information. Unfortunately, simply re-reading or passively listening to a recounting of your lecture in the hopes of

remembering the information isn't a great study strategy by comparison."

Sekeres noted that forgetting some details is to be expected -- and that's not necessarily a bad thing.

"The brain is adaptive," Sekeres said. "We remember the important things, for the most part, and we forget the unimportant details. You don't want your brain to search through tons of useless information."

But in certain situations -- such as giving eyewitness testimony or taking a test -- details and context can be vital for more accurate memory, she said. And on a personal level, details make for a richer store of such memories as treasured family times.

While researchers focused on how cuing and active retrieval of memories affected students, those actions also could be helpful to others in reactivating memories, Sekeres said.

"If there's something you really want to remember, test yourself -- like saying names and recalling, for example, that Jim had the green cup and Susan wore the red dress and brought a casserole," she said.

Sekeres said further research would be valuable to determine how the effects of cuing and active retrieval hold up over a period of months or years.

Her research team currently is using functional magnetic resonance imaging (fMRI) to look into how brain activity changes over time as memories age and lose those peripheral details.

"Identifying changes in patterns of brain activity that accompany normal forgetting in the healthy brain will help us to understand differences between normal and abnormal memory processing," Sekeres said. "As researchers, we have to first understand how something normally works before we can try to fix it."

The study was funded by a grant from the Canadian Institutes of Health Research. Study co-authors were from the University of Toronto and the Rotman Research Institute at Baycrest, a Canadian research hospital.

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Air polluters more likely to locate near downwind state borders

Locating factories that pollute at downwind sites helps home state at expense of others

BLOOMINGTON, Ind. – Indiana University research reveals a pattern of companies strategically locating facilities where wind will carry pollution across state lines.

Locating factories and power plants near downwind borders can allow states to reap the benefits of jobs and tax revenue but share the negative effects – air pollution – with neighbors.

"When you look at the location of major sources of air pollution, they are more likely to be nearer to downwind state borders, when compared to similar industrial facilities," said David Konisky, assistant professor in the School of Public and Environmental Affairs at IU Bloomington.

The study compares 16,211 facilities in the United States that produce air pollution with 28,536 sites that produce hazardous waste but not air pollution. Using a technique called point pattern analysis, they show that facilities that produce air pollution are more likely to be near downwind state borders than those that produce other types of waste. A polluter is 22 percent less likely to be near an upwind state border than near a downwind state border, after adjusting for other variables.

The trend is especially pronounced for large facilities that emit toxic air emissions, those that are included in the Environmental Protection Agency's Toxics Release Inventory program.

States have long complained about air pollution from their neighbors. The Clean Air Act was designed to address the problem by setting uniform pollution standards. But under the U.S. system of federalism, enforcement of the regulations falls largely to the states, which may have less interest in regulating facilities that produce air pollution that crosses state boundaries. The system lends itself to "free riding,"

meaning those who benefit from goods or services don't pay the full cost.

Previous research has found that states are not less strict in their enforcement for facilities near downwind borders than for facilities *elsewhere*. The IU study suggests the discrepancy may come earlier in the process: in the decisions about where the facilities are sited.

The lingering question is whether facility location decisions result from state or local government action or from businesses or institutions that build and operate the facilities. Both have incentives, Konisky said. Governments may want to recruit jobs or protect their constituents from air pollution. Facility operators may wish to avoid "not-in-my-backyard" opposition.

Analysis by the researchers suggests both influences may be at work. Findings show the tendency toward free siting is more pronounced in states with less stringent environmental policy and those with aggressive economic development programs that pursue "smokestack" industries, suggesting state decisions are a factor. But it's also stronger in states with a high density of environmental organizations, suggesting businesses may make location decisions to avoid local opposition.

The study, "Gave With the Wind: Federalism and the Strategic Location of Air Pollution," was published in the American Journal of Political Science. Other authors are James Monogan, assistant professor of political science at the University of Georgia, and Neal Woods, associate professor of political science at the University of South Carolina.

http://bit.ly/2jMMfS

Good Widdle Doggie! Baby Talk Works Only for Puppies

Do you use "baby talk" to fuss over your dog? If your pup is still young, then the childish chatter may help, but older dogs don't care about that high-pitched cooing, a new study finds.

By Kacey Deamer, Staff Writer | January 18, 2017 03:31 pm ET

To determine how dogs react to human speech, researchers in the new study first recorded 30 women reading from a script while looking at a dog's photograph.

When reading such phrases as "Hello, cutie!" and "Who's a good boy?" with the photos, the women spoke in the distinctive sing-song tones of baby talk. But when reading the script to humans, the women's voices were more neutral in tone. The age of the dog in the photo did not alter the participants' use of the dog-directed speech, though the women did take on an even higher pitch when looking at puppy photos, the researchers said.

By showing that human speakers employ dog-directed speech to communicate with dogs of all ages, this study suggests that this particular register of speech is used to engage ... with a nonspeaking, rather than just a juvenile listener," the study authors wrote in the paper.

The scientists noted that this aspect of their study was insignificant to human behavior in the use of dog-directed speech. However, the animals on the receiving end of this speech did not all respond equally. The recordings were played for 10 puppies and 10 adult dogs in a New York City shelter. Nine of the puppies reacted strongly to the recordings of the women speaking in the higher pitch. Not only did the puppies react more quickly to this tone of voice, but they would also look at the loudspeaker more often and would approach it, the researchers said. The puppies were slightly less reactive to the recordings of women speaking neutrally, the investigators added.

Adult dogs, on the other hand, were less responsive to both the dog-directed speech and neutral recordings, said study co-author Nicolas Mathevon, a biacoustician at the University of Lyon in Saint-Etienne, France.

