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Astronomers find a new type of planet: The 'mega-Earth'

Astronomers announced today that they have discovered a new type of planet - a rocky world weighing 17 times as much as Earth.

Theorists believed such a world couldn't form because anything so hefty would grab hydrogen gas as it grew and become a Jupiter-like gas giant. This planet, though, is all solids and much bigger than previously discovered "super-Earths," making it a "mega-Earth."

"We were very surprised when we realized what we had found," says astronomer Xavier Dumusque of the Harvard-Smithsonian Center for Astrophysics (CfA), who led the data analysis and made the discovery.

"This is the Godzilla of Earths!" adds CfA researcher Dimitar Sasselov, director of the Harvard Origins of Life Initiative. "But unlike the movie monster, Kepler-10c has positive implications for life." The team's finding was presented today in a press conference at a meeting of the American Astronomical Society (AAS).

The newfound mega-Earth, Kepler-10c, circles a sunlike star once every 45 days. It is located about 560 light-years from Earth in the constellation Draco. The system also hosts a 3-Earth-mass "lava world," Kepler-10b, in a remarkably fast, 20-hour orbit.

Kepler-10c was originally spotted by NASA's Kepler spacecraft. Kepler finds planets using the transit method, looking for a star that dims when a planet passes in front of it. By measuring the amount of dimming, astronomers can calculate the planet's physical size or diameter. However, Kepler can't tell whether a planet is rocky or gassy.

Kepler-10c was known to have a diameter of about 18,000 miles, 2.3 times as large as Earth. This suggested it fell into a category of planets known as mini-Neptunes, which have thick, gaseous envelopes.

The team used the HARPS-North instrument on the Telescopio Nazionale Galileo (TNG) in the Canary Islands to measure the mass of Kepler-10c. They found that it weighed 17 times as much as Earth - far more than expected. This showed that Kepler-10c must have a dense composition of rocks and other solids.

"Kepler-10c didn't lose its atmosphere over time. It's massive enough to have held onto one if it ever had it," explains Dumusque. "It must have formed the way we see it now."

Planet formation theories have a difficult time explaining how such a large, rocky world could develop. However, a new observational study suggests that it is not alone.

Also presenting at AAS, CfA astronomer Lars A. Buchhave found a correlation between the period of a planet (how long it takes to orbit its star) and the size at which a planet transitions from rocky to gaseous. This suggests that more mega-Earths will be found as planet hunters extend their data to longer-period orbits.

The discovery that Kepler-10c is a mega-Earth also has profound implications for the history of the universe and the possibility of life. The Kepler-10 system is about 11 billion years old, which means it formed less than 3 billion years after the Big Bang.

The early universe contained only hydrogen and helium. Heavier elements needed to make rocky planets, like silicon and iron, had to be created in the first generations of stars. When those stars exploded, they scattered these crucial ingredients through space, which then could be incorporated into later generations of stars and planets. This process should have taken billions of years. However, Kepler-10c shows that the universe was able to form such huge rocks even during the time when heavy elements were scarce.

"Finding Kepler-10c tells us that rocky planets could form much earlier than we thought. And if you can make rocks, you can make life," says Sasselov.

This research implies that astronomers shouldn't rule out old stars when they search for Earth-like planets. And if old stars can host rocky Earths too, then we have a better chance of locating potentially habitable worlds in our cosmic neighborhood.

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Anti-diabetic drug slows aging and lengthens lifespan

A study by Belgian doctoral researcher Wouter De Haes (KU Leuven) and colleagues provides new evidence that metformin, the world's most widely used anti-diabetic drug, slows ageing and increases lifespan.

In experiments reported in the journal Proceedings of the National Academy of Sciences, the researchers tease out the mechanism behind metformin's age-slowing effects: the drug causes an increase in the number of toxic oxygen molecules released in the cell and this, surprisingly, increases cell robustness and longevity in the long term. Mitochondria – the energy factories in cells – generate tiny electric currents to provide the body's cells with energy. Highly reactive oxygen molecules are produced as a by-product of this process.

While these molecules are harmful because they can damage proteins and DNA and disrupt normal cell functioning, a small dose can actually do the cell good, say the researchers: "As long as the amount of harmful oxygen molecules released in the cell remains small, it has a positive long-term effect on the cell. Cells use the reactive oxygen particles to their advantage before they can do any damage," explains Wouter De Haes. "Metformin causes a slight increase in the number of harmful oxygen molecules. We found that this makes cells stronger and extends their healthy lifespan."

It was long thought that harmful reactive oxygen molecules were the very cause of ageing. The food and cosmetics industries are quick to emphasise the 'anti-ageing' qualities of products containing antioxidants, such as skin creams, fruit and vegetable juices, red wine and dark chocolate.

But while antioxidants do in fact neutralise harmful reactive oxygen molecules in the cell, they actually negate metformin's anti-ageing effects because the drug relies entirely on these molecules to work.

The researchers studied metformin's mechanism in the tiny roundworm Caenorhabditis elegans, an ideal species for studying ageing because it has a lifespan of only three weeks. "As they age, the worms get smaller, wrinkle up and become less mobile. But worms treated with metformin show very limited size loss and no wrinkling. They not only age slower, but they also stay healthier longer," says Wouter De Haes. "While we should be careful not to over-extrapolate our findings to humans, the study is promising as a foundation for future research."

Other studies in humans have shown that metformin suppresses some cancers and heart disease. Metformin could even be an effective drug for counteracting the general effects of ageing, say the researchers.

The study was carried out by Wouter De Haes under the supervision of Liesbet Temmerman and Professor Liliane Schoofs (KU Leuven) and in close collaboration with Professor Bart Braeckman (Ghent University).

http://www.eurekalert.org/pub_releases/2014-06/osu-tpf053014.php

Tracking potato famine pathogen to its home may aid $6 billion global fight

Phytophthora infestans originated in a pretty, alpine valley in central Mexico and co-evolved with potatoes

CORVALLIS, Ore. – The cause of potato late blight and the Great Irish Famine of the 1840s has been tracked to a pretty, alpine valley in central Mexico, which is ringed by mountains and now known to be the ancestral home of one of the most costly and deadly plant diseases in human history.

Research published today in the Proceedings of the National Academy of Sciences, by researchers from Oregon State University, the USDA Agricultural Research Service and five other institutions, concludes that Phytophthora infestans originated in this valley and co-evolved with potatoes over hundreds or maybe a few thousand years, and later spread repeatedly to much of the world.

Knowing the origin of the pathogen does more than just fill in a few facts in agricultural history, the scientists say. It provides new avenues to discover resistance genes, and helps explain the mechanisms of repeated emergence of this disease, which to this day is still the most costly potato pathogen in the world.

Potato late blight continues to be a major threat to global food security and at least $6 billion a year is spent to combat it, mostly due to the cost of fungicides and substantial yield losses. But P. infestans is now one of the few plant pathogens in the world with a well-characterized center of origin.

"This is immensely important," said Niklaus Grunwald, who is a courtesy professor in the Department of Botany and Plant Pathology in the College of Agricultural Sciences at Oregon State University, a researcher with the USDA Agricultural Research Service, and lead author on the study. "This is just a textbook example of a center of origin for a pathogen, and it's a real treat," Grunwald said. "I can't think of another system so well understood. This should allow us to make significant headway in finding additional genes that provide resistance to P. infestans."

Finding ways to genetically resist the potato late blight, scientists say, could help reduce the use of fungicides, and the expense and environmental concerns associated with them.

There had been competing theories about where P. infestans may have evolved, with the leading candidates being the Toluca Valley near Mexico City, or areas in South America where the potato itself actually evolved thousands of years ago.

Gene sequencing technology used by this research group helped pin down the Toluca Valley as the ancestral hot spot. The P. infestans pathogen co-evolved there hundreds of years ago with plants that were distant cousins of modern potatoes, which produced tubers but were more often thought of as a weed than a vegetable crop.

Today, the newly-confirmed home of this pathogen awaits researchers almost as a huge, natural laboratory, Grunwald said. Since different potato varieties, plants and pathogens have been co-evolving there for hundreds of years, it offers some of the best hope to discover genes that provide some type of resistance.

Along with other staple foods such as corn, rice and wheat, the potato forms a substantial portion of the modern human diet. A recent United Nations report indicated that every person on Earth eats, on average, more than 70 pounds of potatoes a year. Potatoes contain a range of vitamins, minerals, phytochemicals, fiber and – for hungry populations – needed calories.

It's believed that the potato was first domesticated more than 7,000 years ago in parts of what are now Peru and Bolivia, and it was brought to Europe by Spanish explorers in the late 1500s. A cheap and plentiful crop that can grow in many locations, the ability to increase food production with the potato eventually aided a European population boom in the 1800s.

But what the New World provided, it also took away - in the form of a potato late blight attack that originated from Mexico, caused multiple crop failures and led,

among other things, to the Irish potato famine that began in 1845. Before it was over, 1 million people had died and another 1 million emigrated, many to the U.S. That famine was exacerbated by lack of potato diversity, as some of the varieties most vulnerable to P. infestans were also the varieties most widely cultivated.

Collaborators on the research were from the University of Florida, the James Hutton Institute in Scotland, the University of the Andes in Colombia, Cornell University, and the International Potato Center in Beijing. It was supported by the U.S. Department of Agriculture and the Scottish government.

http://www.eurekalert.org/pub_releases/2014-06/teia-moa060214.php

Modern ocean acidification is outpacing ancient upheaval, study suggests

Rate may be 10 times faster, according to new data

Some 56 million years ago, a massive pulse of carbon dioxide into the atmosphere sent global temperatures soaring. In the oceans, carbonate sediments dissolved, some organisms went extinct and others evolved.

Scientists have long suspected that ocean acidification played a part in the crisis - similar to today, as manmade CO2 combines with seawater to change its chemistry. Now, for the first time, scientists have quantified the extent of surface acidification from those ancient days, and the news is not good: the oceans are on track to acidify at least as much as they did then, only at a much faster rate.

In a study published in the latest issue of Paleoceanography, the scientists estimate that surface ocean acidity increased by about 100 percent in a few thousand years or more, and stayed that way for the next 70,000 years. In this radically changed environment, some creatures died out while others adapted and evolved. The study is the first to use the chemical composition of fossils to reconstruct surface ocean acidity at the Paleocene-Eocene Thermal Maximum (PETM), a period of intense warming on land and throughout the oceans due to high CO2.

"This could be the closest geological analog to modern ocean acidification," said study coauthor Bärbel Hönisch, a paleoceanographer at Columbia University's Lamont-Doherty Earth Observatory. "As massive as it was, it still happened about 10 times more slowly than what we are doing today."

The oceans have absorbed about a third of the carbon humans have pumped into the air since industrialization, helping to keep temperatures lower than they would be otherwise. But that uptake of carbon has come at a price. Chemical reactions caused by that excess CO2 have made seawater grow more acidic, depleting it of the carbonate ions that corals, mollusks and calcifying plankton need to build their shells and skeletons.

In the last 150 years or so, the pH of the oceans has dropped substantially, from 8.2 to 8.1--equivalent to a 25 percent increase in acidity. By the end of the century, ocean pH is projected to fall another 0.3 pH units, to 7.8. While the researchers found a comparable pH drop during the PETM--0.3 units--the shift happened over a few thousand years. "We are dumping carbon in the atmosphere and ocean at a much higher rate today - within centuries," said study coauthor Richard Zeebe, a paleoceanographer at the University of Hawaii. "If we continue on the emissions path we are on right now, acidification of the surface ocean will be way more dramatic than during the PETM."

Ocean acidification in the modern ocean may already be affecting some marine life, as shown by the partly dissolved shell of this planktic snail, or pteropod, caught off the Pacific Northwest.

The study confirms that the acidified conditions lasted for 70,000 years or more, consistent with previous model-based estimates. "It didn't bounce back right away," said Timothy Bralower, a researcher at Penn State who was not involved in the study. "It took tens of thousands of years to recover."

From seafloor sediments drilled off Japan, the researchers analyzed the shells of plankton that lived at the surface of the ocean during the PETM. Two different methods for measuring ocean chemistry at the time - the ratio of boron isotopes in their shells, and the amount of boron --arrived at similar estimates of acidification. "It's really showing us clear evidence of a change in pH for the first time," said Bralower.

What caused the burst of carbon at the PETM is still unclear. One popular explanation is that an overall warming trend may have sent a pulse of methane from the seafloor into the air, setting off events that released more earth-warming gases into the air and oceans. Up to half of the tiny animals that live in mud on the seafloor - benthic foraminifera - died out during the PETM, possibly along with life further up the food chain.

Other species thrived in this changed environment and new ones evolved. In the oceans, dinoflagellates extended their range from the tropics to the Arctic, while on land, hoofed animals and primates appeared for the first time. Eventually, the oceans and atmosphere recovered as elements from eroded rocks washed into the sea and neutralized the acid.

Today, signs are already emerging that some marine life may be in trouble. In a recent study led by Nina Bednarsek at the U.S. National Oceanic and Atmospheric Administration, more than half of the tiny planktic snails, or pteropods, that she and her team studied off the coast of Washington, Oregon and California showed badly dissolved shells. Ocean acidification has been linked to the widespread death of baby oysters off Washington and Oregon since 2005, and may also pose a threat to coral reefs, which are under additional pressure from pollution and warming ocean temperatures.

"Seawater carbonate chemistry is complex but the mechanism underlying ocean acidification is very simple," said study lead author Donald Penman, a graduate student at University of California at Santa Cruz. "We can make accurate predictions about how carbonate chemistry will respond to increasing carbon dioxide levels. The real unknown is how individual organisms will respond and how that cascades through ecosystems."

Other authors of the study, which was funded by the U.S. National Science Foundation: Ellen Thomas, Yale University; and James Zachos, UC Santa Cruz

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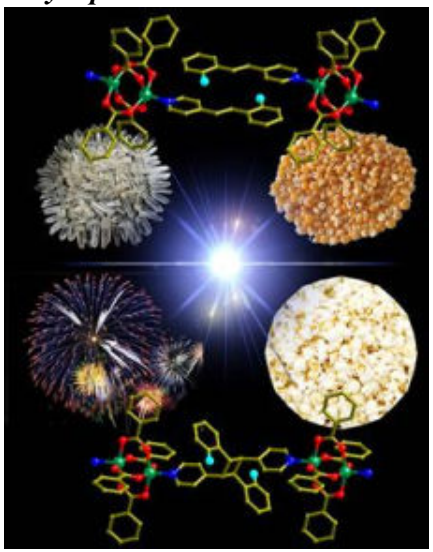
Scientists demonstrate rare chemical phenomenon that could be harnessed to harvest solar energy

First instance of a "photosalient effect" driven by a photochemical reaction in solids to be reported



A schematic diagram showing the popping nature of the crystals under UV light, a property that is very similar to the popping of corns on a hot plate. **Credit: National University of Singapore**

A team of international scientists led by Professor Jagadese J Vittal of the Department of Chemistry at the National University of Singapore's (NUS) Faculty of Science has successfully unraveled the chemical reaction responsible for propelling microscopic crystals to leap distances up to hundreds of times their own size when they are exposed to ultraviolet (UV) light.