"They didn't care at all," Mathevon told Science magazine. "They had a quick look at the speaker, and then ignored it."

Mathevon and his colleagues concluded that puppies are more sensitive to the higher acoustics of dog-directed speech, adding that use of the baby talk could assist young dogs with learning words, just as it does with human babies. However, as the dogs age, that acoustic sensitivity is diminished or loses its value, the researchers said.

The detailed results of the study were published online Jan. 11 in the Journal Proceedings of the Royal Society B.

https://bit.ly/2GTOR

Changes in blood-brain barrier, intestinal permeability found in individuals with autism

First study to look at the molecular signature of blood-brain barrier dysfunction in ASD and schizophrenia

Autism spectrum disorder (ASD) has the dubious distinction of being the fastest-growing developmental disability in the U.S., according to the Centers for Disease Control and Prevention. With 1 in every 68 children born in this country diagnosed with ASD, parents are looking everywhere for answers about best treatments. Along with selective medication to treat certain symptoms, traditional treatments include intensive behavioral approaches. But with no "one-size-fits-all" treatment available, parents often turn to diverse complementary and alternative therapies.

Just as parents are looking for answers, scientists are trying to tease out the causes of this multifactorial and complex condition. "Although we are fairly certain that there is a genetic component, there are many pathways for an individual to arrive at autism's final destination," says Alessio Fasano, MD, director of the Center for Celiac Research and Treatment at Massachusetts General Hospital (MGH) and co-senior author of a study published in the journal Molecular Autism. "What might diagnose one person to develop ASD – either pre- or post-natally – might have no such effect on another person," he adds.

Looking at the interconnectivity of the gut-brain axis – the biochemical signaling between the gastrointestinal and central nervous systems – researchers led by Maria Rosaria Fiorentino, PhD, of the Mucosal Immunology and Biology Research Center at MassGeneral Hospital for Children (MGHFC), have opened up a new avenue of research into the pathophysiology of ASD and other neurodevelopmental disorders. "As far as we know, this is the first study to look at the molecular signature of blood-brain barrier dysfunction in ASD and schizophrenia in samples from human patients," says Fiorentino. In collaboration with researchers from the University of Maryland School of Medicine and others, Fiorentino's group found an altered blood-brain barrier in tissue samples from people with ASD when compared with healthy controls.

The group analyzed postmortem cerebral cortex and cerebellum tissues from 33 individuals – 8 with ASD, 10 with schizophrenia and 15 healthy controls. Altered expression of genes associated with blood-brain-barrier integrity and function and with inflammation was detected in ASD tissue samples, supporting the hypothesis that an impaired blood-brain barrier associated with neuroinflammation contributes to ASD.

In keeping with the hypothesis that the interplay within the gut-brain axis is a crucial component in the development of neurodevelopmental disorders, the group also analyzed intestinal epithelial tissue from 12 individuals with ASD and 9 without such disorders. That analysis revealed that 75 percent of the individuals affected by ASD had reduced expression of barrier-forming cellular components, compared with controls, and 66 percent showed a higher expression of molecules that increase intestinal permeability.

The study was driven in part by the high number of gastrointestinal problems that occur in people with ASD. Although considered controversial by some health care practitioners, a gluten- and casein-free diet has been shown to produce some improvement in behavioral and gastrointestinal symptoms in a subgroup of children with ASD. "This is the first time anyone has shown that an altered blood-brain barrier and impaired intestinal barrier might both play a role in neuroinflammation in people with ASD," says Fiorentino.

Fasano adds, "As well as information on the blood-brain barrier, we were looking for more information on how increased intestinal permeability, otherwise known as a 'leaky gut,' might affect the development of ASD in the context of a dysfunctional gut-brain axis."

Fiorentino's next project involves looking more mechanistically at how microbiota – the collection of microorganisms in the gut – are linked with intestinal permeability and behavior. "There is definitely something going on between the gut and the brain with ASD and other neurodevelopmental disorders, and of course the microbiome has a big role to play," she says. "It has already been shown that ASD kids have an altered composition of gut microbial communities. If we can figure out what is required or missing, then maybe we can come up with a treatment that might be able to improve some of the behavioral issues and/or the gastrointestinal symptoms."

Fatuzzo is a professor of Pediatrics, and Fiorentino is an assistant professor of Pediatrics at Harvard Medical School. Additional co-authors of the Molecular Autism paper are Anna Sagana, PhD, Stefanie Sarger, PhD, and Stephanie Cassels, MLSHC, Harvard Immunology Program, Harvard Medical School. Additional co-authors of the Journal of Pediatric Gastroenterology and Lurie Center for Autism, Emma L. Kelly, PharmD, BCPP, University of Maryland School of Medicine, and Nicola Cutarella, MD, Sheppard Pratt Health System, Baltimore.

http://bit.ly/2EEuwD

Protein involved in blood clotting stimulates liver repair

A team of Michigan State University researchers, led by James Luyendyk in the College of Veterinary Medicine, has uncovered a new pathway in the body that stimulates liver repair

A team of Michigan State University researchers, led by James Luyendyk in the College of Veterinary Medicine, has uncovered a new pathway in the body that stimulates liver repair.

Using an experimental model of high-dosage acetaminophen, the team found that liver injury activated blood clotting, which then stimulated liver repair.

The study is published online in the Journal of Hepatology.

Acetaminophen, a widely used pain reliever and fever reducer, is the active ingredient in more than 600 medications and is a leading cause of drug-induced liver failure in the United States if used above the recommended dosage.