This popping effect, akin to the bursting of popcorn kernels at high temperatures, demonstrates the conversion of light into mechanical motion. It is the first instance of a "photosalient effect" driven by a photochemical reaction in solids to be reported.

The rare phenomenon provides a new way to transfer light energy into mechanical motion, and potentially offers a fresh approach to harness solar energy to power light-driven actuators and mechanical devices. These novel findings were published as the cover story in the English version of German scientific journal Angewandte Chemie International Edition on 2 June 2014.

Popcorn-like explosion of tiny crystals demonstrated

The NUS team has been actively looking for ways to control the reactivity of solids. While studying the metal complex polymerisation in the solid state, Mr Raghavender Medishetty, a PhD candidate, and Ms Bai Zhaozhi, a third-year undergraduate student, of the Department of Chemistry at the NUS Faculty of Science, found that very tiny crystals leap violently when exposed to UV light. Interestingly, even when the crystals are irradiated with weak UV light, the single crystals burst violently to travel up to hundreds of times their sizes. Such a distance is equivalent to that of a human jumping few hundred metres.

To understand the reactions behind the self-actuation of the crystals, the NUS team worked with a research team from the New York University Abu Dhabi led by Associate Professor Panče Naumov to capture the rapid motion of the crystals with an optical microscope coupled to a high-speed camera. They also collaborated with a research team from the Max Plank Institute for Solid State Research in Germany, led by Professor Robert E. Dinnebier to model the kinetics by time-resolved powder X-ray diffraction methods.

Through the use of a variety of analytical methods, the researchers discovered that the cause for the popping and disintegration of these single crystals was due to the strain generated during the photochemical reaction in the crystal, leading to the formation of metal coordination polymers. Sudden expansion of volume during this reaction results in the release of the stress in the form of ballistic events. Such a chemical reaction is very similar to the popping of corn kernels on a hot plate as a result of rapid expansion of the inner kernel compared to the outer shell.

Elaborating on the findings, Prof Vittal said, "Photoactuated movements are induced by the application of light to certain type of crystals, but they are observed to be less efficient than the biomechanical motions of plant and animal tissues. In our work, we observed that the conversion of energy in the crystals may be able to mimic the motility of biological systems and provide a new way to transfer light energy into mechanical motion." NUS scientists demonstrate rare chemical phenomenon that could be harnessed to harvest solar energy

PhD candidate Mr Raghavender Medishetty (left) and Professor Jagadese J Vittal (right), Department of Chemistry at the NUS Faculty of Science Credit: National University of Singapore

He added, "Our work validates that the so called "bad" UV light from sources such as the sun can be utilised to convert chemical reactions to drive mechanical motions with practical uses. Knowledge and application of such behaviour is very important towards addressing the global energy crisis."

This study opens doors for further studies into materials for alternative energy conversion.

Further research

The NUS research team is examining a series of new compounds to better understand the mechanism and enhance the efficiency of the photosalient effect.

They are also conducting systematic studies to look into the effects of chemical modification on the photosalient effect.

The team hopes to eventually develop new materials that could convert solar energy effectively into mechanical energy. In addition, the team also hopes to leverage on the principle of the photosalient effect to create a new source of reversible chemical energy by controlling the shape and size of crystals used for energy conversion.

http://nyti.ms/1tO45mY

What's Lost as Handwriting Fades

Does handwriting matter?

By MARIA KONNIKOVA JUNE 2, 2014

Not very much, according to many educators. The Common Core standards, which have been adopted in most states, call for teaching legible writing, but only in kindergarten and first grade. After that, the emphasis quickly shifts to proficiency on the keyboard.

But psychologists and neuroscientists say it is far too soon to declare handwriting a relic of the past. New evidence suggests that the links between handwriting and broader educational development run deep.

Children not only learn to read more quickly when they first learn to write by hand, but they also remain better able to generate ideas and retain information. In other words, it's not just what we write that matters - but how.

"When we write, a unique neural circuit is automatically activated," said Stanislas Dehaene, a psychologist at the Collège de France in Paris. "There is a core recognition of the gesture in the written word, a sort of recognition by mental simulation in your brain.

Handwriting is being dropped in public schools - that could be bad for young minds.

Google's new hands-free computer is finding its way into operating rooms.

"And it seems that this circuit is contributing in unique ways we didn't realize," he continued. "Learning is made easier."

A 2012 study led by Karin James, a psychologist at Indiana University, lent support to that view. Children who had not yet learned to read and write were presented with a letter or a shape on an index card and asked to reproduce it in one of three ways: trace the image on a page with a dotted outline, draw it on a blank white sheet, or type it on a computer. They were then placed in a brain scanner and shown the image again.

The researchers found that the initial duplication process mattered a great deal. When children had drawn a letter freehand, they exhibited increased activity in three areas of the brain that are activated in adults when they read and write: the left fusiform gyrus, the inferior frontal gyrus and the posterior parietal cortex.

By contrast, children who typed or traced the letter or shape showed no such effect. The activation was significantly weaker.

Dr. James attributes the differences to the messiness inherent in free-form handwriting: Not only must we first plan and execute the action in a way that is not required when we have a traceable outline, but we are also likely to produce a result that is highly variable. That variability may itself be a learning tool. "When a kid produces a messy letter," Dr. James said, "that might help him learn it."

Our brain must understand that each possible iteration of, say, an "a" is the same, no matter how we see it written. Being able to decipher the messiness of each "a" may be more helpful in establishing that eventual representation than seeing the same result repeatedly. "This is one of the first demonstrations of the brain being changed because of that practice," Dr. James said.

In another study, Dr. James is comparing children who physically form letters with those who only watch others doing it. Her observations suggest that it is only the actual effort that engages the brain's motor pathways and delivers the learning benefits of handwriting.

The effect goes well beyond letter recognition. In a study that followed children in grades two through five, Virginia Berninger, a psychologist at the University of Washington, demonstrated that printing, cursive writing, and typing on a keyboard are all associated with distinct and separate brain patterns - and each results in a distinct end product. When the children composed text by hand, they not only consistently produced more words more quickly than they did on a keyboard, but expressed more ideas. And brain imaging in the oldest subjects suggested that the connection between writing and idea generation went even further. When these children were asked to come up with ideas for a composition, the ones with better handwriting exhibited greater neural activation in areas associated with working memory - and increased overall activation in the reading and writing networks.

It now appears that there may even be a difference between printing and cursive writing - a distinction of particular importance as the teaching of cursive disappears in curriculum after curriculum. In dysgraphia, a condition where the ability to write is impaired, usually after brain injury, the deficit can take on a curious form: In some people, cursive writing remains relatively unimpaired, while in others, printing does.

In alexia, or impaired reading ability, some individuals who are unable to process print can still read cursive, and vice versa - suggesting that the two writing modes activate separate brain networks and engage more cognitive resources than would be the case with a single approach.

Dr. Berninger goes so far as to suggest that cursive writing may train self-control ability in a way that other modes of writing do not, and some researchers argue that

it may even be a path to treating dyslexia. A 2012 review suggests that cursive may be particularly effective for individuals with developmental dysgraphia - motor-control difficulties in forming letters - and that it may aid in preventing the reversal and inversion of letters.

Cursive or not, the benefits of writing by hand extend beyond childhood. For adults, typing may be a fast and efficient alternative to longhand, but that very efficiency may diminish our ability to process new information. Not only do we learn letters better when we commit them to memory through writing, memory and learning ability in general may benefit.