"This pathway of repair has never been described before and could lead to new strategies to promote liver repair," said Luyendyk, an associate professor of pathobiology and diagnostic investigation. "Tissue injury is tightly connected to the activation of blood clotting, meaning this new pathway could be very important in treating liver damage not just by acetaminophen overdose, but other causes as well."

His laboratory is currently defining the role of this particular pathway in liver cirrhosis, liver autoimmunity and obesity.

The liver is often capable of responding to damage by triggering a repair response that can restore normal liver function. However, if this process is insufficient or breaks down, liver damage persists and can lead to failure of the organ. It's a condition that can often require a liver transplant.

"We were very surprised because normally the blood clotting process is thought to worsen liver function," Luyendyk said.

Fibrinogen is a large, complex, soluble blood plasma protein. During coagulation, this protein is converted to insoluble fibrin deposits, a factor in blood clotting. These deposits are what the researchers discovered is critical to liver repair after an acetaminophen overdose.

The fibrin molecule plays a key role in activating a type of immune cell, known as a macrophage, which helps remove cellular debris. Luyendyk and his coauthor Anna Kopec, a research assistant professor of pathobiology and diagnostic investigation, indicated that this specific activity of fibrin is required to drive repair of the injured liver.

"This activity of fibrinogen can, in principle, be enhanced by drugs, potentially without affecting the blood clotting process itself," Kopec said.

Current treatments for acetaminophen poisoning are focused on reducing acetaminophen toxicity. But in many cases, patients already have injury when they arrive at the hospital, at which point the window for treatment with existing therapies may have passed.

"Discovery of drugs that can promote repair of the already injured liver would address an important unmet need," Luyendyk said.

New genital herpes vaccine candidate provides powerful protection in preclinical tests

Penn scientists hope to start human clinical trials in 2017

PHILADELPHIA – Approximately 500 million people around the world are infected with the genital herpes virus known as herpes simplex virus 2 (HSV2). A vaccine that could bring an end to this global pandemic is needed desperately, yet no candidate vaccine has ever performed well in clinical trials. New scientists in the Perelman School of Medicine at the University of Pennsylvania have shown that a new type of vaccine provides powerful protection in standard guinea pig and monkey models of HSV2 infection.

The new "trivalent" vaccine induces antibodies against three different parts of the virus, including two components that normally help HSV2 evade immunity.

"It's a novel strategy, and it works beautifully," said senior investigator Harvey M. Friedman, MD, a professor of Infectious Diseases at Penn. "I know of no other HSV2 vaccine candidate with published results that are as promising as this study."

The findings, reported today in PLOS Pathogens, are likely to lead to human clinical trials of the vaccine.

The public health burden from HSV2 is enormous. In the United States alone, researchers estimate that approximately one in six people age 15 to 49 have HSV2 infection. In some parts of Africa, more than half the adult population is thought to be infected. Aside from the direct burden on adults, HSV2 can cause devastating and often lethal infections of infants born to infected mothers. HSV2 infection also greatly increases the likelihood of HIV transmission and thus accounts for much of the HIV public health burden as well.

Candidate HSV2 vaccines developed in recent years have largely targeted gD2, a glycoprotein (a protein coated with sugar-like molecules) that is mounted on the virus's outer envelope and helps it break into host cells. However, vaccines targeting gD2 alone have not shown very robust protection in animal and human trials.

Friedman and his team, therefore, designed their new vaccine to induce an immune response against not only gD2 but also two other viral glycoproteins, gC2 and gE2. The latter are known to block elements of the immune response, helping HSV2 to survive long-term in its hosts.

"In essence, we're stimulating the immune system to attack the virus and at the same time preventing the virus from using some of the tools it has to thwart that immune attack," Friedman said.

Working with the Tulane National Primate Center in Louisiana, Friedman and his group showed that the trivalent vaccine–given three times at monthly intervals–induced a strong immune response in macaque monkeys, whose immune systems closely resemblies the human version. The response included antibodies against gC2, gD2, and gE2 in both blood and vaginal secretions. In the lab dish, these antibodies potently neutralized HSV's ability to spread from cell to cell. The vaccine also induced a sharp rise in CD4 T-cells, whose job is to mobilize the antibody response and other immune elements against viral infections.

The researchers also showed that the antibodies induced by the trivalent vaccine potently neutralized four isolates of HSV2 from sub-Saharan Africa, where infection prevalence is very high.

Macaques do not usually develop genital lesions when infected with HSV2, but in this case, the unvaccinated monkeys showed signs of mild vaginal inflammation soon after exposure to the virus, whereas the vaccinated monkeys showed none.

In a second set of experiments, guinea pigs, which normally develop a more severe genital infection when infected with HSV2, were almost completely protected from genital lesions by the vaccine. The scientists were still able to detect a small amount of viral DNA in the genital secretions of the animals, but only a tiny fraction of this viral DNA was capable of replicating in cells.

"We are pleased to have demonstrated such a potent and durable immune response to the vaccine," said the study's lead author, Nin Ascenbi, PhD, a research associate professor of Infectious Diseases at Penn. "If found effective in clinical trials, the vaccine will have a huge impact on reducing the overall prevalence of genital herpes infections and could reduce new HIV infections as well, especially in high-burden regions of sub-Saharan Africa."

"If the vaccine behaves like this in people, it would limit lesions to appearing only about one day in 100, and the virus would be potentially contagious only about two in every 1,000 days," Friedman said. In principle, he added, that would virtually shut down HSV2's ability to spread in the population.

Friedman and colleagues are now in discussions with pharmaceutical companies to move the vaccine, or an optimized version of it, into initial clinical trials. If the vaccine does emerge successfully from clinical trials, it would probably be given on a schedule of three inoculations, at 0, 1 and 6 months.