Two psychologists, Pam A. Mueller of Princeton and Daniel M. Oppenheimer of the University of California, Los Angeles, have reported that in both laboratory settings and real-world classrooms, students learn better when they take notes by hand than when they type on a keyboard. Contrary to earlier studies attributing the difference to the distracting effects of computers, the new research suggests that writing by hand allows the student to process a lecture's contents and reframe it - a process of reflection and manipulation that can lead to better understanding and memory encoding.

Not every expert is persuaded that the long-term benefits of handwriting are as significant as all that. Still, one such skeptic, the Yale psychologist Paul Bloom, says the new research is, at the very least, thought-provoking.

"With handwriting, the very act of putting it down forces you to focus on what's important," he said. He added, after pausing to consider, "Maybe it helps you think better."

Maria Konnikova is a contributing writer for The New Yorker online and the author of "Mastermind: How to Think Like Sherlock Holmes."

http://bit.ly/1i8w5g2

New amyloid-reducing compound could be a preventive measure against Alzheimer's

NYU Langone researchers identify promising treatment target molecule in mice studies

Scientists at NYU Langone Medical Center have identified a compound, called 2-PMAP, in animal studies that reduced by more than half levels of amyloid proteins in the brain associated with Alzheimer's disease. The researchers hope that someday a treatment based on the molecule could be used to ward off the neurodegenerative disease since it may be safe enough to be taken daily over many years.

"What we want in an Alzheimer's preventive is a drug that modestly lowers amyloid beta and is also safe for long term use," says Martin J. Sadowski, MD, PhD, associate professor of neurology, psychiatry, and biochemistry and molecular pharmacology, who led the research to be published online June 3 in the journal Annals of Neurology. "Statin drugs that lower cholesterol appear to have those properties and have made a big impact in preventing coronary artery disease. That's essentially what many of us envision for the future of Alzheimer's medicine."

The 2-PMAP molecule that Dr. Sadowski's team identified is non-toxic in mice, gets easily into the brain, and lowers the production of amyloid beta and associated amyloid deposits.

The prime target for Alzheimer's prevention is amyloid beta. Decades before dementia begins, this small protein accumulates in clumps in the brain. Modestly lowering the production of amyloid beta in late middle age, and thus removing some of the burden from the brain's natural clearance mechanisms, is believed to be a good prevention strategy. Researchers two years ago reported that something like this happens naturally in about 0.5 percent of Icelanders, due to a mutation they carry that approximately halves amyloid beta production throughout life. These fortunate people show a slower cognitive decline in old age, live longer, and almost never get Alzheimer's.

Prevention of Alzheimer's dementia is now considered more feasible than stopping it after it has begun, when brain damage is already severe. Every prospective Alzheimer's drug in clinical trials has failed even to slow the disease process at that late stage. "The key is to prevent the disease process from going that far," Dr. Sadowski says.

Dr. Sadowski and colleagues screened a library of compounds and found that 2-PMAP reduced the production of amyloid beta's mother protein, known as amyloid precursor protein (APP). The APP protein normally is cut by enzymes in a way that leaves amyloid beta as one of the fragments. Dr. Sadowski's team found that 2-PMAP, even at low, non-toxic concentrations, significantly reduced APP production in test cells, lowering amyloid beta levels by 50 percent or more.

The scientists subsequently found that 2-PMAP had essentially the same impact on APP and amyloid beta in the brains of living mice. The mice were engineered to have the same genetic mutations found in Alzheimer's patients with a hereditary form of the disease, causing overproduction of APP and Alzheimer's-like amyloid deposits. A five-day treatment with 2-PMAP lowered brain levels of APP and, even more so, levels of amyloid beta. Four months of treatment sharply reduced the amyloid deposits and prevented the cognitive deficits that are normally seen in these transgenic mice as they get older.

Dr. Sadowski and his laboratory are now working to make chemical modifications to the compound to improve its effectiveness. But 2-PMAP already seems to have advantages over other amyloid-lowering compounds, he says. One is that it can

cross efficiently from the bloodstream to the brain, and thus doesn't require complex modifications that might compromise its effects on APP.

The compound also appears to have a highly selective effect on APP production, by interfering with the translation of APP's gene transcript into the APP protein itself.

The best known candidates for Alzheimer's preventives lower amyloid by inhibiting the secretase enzymes that cleave amyloid beta from APP, tending to cause unwanted side-effects via their off target interference with the processing of other client proteins cleaved by these enzymes. A clinical trial of one secretase inhibitor was halted in 2010 after it was found to worsen dementia and cause a higher incidence of skin cancer.

Alzheimer's disease, the most common form of dementia, currently afflicts more than five million Americans, according to the Alzheimer's Association. Unless preventive drugs or treatments are developed, the prevalence of Alzheimer's is expected to triple by 2050.

Other NYU Langone researchers contributing to the study were lead author Ayodeji A. Asuni, PhD; Maitea Guridi, MS; Joanna E. Pankiewicz, MD, PhD; and Sandrine Sanchez, PhD.

U.S. Patent No. 8,658,677 was recently issued for the compound discussed in this release.

Funding for the research was provided in part by the National Institutes of Health (grants R01 AG31221 and K02 AG34176).

http://www.eurekalert.org/pub_releases/2014-06/uoia-bsl060314.php

Brain signals link physical fitness to better language skills in kids

Children who are physically fit have faster and more robust neuro-electrical brain responses during reading than their less-fit peers, researchers report.

CHAMPAIGN, Ill. - These differences correspond with better language skills in the children who are more fit, and occur whether they're reading straightforward sentences or sentences that contain errors of grammar or syntax.

The new findings, reported in the journal Brain and Cognition, do not prove that higher fitness directly influences the changes seen in the electrical activity of the brain, the researchers say, but offer a potential mechanism to explain why fitness correlates so closely with better cognitive performance on a variety of tasks.

"All we know is there is something different about higher and lower fit kids," said University of Illinois kinesiology and community health professor Charles Hillman, who led the research with graduate student Mark Scudder and psychology professor Kara Federmeier. "Now whether that difference is caused by fitness or maybe some third variable that (affects) both fitness and language processing, we don't know yet."

The researchers used electroencephalography (EEG), placing an electrode cap on the scalp to capture some of the electrical impulses associated with brain activity. The squiggly readouts from the electrodes look like seismic readings captured during an earthquake, and characteristic wave patterns are associated with different tasks.

These patterns are called "event-related potentials" (ERPs), and vary according to the person being evaluated and the nature of the stimulus, Scudder said.

For example, if you hear or read a word in a sentence that makes sense ("You wear shoes on your feet"), the component of the brain waveform known as the N400 is less pronounced than if you read a sentence in which the word no longer makes sense ("At school we sing shoes and dance," for example), Scudder said.

"We focused on the N400 because it is associated with the processing of the meaning of a word," he said. "And then we also looked at another ERP, the P600, which is associated with the grammatical rules of a sentence." Federmeier, a study co-author, is an expert in the neurobiological basis of language. Her work inspired the new analysis.

The researchers found that children who were more fit (as measured by oxygen uptake during exercise) had higher amplitude N400 and P600 waves than their less-fit peers when reading normal or nonsensical sentences. The N400 also had shorter latency in children who were more fit, suggesting that they processed the same information more quickly than their peers.

Most importantly, the researchers said, these differences in brain activity corresponded to better reading performance and language comprehension in the children who were more fit. "Previous reports have shown that greater N400 amplitude is seen in higher-ability readers," Scudder said.

"Our study shows that the brain function of higher fit kids is different, in the sense that they appear to be able to better allocate resources in the brain towards aspects of cognition that support reading comprehension," Hillman said.

More work must be done to tease out the causes of improved cognition in kids who are more fit, Hillman said, but the new findings add to a growing body of research that finds strong links between fitness and healthy brain function.

Many studies conducted in the last decade, on children and older adults, "have repeatedly demonstrated an effect of increases in either physical activity in one's lifestyle or improvements in aerobic fitness, and the implications of those health behaviors for brain structure, brain function and cognitive performance," Hillman said.

The National Institute of Child Health and Human Development at the National Institutes of Health supported this research.

The paper, "The Association Between Aerobic Fitness and Language Processing in Children: Implications for Academic Achievement," is available online or from the U. of I. News Bureau.

http://bit.ly/1kGOblU

New technology successfully removes heavy metals from water

Technology capable of removing contaminants at low cost and with an efficiency that surpasses existing technologies

The methods traditionally used to remove heavy metals from wastewater have limitations because they only withdraw a certain percentage and the remaining amount is very difficult to remove. This motivated a young graduate researcher at the National Polytechnic Institute (IPN) in Mexico, Gabriel Ramirez Monter, to create a technology capable of removing such contaminants at low cost and with an efficiency that surpasses existing technologies. According to Monter Ramirez, this project led him to design some structures called dendrimers, which are highly branched molecules with shape similar to a shrub or a tree with multiple branches. "Dendrimers adhere and spread on a microfiltration membrane; ie, thin sheets of porous material that are not normally capable of retaining heavy metals due to its pore size. Once placed, it achieves total removal of heavy metal ions in the same way a marine anemone would act, using tentacles to concentrate and catch food; in this case, the branches of the dendrimers capture pollutants," says the researcher. He explains that through dendrimers the team converted a microfiltration membrane into a nanofiltration one. "Another advantage of these structures is that they can be washed and reused, plus the captured metals are removed without problem." Highlighting his business plan, which he called "Nanoestructurados Bromelia", it integrates his master's work, led by Dr. Irina Victorovna Lijanova attached to the Centre for Research and Technological Innovation (CIITEC) of the IPN, which has optimized technologies for removal of heavy metals.

Currently, the entrepreneurial project is linked to the company "Nanotecnología México" that specializes in nanomaterials with applications for the environment and is a leading provider of Mexican Oil (Pemex) in the refining area for sewage cleanup. "The firm was interested in the development of this technology and its commercialization," said Monter Ramirez.

"In Mexico, the problem of heavy metals is associated with industrial progress and important economic activities such as mining or even the oil industry, in both refining and petrochemicals; those are the markets we want to focus on," he stresses.

http://bit.ly/Tm4TEJ

Do Clinical Guidelines Still Make Sense? No

Time to take stock of clinical practice guidelines and ask whether this seemingly rational undertaking has achieved any meaningful goals

Ross E.G. Upshur, BA (Hons) MA, MD, MSc

The last 25 years have seen a dramatic increase in clinical practice guidelines, as well as considerable efforts to establish quality standards, and the growth of an extensive research literature on the uptake and use (or lack thereof) of clinical practice guidelines in routine clinical practice. Perhaps it is time to take stock of these efforts and ask whether this seemingly rational undertaking has achieved any meaningful goals in advancing health care and whether this massive collective undertaking has been worthwhile. Personally, I am skeptical.

It is important to understand the history and evolution of clinical practice guidelines and see their growth as much in sociocultural as scientific terms. Clinical practice guidelines have the virtue of *prima facie* authority and increasingly are used to set standards of practice. Since the 1970s there has been a massive expansion of clinical practice guidelines grounded in the complex forces shaping late 20th century medicine.[1] One potent force is the need for regulatory standardization of practice in the face of documented practice variations and concerns about professional competence. As Weisz and colleagues conclude:

Every effort to regulate increasingly unwieldy health care systems seems to produce complex mechanisms that require even more rules and conventions in order to function. Accordingly we now have layer upon layer of guidelines and protocols….clinical guidelines remain closely linked to the many other forms of regulatory standardization that aim to bring order, predictability and commensurability to an increasingly vast and heterogeneous domain."[1 (p. 716)]

There are 2 dimensions to the vast and heterogeneous domain: the realm of clinical practice guidelines and the increasingly heterogeneous patient population to which these guideline apply.

The increase in number of clinical practice guidelines is impressive. In 1990 there were 73 entries in PubMed. This grew to 7,508 in 2012. Thousands of clinical practice guidelines are produced annually and several hundred are relevant to family medicine. It has been well established that practicing physicians have limited time to read[2] and well documented that adhering to clinical practice guidelines for common chronic diseases is not feasible given the time permitted to practitioners.[3]

Given the sheer number of clinical practice guidelines promulgated by so many diverse authoritative bodies, it is not surprising that uptake by frontline clinicians is low. This is evident in many studies, including several in this issue of *Annals of Family Medicine*. This lack of integration into practice speaks as much to the limitations of the idea of clinical practice guidelines as to perceived limitations of frontline clinicians in maintaining competence and keeping up with the latest research. Success in implementation and improvement of practice seems particularly resource intensive, as the study by Mold et al demonstrates.[4] Considerable effort was required for modest absolute short term improvement in

process indicators. Is there something mistaken about clinical practice guidelines in the first place?

My practice consists of mostly seniors with multiple chronic diseases. I sometimes tease them by asking what their disease and comorbidity is today. This usually brings a quizzical look and request for clarification. I then say I need to figure out which clinical practice guideline to apply, depending on which chronic conditions are most bothersome that day. The jest belies an important and overlooked limitation. Clinical practice guidelines are devised by people with an interest in a single disease for patients who have that particular disease. Recommendations are often made with little or no consideration for other conditions that may plague patients or the priorities they themselves assign to their health conditions. Multimorbidity is the rule, not the exception, and with age this becomes more true.[5] In Canada, an estimated 40% of patients aged over 80 years have 4 or more chronic conditions.[6] There are at least 20 common chronic conditions that afflict older adults. Consequently one finds there are 4, 845 possible combinations of 4 chronic conditions out of 20. It is quite unlikely that any clinical practice guideline will cover this range of possibility in sufficient detail to be directive. It is even less likely that there will be "evidence" from randomized trials that is directive to patients and clinicians and captures this heterogeneity.

Goodman et al attempt to address the problems of creating clinical practice guidelines in the face of an inherently heterogeneous patient population. Their approach, however, runs the risk of adding another layer of complication to the creation of guidelines.[7] Although not explicitly stated by the authors, one possible way forward is to acknowledge the high prevalence of multiple chronic conditions. A second is to be honest about the inflation of uncertainty concerning the harms and benefits of individual therapies as the burden of multiple chronic conditions increases. The utility of any disease-specific clinical practice guideline also declines as this burden increases. Third is the call to increase focus on patient-centeredness. I also suggest seeking alignment of treatment goals among patients, care givers, and clinicians as an important priority.[8] There is also great lack of clarity about the outcomes being pursued with the vast armamentarium of diagnostic and therapeutic power at physicians' disposal. Clarity on desired outcomes in this context is urgently needed.[9]

Perhaps it is time to reconsider the goals of clinical practice guidelines in the context of rethinking the ends of medicine itself in the era of multiple chronic conditions. Clinicians need new skills and tools to provide optimal care for this growing population. An urgent priority is decision aids embedded in clinical practice guidelines to assist patients and clinicians in setting priorities for management choices. Some patients may wish less emphasis on risk reduction, particularly when putative benefits are difficult to discern among multiple competing risks. As well, clinical practice guideline processes should indicate, in the manner of the Grading of Recommendations Assessment, Development and Evaluation Working Group, how multimorbidity influences the quality of evidence and strength of recommendations being made. Perhaps the energy and industry that has characterized the clinical practice guideline process could be focused on creating these skills and tools. This is a task for which family physicians are ideally suited to take leadership.