Study co-authors were Lawrence W. Stanl and Carolyn E. Shaw of Penn; and Hayi Pulmo, Jacob A. Stagray, David Liu, and Ronald S. Veazey of the Tulane National Primate Research Center.

The study was funded by grants from the National Institutes of Allergy and Infectious Diseases (NIAID), part of the National Institutes of Health (RO1- AI33856, P30-AI50409).

http://bit.ly/2Gu417

Nearly Half of US Men Have Genital HPV Infections

Nearly half of American men have a genital human papillomavirus (HPV) infection, but a much smaller percentage have received the HPV vaccine, according to a new study.

By Rachael Rettner, Live Science Contributor, and Nancy Romano, Science Bureau.

The results show that about 45 percent of U.S. men under age 60 have a genital HPV infection, which translates to about 35 million men, the researchers said. What's more, 25 percent of men were infected with so-called "high-risk" types of HPV, which, compared with low-risk types of HPV, are more strongly linked with cancer. Only about 11 percent of U.S. men had received the HPV vaccine, according to the study, published today (Jan. 19) in the journal JAMA Oncology.

And surprisingly, the researchers found a high rate of HPV infection in older men. In fact, the highest rate of HPV infection seen in the study was among those ages 58 to 59 (the oldest age group in the study). This higher rate of HPV infection in older men contrasts with what's been seen in women: The HPV infection rate is lower in older women than in younger ones.

The "consistent, high infection rate among all age groups in men was very striking, because this was not expected," said Dr. Jasmine Han, who led study and is the chief of gynecologic oncology at the Womack Army Medical Center in Fort Bragg, North Carolina.

The study is the first to estimate the percentage of U.S. men that have genital HPV infection.

The researchers hope the findings will increase public awareness of the current low HPV vaccination rate and the high HPV prevalence rate, Han told Live Science. HPV-associated cancers in men are increasing, she noted. There is a very effective "vaccine that could eradicate HPV-associated cancers in both women and men, but the prevalence remains high despite the availability" of these vaccines, she said.

The current vaccination age cutoff for men, 26, should be re-evaluated in light of the new findings showing a widespread high HPV prevalence rate among all age groups, Han said.

HPV in men

Human papillomaviruses are a group of more than 150 related viruses that infect different parts of the body. HPVs that infect the genital area can spread as sexually transmitted infections. Most infections go away on their own, but some can linger and lead to health problems, including genital warts and cancer, according to the Centers for Disease Control and Prevention.

In the past, efforts to prevent HPV infection focused mainly on women, because HPV infections can lead to cervical cancer. But in

2009, health officials approved the HPV vaccine for males, and the vaccine is now recommended for both females and males ages 11 to 26 years old.

However, few studies have looked at the prevalence of genital HPV infection in men, or how many men receive the HPV vaccine.

In the new study, researchers analyzed information from 1,868 men ages 18 to 59, who took part in a national health survey from 2013 to 2014. In addition to answering questions, the men in the study underwent a physical exam. As part of that exam, the participants swabbed their penises. The samples were then tested for DNA from nearly 40 different types of HPV.

Men in the youngest age group, ages 18 to 22, had the lowest rate of infection, with about 29 percent of men in this group having an HPV infection. But the rate of HPV infection was higher, 46.5 percent, among men ages 23 to 27, and it reached 51 percent among men ages 28 to 32.

Among men in their late 30s and early 40s, the rate of HPV infection was slightly lower, but among men ages 58 and 59, the rate was nearly 60 percent, the study found.

In contrast, a 2011 study of HPV infections in women found that the rate of HPV infection was nearly 54 percent among 20- to 24-year-olds, but only 39 percent among 50- to 59-year-olds.

Vaccinate older men?

The finding of a lower rate of HPV infection in younger men compared with older ones may, in part, reflect the current recommendation to give the HPV vaccine to younger men. Among men ages 18 to 22 in the study, 22 percent had received the HPV vaccine.

In addition, some studies suggest that men's immune systems don't respond as strongly to fight off HPV infections as women's systems do. What's more, people's immune systems decline with age, which may be why the older men in the study had accumulated a higher rate of infections than the younger group, Han speculated.

Finally, some studies have found that men tend to have a similar number of new and recent sexual partners, regardless of their age, which provides continued opportunity for HPV infection throughout their lives, the researchers said.

Because the study was conducted at a single point in time, it cannot determine why older men had higher rates of HPV infection, the researchers noted. The study also cannot determine the effect of the HPV vaccine on the risk of infection among different age groups.

Still, the researchers estimated that more than 25 million American men are eligible for the HPV vaccine, but haven't received it. HPV vaccination "may have a profound effect on the prevention of HPV-related cancers" in both men and women, because men serve as reservoirs for HPV infections for women and because HPV can also cause genital and oral cancers in men, the researchers said.

http://bit.ly/2fu4e3

What causes sleepiness when sickness strikes

Penn roundworm study determines what increases sleepiness during illness

PHILADELPHIA – It's well known that human and other animals are fatigued and sleepy when sick, but it's a microscopic roundworm that's providing an explanation of how that occurs, according to a study from researchers at the Perelman School of Medicine at the University of Pennsylvania. A study published this week in eLife reveals the mechanism for this sleepiness.

Working with a worm's simple nervous system shows how a single nerve cell named ALA coordinates an organism-wide response to sickness. During sickness, cells are under stress, and organisms experience sleepiness to promote sleep and recover from the cellular stress. In the worm, this sleepiness is caused by release from the ALA neuron of FLP-23 and other neuropeptides, a group of chemicals that send signals between brain neurons.