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The author received funding support from the Canada Research Chair in Primary Care Research.

http://bit.ly/1kCQgrU

Manitoba stops zebra mussel invasion with fertilizer

Canadian conservation authorities on Tuesday celebrated a succesful test using liquid fertilizer to kill invasive Zebra mussels in a lakefront harbor in the western province of Manitoba.

"The treatment process came to a successful end at Winnipeg Beach Harbour on Monday with all... mussels pulled from the harbor confirmed dead after day nine of the estimated 10 day treatment process," Manitoba conservation authorities said a statement. The fight against the mussels will now move to three other nearby harbors, they added.

The small freshwater mussels are native to Eastern Europe and Western Asia. They were discovered in the four Lake Winnipeg harbors last year. "We need to take immediate action to combat the threat of a zebra mussel infestation in Lake Winnipeg... before they spread further and cause permanent damage to the ecosystem or to Manitoba waterways," Conservation Minister Gord Mackintosh said at the time.

Conservation officers killed them off by applying liquid potash to waters for 10 days and closing off the harbor with a gated silt curtain to keep the potash in.

Liquid potash is a plant nutrient mined in vast quantities in neighboring Saskatchewan province and sold to farmers worldwide. Dumping it in a lake does not impact fish, nor water quality, its concentration eventually dissipating.

Zebra mussels (Dreissena polymorpha) reproduce at an alarming rate, damaging harbors and waterways, ships, water treatment plants and power plants, as well as disrupting the aquatic food chain. They were first detected in North America in 1988 in the Great Lakes, after catching a ride in the ballasts of transport ships, before spreading across the continent.

Millions of dollars are spent annually to fight the scourge, with mixed results.

http://bbc.in/1kGWR3q

Centenarians 'outliving diseases of old age'

Centenarians have found a way to beat the common diseases of old age, such as cancer and heart disease, research suggests.

By Nick Triggle Health correspondent, BBC News

The study by King's College London found they were more likely to die of infections such as pneumonia, unlike younger groups of elderly people.

Researchers said 28% of 100- to 115-year-olds died of "old age" and a fifth of pneumonia. Cancer claimed the lives of fewer than 5% and heart disease fewer than 9%. The study was based on an analysis of 36,000 death certificates. By comparison, these diseases were the most common reasons for death among the 80- to 84-year-old age group, with cancer responsible for 25% of deaths and heart disease nearly a fifth.

Boost high quality care

Lead researcher Dr Catherine Evans said the findings raised important questions for health and care services. "Centenarians have outlived death from chronic illness, but they are a group living with increasing frailty and vulnerability to pneumonia and other poor health outcomes.

"We need to plan for healthcare services that meet the 'hidden needs' of this group, who may decline rapidly if they succumb to an infection or pneumonia.

"We need to boost high-quality care-home capacity and responsive primary and community health services to enable people to remain in a comfortable, familiar environment in their last months of life."

The study, published in the journal PLOS Medicine, said this was going to become even more important as the number of centenarians increased.

According to latest Office for National Statistics data, there are more than 13,000 centenarians living in the UK, but by 2066 that number is expected to increase to more than 500,000.

The researchers pointed out that, in the UK, far fewer very old people ended up dying in care homes compared with other European countries, such as the Netherlands and Finland. Dr Evans added: "Hospital admission in the last weeks of life accounts for a third of the total cost of end-of-life care per patient."

http://bit.ly/1hkbZEt

One and done: New antibiotic could provide single-dose option

new single-dose antibiotic is as effective against MRSA as a twice-daily infusion given for up to 10 days

DURHAM, N.C. – In the battle against stubborn skin infections, including methicillin-resistant Staphylococcus aureus (MRSA), a new single-dose antibiotic is as effective as a twice-daily infusion given for up to 10 days, according to a large study led by Duke Medicine researchers.

Researchers said the advantage of the new drug, oritavancin, is its potential to curtail what has been a key driver of antibiotic resistance: a tendency for patients to stop taking antibiotics once they feel better. In such instances, the surviving bacteria may become impervious to the drugs designed to fight them.

"The prolonged activity is what makes oritavancin distinctive," said G. Ralph Corey, M.D., lead author of the study published June 5, 2014, in the New England Journal of Medicine (NEJM). "This drug has a long half-life, which allows for a single-dose treatment."

Corey, a professor of medicine and infectious diseases at Duke University School of Medicine, led a three-year study of oritavancin that encompassed two large clinical trials enrolling nearly 2,000 patients. Findings from the trials will be presented to the U.S. Food and Drug Administration as part of the drug's approval application.

Results reported in the NEJM are for the first of the two clinical trials, which included 475 patients randomized to take the investigational drug, and 479 patients

following a typical regimen of vancomycin, including two infusions a day, for seven to 10 days.

Researchers found that the single intravenous dose of oritavancin was as effective as vancomycin in shrinking the size of the lesion and reducing fever. Both were also similar in rates of requiring a rescue antibiotic.

The new antibiotic also performed similarly to vancomycin in reducing the area of the wound by 20 percent or more within the first 48-72 hours of treatment, and in curing the patients of infection, including those infected with MRSA.

"Having a single-dose drug could potentially prevent hospitalizations or reduce the amount of time patients would spend in the hospital," Corey said.

In addition to Corey, study authors include Heidi Kabler of Sunrise Hospital and Medical Center in Las Vegas; Purvi Mehra and William O'Riordan of Sharp Chula Vista Medical Center in Chula Vista, Calif.; Sandeep Gupta of MV Hospital and Research Center in Lucknow, India; J. Scott Overcash of Sharp Grossmont Hospital in San Diego; Ashwin Porwal of Inamdar Multispecialty Hospital in Pune, India; Philip Giordano of Orlando Health in Orlando, Fla.; Christopher Lucasti of Somers Point, N.J.; and Antonio Perez, Samantha Good, Hai Jiang and Greg Moeck of The Medicines Company.

The study was funded by The Medicines Company, which owns and is seeking to market oritavancin. Corey was a paid consultant to The Medicines Company and the principle investigator of the SOLO trials, the three-year study of oritavancin.

http://bit.ly/1urAdyU

Surprisingly strong magnetic fields challenge black holes' pull

Analysis of radio waves from black holes shows long-neglected magnetic fields have an unexpected presence

A new study of supermassive black holes at the centers of galaxies has found magnetic fields play an impressive role in the systems' dynamics. In fact, in dozens of black holes surveyed, the magnetic field strength matched the force produced by the black holes' powerful gravitational pull, says a team of scientists from the U.S. Department of Energy's Lawrence Berkeley National Laboratory (Berkeley Lab) and Max Planck Institute for Radio Astronomy (MPIfR) in Bonn, Germany. The findings are published in this week's issue of Nature.

"This paper for the first time systematically measures the strength of magnetic fields near black holes," says Alexander Tchekhovskoy, the Berkeley Lab researcher who helped interpret the observational data within the context of existing computational models. "This is important because we had no idea, and now we have evidence from not just one, not just two, but from 76 black holes."

Previously, Tchekhovskoy, who is also a postdoctoral fellow at the University of California, Berkeley, had developed computational models of black holes that included magnetic fields. His models suggested a black hole could sustain a magnetic field that was as strong as its gravity, but there was not yet observational evidence to support this prediction. With the two forces balancing out, a cloud of gas caught on top of the magnetic field would be spared the pull of gravity and instead levitate in place.

The magnetic field strength was confirmed by evidence from jets of gas that shoot away from supermassive black holes. Formed by magnetic fields, these jets produce a radio emission. "We realized that the radio emission from black holes' jets can be used to measure the magnetic field strength near the black hold itself," says Mohammad Zamaninasab, the lead author of the study, who did the work while at MPIfR.