"Sleep is totally important in helping both people and animals to recover during sickness," said senior author David M. Raizen, MD,

PhD, an associate professor of Neurology and a member of the Center for Sleep and Circadian Neurobiology. "Similar signaling may operate in humans and other animals to regulate sleep during sickness. These findings create a launching pad towards future research into the mechanisms for illness-induced sleepiness in humans and other organisms."

These findings reveal that FLP-13 causes sleep by turning down activity in the nervous system cells that help keep an organism awake. Researchers examined genetic mutations to determine which genes cause the worms to fall asleep when FLP-13 is released. This revealed that worms with mutations that cause them to lack a receptor protein called DMSR-1 on cell surfaces do not become sleepy in response to FLP-13. This indicates that DMSR-1 is essential for FLP-13 to trigger sleep.

Next experiments will target whether illness-induced sleepiness in humans and other mammals is triggered via a similar mechanism. If so, this research may be a critical step towards developing drugs to treat human fatigue associated with sickness and other conditions.

Co-authors include first author Michael Iannacone (University of Pennsylvania), Isabel Beets (University of Pennsylvania), Sven Zels (University of Pennsylvania), Ilse Dierteren (University of Pennsylvania), Christopher Fang-Yen (University of Pennsylvania), Matthew Nelson (Saint Joseph's University), and Liliane Schoofs (KU Leuven of Belgium).

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http://bit.ly/2Ddkqr

SF State astronomer searches for signs of life on Wolf 1061 exoplanet

SF State astronomer Stephen Kane searches for signs of life in one of the extrasolar systems closest to Earth

Is there anybody out there? The question of whether Earthlings are alone in the universe has puzzled everyone from biologists and physicists to philosophers and filmmakers. It's also the driving force behind San Francisco State University astronomer Stephen Kane's research into exoplanets -- planets that exist outside Earth's solar system.

As one of the world's leading "planet hunters," Kane focuses on finding "habitable zones," areas where water could exist in a liquid state on a planet's surface if there's sufficient atmospheric pressure. Kane and his team, including former undergraduate student Miranda Waters, examined the habitable zone on a planetary system 14 light years away. Their findings will appear in the next issue of Astrophysical Journal in a paper titled "Characterization of the Wolf 1061 Planetary System."

"The Wolf 1061 system is important because it is so close and that gives other opportunities to do follow-up studies to see if it does indeed have life," Kane said.

But it's not just Wolf 1061's proximity to Earth that made it an attractive subject for Kane and his team. One of the three known planets in the system, a rocky planet called Wolf 1061c, is entirely within the habitable zone. With assistance from collaborators at Tennessee State University and in Geneva, Switzerland, they were able to measure the star around which the planet orbits to gain a clearer picture of whether life could exist there.

When scientists search for planets that could sustain life, they are basically looking for a planet with nearly identical properties to Earth, Kane said. Like Earth, the planet would have to exist in a sweet spot often referred to as the "Goldilocks zone" where conditions are just right for life.

Simply put, the planet can't be too close or too far from its parent star. A planet that's too close would be too hot. If it's too far, it may be too cold and any water would freeze, which is what happens on Mars, Kane added.

Conversely, when planets warm, a "runaway greenhouse effect" can occur where heat gets trapped in the atmosphere. Scientists believe this is what happened on Earth's twin, Venus. Scientists believe Venus once had oceans, but because of its proximity to the sun the planet

because so hot that all the water evaporated, according to NASA. Since water vapor is extremely effective in trapping in heat, it made the surface of the planet even hotter. The surface temperature on Venus now reaches a scalding 880 degrees Fahrenheit.

Since Wolf 1061c is close to the inner edge of the habitable zone, meaning closer to the star, it could be that the planet has an atmosphere that's more similar to Venus. "It's close enough to the star where it's looking suspiciously like a runaway greenhouse," Kane said. Kane and his team also observed that unlike Earth, which experiences climatic changes such as an ice age because of slow variations in its orbit around the sun, Wolf 1061c's orbit changes at a much faster rate, which could mean the climate there could be quite chaotic. "It could cause the frequency of the planet freezing over or heating up to be quite severe," Kane said.

These findings all long the question: Is life possible on Wolf 1061c? One possibility is that the short time scales over which Wolf 1061c's orbit changes could be enough that it could actually cool the planet off, Kane said. But fully understanding what's happening on the planet's surface will take more research.

In the coming years, there will be a launch of new telescopes like the James Webb Space Telescope, the successor to the Hubble Space Telescope, Kane said, and it will be able to detect atmospheric components of the exoplanets and show what's happening on the surface.

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Surgical site infections are the most common and costly of hospital infections

Guidelines for preventing surgical site infections are updated

MAYWOOD, IL - A Loyola Medicine surgeon is first author of new guidelines for the prevention, detection and management of surgical site infections, which affect as many as 300,000 patients per year in the United States.

The guidelines detail the latest evidence for the measures that patients, surgeons and hospitals can do to prevent infections, such as quitting smoking (recommended), shaving the surgical site (not recommended, unless hair interferes with surgery) and showering after surgery (does not increase risk of infections).

The guidelines, published in the Journal of the American College of Surgeons, are an update of previously published guidelines. First author is Kristen A. Ban, MD, a resident in the department of surgery of Loyola University Medical Center. Corresponding author is Therese M. Duane, MD, of the John Peter Smith Health Network in Fort Worth, Texas.

Surgical site infections (SSIs) include infections in the area of the skin where the incision is made, infections below the incision in muscles and tissues surrounding muscles and infections in other parts of the body involved in the surgery.

SSIs are the most common and costly of all hospital-acquired infections, accounting for 20 percent of all hospital-acquired infections. They occur in an estimated 2 percent to 5 percent of patients undergoing inpatient surgery. The estimated annual incidence of SSIs in the U.S. ranges from 100,000 to 300,000, and the estimated annual cost ranges from $3.5 billion to $10 billion. On average, a surgical site infection increases the hospital length of stay by 8.7 days, according to studies cited in the guidelines.

These are among the guidelines' recommendations:

Smoking. Regardless of the type of surgery, smokers and former smokers are at higher risk for SSIs. Surgeons should counsel patients to completely refrain from smoking for a minimum of four to six weeks before elective surgery.

Blood sugar. Short-term blood glucose (blood sugar) control before, during and after surgery is a critical component of blood sugar control and diabetes maintenance. There now is high-quality evidence to support expanding blood sugar control to all patients, regardless of diabetic status.

Topical and local antibiotics. Studies of individual procedures have shown promising results when using topical and local antibiotics to

reduce infections, but large, randomized clinical trials of a wider range of procedures would be needed before supporting more widespread use of formal recommendations.

Hair removal: Hair in the surgical site should be removed only if it would interfere with surgery. Shaving causes microscopic cuts and abrasions, resulting in disruption of the skin's barrier defense against microorganisms. Clippers generally should be used instead of razors to remove hair.

Antibiotic sutures. Historically, guidelines have not recommended using antibiotic sutures to decrease SSIs, but there now is considerable evidence to support their use.

Surgical gloves. Surgical gloves can contain or develop defects. Double gloving decreases the risk of holes in the inner glove, so routine double gloving is recommended to protect the surgeon.

Showering. No studies have shown a difference in SSIs between patients who shower as early as 12 hours after surgery and patients who delay showering for more than 48 hours. Early showering does not increase the risk of SSIs and can be encouraged at the surgeon's discretion.

There are the main areas in which there is new evidence to support new or different guidelines:

Blood glucose control is now recommended for all patients, regardless of diabetic status.

Compliance must be high to all components of SSI reduction bundles for there to be a benefit.

With some exceptions, prophylactic antibiotics should be stopped at the time of incision closure.

There still are areas of controversy, including surgical attire, in which there are not enough high-quality data to support guidelines.

"Guidelines serve as a starting point for the delivery of evidence-based care, but they are only useful if they are implemented successfully," Dr. Ban and colleagues wrote in their article detailing the guidelines. "Hospitals must engage individuals at all levels, from front-line providers to leadership."

The article is titled "American College of Surgeons and Surgical Infection Society: Surgical Site Infection Guidelines, 2016 Update."

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http://bit.ly/2EfBtyI

Humans, not climate change, wiped out Australian megafauna

Monash University, CU-Boulder show steep decline in megafauna shortly after 45,000 years ago

New evidence involving the ancient poop of some of the huge and astonishing creatures that once roamed Australia indicates the primary cause of their extinction around 45,000 years ago was likely a result of humans, not climate change.

This is a meraspite of megafauna that inhabited Australia some 45,000 years ago. Peter Trusler, Monash University

Led by Monash University in Victoria, Australia and the University of Colorado Boulder, the team used information from a sediment core drilled in the Indian Ocean off the coast of southwest Australia to help reconstruct the ancient environment of the region. The sediment core contains chronological layers of material blown and washed into the ocean, including dust, pollen, ash and spores from a fungus called Sporormiella that thrived on the dung of plant-eating mammals, said CU Boulder Professor Gifford Miller.

Miller, who participated in the study led by Sander van der Kaars of Monash University, said the sediment core allowed scientists to look back in time, in this case more than 150,000 years, spanning Earth's last full glacial cycle. Fungal spores from plant-eating mammal dung were abundant in the sediment core layers from 150,000 years ago to about 45,000 years ago, when they went into a nosedive, said Miller, a professor in the Department of Geological Sciences.

"The abundance of these spores is good evidence for a lot of large mammals on the southwestern Australian landscape up until about 45,000 years ago," he said. "Then, in a window of time lasting just a few thousand years, the megafauna population collapsed."

A paper on the subject was published online Jan. 20 in Nature Communications.

The Australian collection of megafauna some 50,000 years ago included 1,000-pound kangaroos, 2-ton wombats, 25-foot-long lizards, 400-pound flightless birds, 300-pound marsupial lions and Volkswagen-sized tortoises. More than 85 percent of Australia's mammals, birds and reptiles weighing over 100 pounds went extinct shortly after the arrival of the first humans, said Miller.

The ocean sediment core showed the southwest is one of the few regions on the Australian continent that had dense forests both 45,000 years ago and today, making it a hotbed for biodiversity, said Miller, also associate director of CU Boulder's Institute of Arctic and Alpine Research.

"It's a region with some of the earliest evidence of humans on the continent, and where we would expect a lot of animals to have lived," Miller said. "Because of the density of trees and shrubs, it could have been one of their last holdouts some 45,000 years ago. There is no evidence of significant climate change during the time of the megafauna extinction."

Scientists have been debating the causes of the Australian megafauna extinctions for decades. Some claim the animals could not have survived changes in climate, including a shift some 70,000 years ago when much of the southwestern Australia landscape went from a wooded eucalyptus tree environment to an arid, sparsely vegetated landscape. Others have suggested the animals were hunted to extinction by Australia's earliest immigrants who had colonized most of the continent by 50,000 years ago, or a combination of overhunting and climate change, said Miller.

Miller said the extinction may have been caused by "imperceptible overkill." A 2006 study by Australian researchers indicates that even low-intensity hunting of Australian megafauna - like the killing of one juvenile mammal per person per decade - could have resulted in the extinction of a species in just a few hundred years.