Other research teams had previously collected radio-emission data from "radio-loud" galaxies using the Very Long Baseline Array, a vast network of radio telescopes in the United States. The researchers analyzed this pre-existing data to create radio-emission maps at different wavelengths. Shifts in jet features between different maps let them calculate the field strength near the black hole.

Based on the results, the team found not only that the measured magnetic fields can be as strong as a black hole's gravity, but that they are also comparable in strength to those produced inside MRI machines found in hospitals--roughly 10,000 times greater than the field of the Earth itself.

Tchekhovskoy says the new results mean theorists must re-evaluate their understanding of black-hole behavior. "The magnetic fields are strong enough to dramatically alter how gas falls into black holes and how gas produces outflows that we do observe, much stronger than what has usually been assumed," he says. "We need to go back and look at our models once again."

http://bit.ly/1mjzN7w

Climate not to blame for the disappearance of large mammals

Human expansion caused the mass extinction of large animals over the last 100,000 years

Was it mankind or climate change that caused the extinction of a considerable number of large mammals about the time of the last Ice Age? Researchers at Aarhus University, Denmark, have carried out the first global analysis of the extinction of the large animals, and the conclusion is clear – humans are to blame. "Our results strongly underline the fact that human expansion throughout the world has meant an enormous loss of large animals," says Postdoctoral Fellow Søren Faurby, Aarhus University.

Was it due to climate change?

For almost 50 years, scientists have been discussing what led to the mass extinction of large animals (also known as megafauna) during and immediately after the last Ice Age.

One of two leading theories states that the large animals became extinct as a result of climate change. There were significant climate changes, especially towards the end of the last Ice Age – just as there had been during previous Ice Ages – and this meant that many species no longer had the potential to find suitable habitats and they died out as a result. However, because the last Ice Age was just one in a long series of Ice Ages, it is puzzling that a corresponding extinction of large animals did not take place during the earlier ones.

Theory of overkill

The other theory concerning the extinction of the animals is 'overkill'. Modern man spread from Africa to all parts of the world during the course of a little more than the last 100,000 years. In simple terms, the overkill hypothesis states that modern man exterminated many of the large animal species on arrival in the new continents. This was either because their populations could not withstand human hunting, or for indirect reasons such as the loss of their prey, which were also hunted by humans.

First global mapping

In their study, the researchers produced the first global analysis and relatively fine-grained mapping of all the large mammals (with a body weight of at least 10 kg) that existed during the period 132,000□,000 years ago – the period during which the extinction in question took place. They were thus able to study the geographical variation in the percentage of large species that became extinct on a much finer scale than previously achieved.

The researchers found that a total of 177 species of large mammals disappeared during this period – a massive loss. Africa 'only' lost 18 species and Europe 19, while Asia lost 38 species, Australia and the surrounding area 26, North America 43 and South America a total of 62 species of large mammals.

The extinction of the large animals took place in virtually all climate zones and affected cold-adapted species such as woolly mammoths, temperate species such as forest elephants and giant deer, and tropical species such as giant cape buffalo and some giant sloths. It was observed on virtually every continent, although a particularly large number of animals became extinct in North and South America, where species including sabre-toothed cats, mastodons, giant sloths and giant armadillos disappeared, and in Australia, which lost animals such as giant kangaroos, giant wombats and marsupial lions. There were also fairly large losses in Europe and Asia, including a number of elephants, rhinoceroses and giant deer.

Weak climate effect

The results show that the correlation between climate change – i.e. the variation in temperature and precipitation between glacials and interglacials – and the loss of megafauna is weak, and can only be seen in one sub-region, namely Eurasia (Europe and Asia). "The significant loss of megafauna all over the world can therefore not be explained by climate change, even though it has definitely played a role as a driving force in changing the distribution of some species of animals. Reindeer and polar foxes were found in Central Europe during the Ice Age, for example, but they withdrew northwards as the climate became warmer," says Postdoctoral Fellow Christopher Sandom, Aarhus University.

Extinction linked to humans

On the other hand, the results show a very strong correlation between the extinction and the history of human expansion. "We consistently find very large rates of extinction in areas where there had been no contact between wildlife and primitive human races, and which were suddenly confronted by fully developed modern humans (Homo sapiens). In general, at least 30% of the large species of animals disappeared from all such areas," says Professor Jens-Christian Svenning, Aarhus University.

The researchers' geographical analysis thereby points very strongly at humans as the cause of the loss of most of the large animals.

The results also draw a straight line from the prehistoric extinction of large animals via the historical regional or global extermination due to hunting (American bison, European bison, quagga, Eurasian wild horse or tarpan, and many others) to the current critical situation for a considerable number of large animals as a result of poaching and hunting (e.g. the rhino poaching epidemic).

The results have just been published in the article Global late Quaternary megafauna extinctions linked to humans, not climate change in *Proceedings of the Royal Society B*.

http://www.eurekalert.org/pub_releases/2014-06/afpi-swh060414.php

Study: When hospital workers get vaccines, community flu rates fall

Public health data in California reveals for every 15 hospital vaccinations, there is one fewer case of flu in the community

Anaheim, Calif- For every 15 healthcare providers who receive the influenza vaccination, one fewer person in the community will contract an influenza-like illness, according to a study using California public health data from 2009 – 2012. In an abstract that will be presented on June 7 at the 41st Annual Conference of the Association for Professionals in Infection Control and Epidemiology (APIC), a researcher analyzed archival data from the California Department of Public Health to determine the relationship between vaccinating healthcare personnel against influenza and the rate of influenza-like illness in the surrounding community.

"This study suggests that there is a strong connection between how many healthcare personnel are vaccinated against the flu and how many cases of influenza-like illnesses are reported in the community," said James F. Marx, PhD, RN, CIC, investigator and founder of Broad Street Solutions, an infection prevention consultancy. "More research would be helpful to further understand the impact of vaccinating healthcare workers on community influenza rates."

For the 2011-2012 influenza season, the influenza vaccination rate of California hospital healthcare personnel was 68 percent. According to Marx, if 90 percent of California healthcare personnel were vaccinated – the goal set by the federal government's Healthy People 2020 initiative – there would be about 30,000 fewer cases of influenza-like illness in California.

Influenza-like illness causes more than 200,000 hospitalizations each year and, on average, 24,000 people die as a result, according to the Centers for Disease Control and Prevention (CDC). Currently, vaccination is the single best way to prevent the flu. Marx said: "It is critical that healthcare providers receive the flu vaccine since they come into contact with our most vulnerable community members."

Beginning last flu season, the County of Los Angeles was one of 12 California counties that began requiring healthcare personnel to receive the influenza vaccination or wear protective masks. APIC recommends that all healthcare personnel – in acute care hospitals, long-term care and other facilities – require annual influenza immunization as a condition of employment unless there are compelling medical contraindications. Read the APIC position paper on influenza vaccination.

"Efforts to promote influenza vaccination of healthcare personnel have traditionally focused on protecting patients inside healthcare facilities," said APIC 2014 President Jennie Mayfield, BSN, MPH, CIC. "Now we have evidence that through enhanced healthcare worker vaccination we can protect the broader community. This represents a tremendous public health opportunity."

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APIC Annual Conference 2014, June 7-9 in Anaheim, California, is the most comprehensive infection prevention conference in the world, with more than 100 educational sessions and workshops led by infection prevention experts and attended by nearly 3,000 individuals. The conference aims to provide infection preventionists with tools and strategies that are easily adaptable and can be implemented immediately to improve prevention programs. The Twitter hashtag #APIC2014 is being used for the meeting.