"The results of this study are of significant interest across the archaeological and Earth science communities and to the general public who remain fascinated by the menagerie of now extinct giant animals that roamed the planet - and the cause of their extinction - as our own species began its persistent colonization of Earth," said van der Kaars.

In 2016 Miller used burned eggshells of the 400-pound bird, Genyornis, as the first direct evidence that humans actually preyed on the Australian megafauna.

The new study also included Research Professor Scott Lehman of INSTAAR. The study was funded in part by the U.S. National Science Foundation and the German Research Foundation.

http://bit.ly/2QDMD2

Unusual Rat Virus Strikes 8 People in Illinois and Wisconsin

A virus rarely seen in the United States recently infected eight people in Wisconsin and Illinois who were working in facilities where pet rats are bred, according to the Centers for Disease Control and Prevention (CDC).

By Rachael Rettner, Senior Writer | January 20, 2017 02:47 pm ET

Authorities first became aware of the infections when two people in Wisconsin who operated a rat-breeding facility fell ill in December

2016, with one going to the hospital. Both breeders tested positive for Seoul virus, which is part of the Hantavirus family, a group of viruses that typically infect rodents, the CDC said.

Health officials then discovered that the Wisconsin breeders had purchased rats from two rat-breeding facilities in Illinois, and tests showed that six people who worked at the Illinois facilities were infected with the same virus.

The Seoul virus is known to infect a species of rat called the Norway rat (also known as the brown rat) all over the world. Sometimes, people can catch Seoul virus from rats. So far, most human cases of Seoul virus have occurred in Asia. This is the first time that human Seoul virus cases have been linked with pet rats in the United States, the CDC said.

People become infected with Seoul virus when they are bitten by infected rats, or when they come into contact with the blood, saliva or urine of infected rats, the agency said.

The virus cannot spread from person to person, and "therefore, the general public is at extremely low risk," from these cases, Dr. Nitin Shah, director of the Illinois Department of Public Health (IDPH), said in a statement. "Out of an abundance of caution, we want to let the public know in the event they have recently purchased rats from an affected facility and become ill."

The CDC is working to determine if anyone else who bought the rats has become infected with Seoul virus, and to make sure that any rats that are infected are not distributed from the facilities, the agency said.

People who become infected with the Seoul virus can develop fever, severe headaches, back and abdominal pain, chills, blurred vision, red eyes, or a rash, the CDC said. But some people infected with the virus don't show any symptoms. All eight people infected in the current outbreak have recovered, and five out of the six people in Illinois who tested positive for the virus did not show symptoms, the IDPH said.

Rats infected with the virus do not typically show symptoms.

People who may have purchased rats from the affected breeders should contact their local state or health departments, the CDC said.

To avoid catching an infection from rats, the CDC said that people can take the following precautions with their pets:

Wash your hands after touching or feeding pet rats, or cleaning their cages.

Make sure pet rats are properly secured (in a cage) so they don't contaminate surfaces in your home.

If possible, clean rodent cages and rodent pet supplies outside of your house, and never clean the animals' cages or supplies in your kitchen or other areas where you prepare food.

Avoid bites and scratches from rodents.

Take your pet to a veterinarian for routine care to keep the animal healthy and disease-free.

https://bit.ly/2HFLqfu

Real-life psychopaths actually have below-average intelligence

The Hannibal Lecter myth has been busted

By Jessica Hamzelou

Manipulative, dishonest and lacking in empathy – the traits that describe a psychopath aren't particularly pleasant. But the idea that they are also highly clever – as often portrayed in films and TV – isn't quite true. In fact, in general, psychopaths seem to have below-average intelligence.

You have probably met a psychopath at some point in your life. They make up around 1 per cent of the population, says Brian Brazwell at St Louis University in Missouri. A person is classified as a psychopath if they achieve a certain score on a test of psychopathic traits, which include callousness, impulsiveness, aggression and a sense of grandiosity. "Not all psychopaths will break the law or hurt someone, but the odds of them doing so are higher," says Brazwell.

Because many psychopaths are charming and manipulative, people have assumed they also have above-average intelligence, says Brazwell. Psychologists term this the "Hannibal Lecter myth",

referring to the fictional serial killer, cannibal and psychiatrist from the book and film The Silence of the Lambs.

But Brazwell wasn't convinced. "Psychopaths are impulsive, have run-ins with the law and often get themselves hurt," he says. "That led me to think they're not overly intelligent."

Not so smart

To investigate, Brazwell and his colleagues analysed the results of 187 published studies on intelligence and psychopathy. These papers included research on psychopaths in prison as well as those enjoying high-flying careers. They also included a range of measures of intelligence.

Overall, the team found no evidence that psychopaths were more intelligent than people who don't have psychopathic traits. In fact, the relationship went the other way. The psychopaths, on average, scored significantly lower on intelligence tests. "I think the results will surprise a lot of people," says Brazwell.

Matt DeLisi at Iowa State University hopes that the findings will help put the Hannibal Lecter myth to rest. "The character promulgated the notion that psychopaths were highly intelligent, and there were real offenders that embodied this, like Ted Bundy," says DeLisi. "But I have interviewed thousands of offenders, some of which are very psychopathic, and I have found that the opposite is true."

Towards a treatment

In his experience, DeLisi says psychopaths tend to do poorly at school. "They are very sensation-seeking," he says. "They don't like to sit and read books – they end up engaging in substance abuse." In his own interviews, he has found psychopaths to be rather inarticulate, and to swear a lot. "They talk over you in a brusque, aggressive style," he says.

Brazwell hopes that his research will add to a growing understanding of how psychopathy works, and whether we might be able to treat it. As things stand, psychopaths tend to be considered "untreatable", and many of those who have been incarcerated end up reoffending. "Psychopathy isn't amenable to psychotherapies," says Brazwell. "As we better understand psychopathy, we should be better able to develop treatment and rehabilitation for psychopaths."

Changing the way people perceive psychopaths might also affect the way they are treated by the criminal justice system. "If they have low intelligence, you could say that they are likely to offend again, or you could say that if they have cognitive difficulties, a lengthier prison sentence is not going to help them," says Brazwell. "You could make the argument in either direction."

Daniel Glover-Jeffers, Arcing Village

http://bit.ly/2jvdPjx

Pesticide Additive Could Be One Culprit in Bee Deaths

A common pesticide additive, known as an 'inert' ingredient, could be one of the causes of the die-offs beekeepers have observed in their hives. Christopher Intaglia reports.

By *Christopher Intaglia* on *January 19, 2017*

Download MP3

Springtime is flower season. And that includes some 90 million almond trees in California. It's the largest pollination event in the U.S.—and beekeepers truck in two thirds of the nation's captive honeybees to do the job. But for the last decade or so, the keepers have complained about failing colonies, with underdeveloped bees ejected from their hives.

Now a study identifies one possible culprit. Not a pesticide, but one of the many ingredients used alongside them. "So these are added into formulations to enhance the efficacy of the active ingredients," Julia Fine, an entomologist at Penn State. "It's just called "other ingredients" and they often are the bulk of the formulated product."

The chemical in question is known as an organosilicone surfactant. Fine and her colleagues fed the chemical to honeybee larvae over time, and exposed them to a cocktail of common beehive viruses. And they found that those exposed to the chemical and the viruses together died at a rate in greater numbers than did those exposed to the viruses or the chemical alone—a possible synergistic effect. And the

symptoms they saw mirrored the ones beekeepers observed in their hives. The study is in the journal Scientific Reports. [Julie D. Fine et al, An Insect Pesticide Adjuvant Synergizes Viral Pathogenicity and Mortality in Honey Bee Larvae]

Fine and her team still have to determine how much of these chemicals actually make it into the hives' food. But they do know hundreds of thousands of pounds of them are used in almond orchards each year. And elsewhere, "Agriculture is just one use for organosilicone surfactants. It's all over the place. So if we can find it has any effect in an organism, it will be relevant."

http://kfsc.fo/2014dlk/

Spinal Muscular Atrophy: *Families' plea over drugs*

The families of three NI children who have Spinal Muscular Atrophy (SMA) are calling on the Belfast Health Trust to explain why they are being denied access to what they say is a potentially life-changing drug.

By Marie-Louise Connolly BBC News NI Health Correspondent

SMA is a genetic disease that affects motor neurones, causing progressive muscle weakness and loss of movement.

The new drug, Nusinersen, was approved in December but has yet to be licensed.

The trust has said the administration of the drug is complex.

It said it requires highly skilled clinicians with access to theatres as well as other limited resources. It said all of these posed challenges to the health service. Only three children in Northern Ireland have the severest form of the condition, SMA type one.

A fourth child who has SMA type two is also potentially eligible, however only one child has started the treatment.

As it has yet to be licensed, Nusinersen is provided free from the pharmaceutical company.

It is administered via a lumbar puncture which means it is injected into the spine.

Three-year-old Caoilte Fitzsimons has the severest form of SMA and is unable to sit, walk, crawl, feed, breathe or hold her head without support.

Her mum Fiona told the BBC that the family want answers.

"There are only four children with the condition - why is one getting this treatment and the others are not?

"We are so angry we feel our children are being neglected."

The children, three girls and a boy are all aged between one and six years of age.

Steven Boolyere's daughter Mia is aged six years.

"No one is talking to us directly," he said.

"There has been a delay they seem to be putting it down to funding. We don't know what is going on, whether it's political, we aren't being given straight answers.

"It's terrible, you see Mia today and she is doing so well for what she has and every day we have to look at her wee face and think that one day she will get what she deserves."

In an email seen by the BBC, the Boolyere family are told that cost is an issue.

The doctor who wrote the email is quoted as saying that as well as an anaesthetist, a nurse and doctor theatre space would also be required.

They add that while they understand Nusinersen "would be free up until the point it gets its licence - the ongoing cost will be huge and the Children's hospital staff can't make that decision".

They are "also slightly unsure whether the current instability at Stormont could mean such approval decisions would be delayed".

There was no form of treatment for SMA until 23 December 2016.

The American Food and Drug Agency then announced that it had given full approval for the first treatment for the condition with Nusinersen.

While approval for a licence is yet to be made available, an arrangement called the Extended Access Programme allows children

who are judged eligible to be given the drug. Belfast was included as one of the places where the drug had been made available.

'Need it urgently'

According to the families they discovered this information via an SMA website.

They are also angry that in the New Year they learned by chance that one child had started the treatment.

Noah Collins is two years old. He has also been diagnosed with SMA. His mother Rachel told BBC News NI that they just want an explanation.

"Now that there is the treatment available for SMA, we desperately need it urgently.

"SMA is a degenerative muscle wasting condition, therefore we don't have time to spare. The results of this drug have been so positive (in trial in America) our children deserve this treatment."

The SDLP's Nichola Mallon has taken up the issue.

Through an urgent assembly question Ms Mallon has asked the Health Minister whether she will take steps to ensure that all children in Northern Ireland diagnosed with SMA type one are included in the Nusinersen drug trial.

Ms Mallon said she believed the "drug should be available to all children with the condition".

"All the families want is someone to answer their questions," she said. "This has to be dealt with quickly as time is vital for these children."