Oral Abstract #426 – Relationship Between Hospital Healthcare Personnel Influenza Vaccination Rates and Community Influenza Rates in California, Saturday, June 7, 2:00-2:15 p.m.

http://bit.ly/1rRakea

Hemorrhagic fevers can be caused by body's antiviral interferon response

A major clue to the mystery of "hemorrhagic fever" syndromes

LA JOLLA, CA - Hemorrhagic fevers caused by Lassa, dengue and other viruses affect more than one million people annually and are often fatal, yet scientists have never understood why only some virus-infected people come down with the disease and others do not.

But now, virologists and immunologists at The Scripps Research Institute (TSRI) have found a major clue to the mystery of "hemorrhagic fever" syndromes. In findings reported this week in an Early Edition of the Proceedings of the National Academy of Sciences, the team showed that Interferon Type I (IFN-I) immune proteins are key drivers of a viral syndrome in mice that closely mimics these human hemorrhagic fevers.

"Blocking IFN-I signaling in certain genetic mouse strains completely prevented disease signs such as vascular leakage leading to death," said TSRI Associate Professor of Immunology Roberto Baccala, who, with TSRI Professor Michael Oldstone, led this study.

While IFN-I proteins traditionally have been considered essential for an effective antiviral response and are still used to treat some chronic viral infections, the new study suggests that these proteins sometimes do much more harm than good - and that blocking them, or specific biological pathways they activate, might be a good therapeutic strategy against hemorrhagic fevers.

Striking Impact

The discovery arose from the team's recent research with the New Zealand Black (NZB) mouse, an inbred laboratory strain whose overactive immune system leads, in midlife, to an autoimmune condition resembling lupus. Curious to see how a viral infection in early life would affect the mice, the team injected a group of the animals with a much-studied mouse virus called lymphocytic choriomeningitis virus (LCMV).

The parental LCMV Armstrong (Clone 53b) caused no symptoms and was quickly cleared by the NZB mice. But a variant (clone 13) that is efficient at infecting cells and causing a persistent infection - yet still causes only mild disease in most other mouse strains - had a strikingly different impact, showing serious signs of illness. Seven to eight days after infection, all the NZB mice that been injected with clone 13 had died.

Further examination revealed leaky blood vessels, fluid and immune virus-specific T cell infiltration into the lungs, decreased platelet counts and other pathological signs reminiscent of human hemorrhagic fevers.

As the scientists knew, LCMV is a member of the family of viruses that includes Lassa virus, which causes one of world's most common hemorrhagic fevers - with a high fatality rate - in a subset of infected patients. "Lassa virus and LCMV infect the same cell type via the same cell-surface receptor," Baccala said. Lassa virus infects hundreds of thousands of individuals annually, culminating in more than 20,000 deaths per year.

Most people infected with Lassa virus experience only mild illness, yet about 20 percent develop the hemorrhagic syndrome. Dengue virus manifests similarly, causing a hemorrhagic syndrome in only a subset of patients. The pathology seen in the LCMV clone 13-infected NZB mice suggested that they could serve as useful models of these human hemorrhagic syndromes, providing clues to how they develop and therapeutic stop-points for their treatment.

A New Target

Baccala and his colleagues soon found evidence that the hyperactivity of the NZB mouse antiviral CD8 cytotoxic T cell response is chiefly to blame for its fatal hemorrhagic disease. The researchers observed powerful CD8+ T cells in higher than normal numbers in affected NZB mouse tissues and a greater number of immune-stimulating molecules on the CD8+ cells' surfaces. This CD8+ T cell overreaction damaged the endothelial cells that line pulmonary blood vessels, causing them to become leaky, which in turn led to the fatal buildup of fluid in the lungs.

IFN-I proteins historically have been known as the chief mobilizers of the protective antiviral response. When Baccala and his colleagues blocked IFN-I signaling, up to a day after infection, the CD8+ T cell response was virtually absent, and levels of clone 13 LCMV rose sharply in the NZB mice. Under these conditions, the mice showed no sign of disease and seemed able to tolerate the high viral load indefinitely - implying that the virus itself is virtually harmless when it doesn't prompt an immune reaction.

"We are now working to determine whether we can target IFN-I itself to treat such conditions or whether we need to target the more specific signals, downstream of IFN-I, that cause pathology," said Baccala.

In addition to Baccala and Oldstone, the co-authors of the study, "Type I interferon is a therapeutic target for virus-induced lethal vascular damage," were Megan J. Welch, Rosana Gonzalez-Quintial, Kevin B. Walsh, John R. Teijaro, Anthony Nguyen, Cherie T. Ng, Brian Martin Sullivan, Alessandro Zarpellon, Zaverio M. Ruggeri, Juan Carlos de la Torre and Argyrios N. Theofilopoulos, all of TSRI. For more information on the paper, see http://www.pnas.org/content/early/2014/05/29/1408148111.abstract

The study was supported by the National Institutes of Health (grants AI099699, AI009484, CA127535, AR53228, AI077719 and HL42846).

http://www.medscape.com/viewarticle/826064?src=rss

More Data on Diet and Dementia

Dementia and Diet: An Update

Bret S. Stetka, MD, Richard S. Isaacson, MD, Hilary P. Glazer, MD

Editor's Note: *While attending the 66th Annual Meeting of the American Academy of Neurology, held in Philadelphia, Pennsylvania, from April 26 through May 3, 2014, Medscape interviewed Richard S. Isaacson, MD, Associate Professor of Neurology at Weill Cornell Medical College in New York, New York, and Hilary P. Glazer, MD, a resident in the Department of Neurology at the University of Miami Miller School of Medicine, via email about their study[1] looking at the possible influence of following Mediterranean dietary patterns on dementia risk.*

Medscape: Can you summarize previously existing data on the potential influence of diet on dementia?

Richard S. Isaacson, MD: There have been a myriad of studies published on dietary interventions in dementia. These range from cohort studies looking at the risk for mild cognitive impairment (MCI) and Alzheimer disease (AD) in normal persons, to randomized trials studying the effect of dietary interventions on cognition, or functional impairment in people with AD. Some dietary interventions have been shown to be effective, whereas others have not, but the investigative quality and statistical power of these studies is quite variable.

Often when these studies are published, they may be highlighted by the mainstream media -- yet physicians, patients, and families may not know how to best interpret the context of varied patient populations, primary outcomes, and effect sizes. The body of evidence suggests that several dietary strategies may be both low-risk and effective in management across the spectrum of AD, including preclinical AD (stage 1), MCI due to AD (stage 2), and dementia due to AD (stage 3), as well as cognitive decline in general.

Medscape: Can you review the objective and methods of your study?

Hilary P. Glazer, MD: We systematically reviewed all studies published since 2002 about dietary interventions to both treat, as well as reduce risk for, MCI and AD. We used currently available American Academy of Neurology guidelines to classify the quality of each study, and then summarized the evidence for each dietary intervention in each clinical scenario studied (strong, moderately strong, weak, or insufficient).

Medscape: Which foods or dietary patterns were associated with an increased or decreased risk for dementia?

Dr. Isaacson: Although the full results of our work are not yet finalized, we reviewed many dozens of studies, including randomized controlled trials (RCTs) and prospective cohort studies, that evaluated various dietary interventions in normal, nondemented persons; patients with MCI; and patients with AD. The interventions included the Mediterranean diet, omega-3 fatty acids, antioxidants, B vitamins, and low-carbohydrate diets.

On the basis of our preliminary review, a combination of B vitamins (folic acid, B_6, and B_{12}) probably improves cognitive impairment in MCI, whereas a Mediterranean diet may improve cognitive function in AD and probably decreases the risk for AD in both MCI patients and nondemented persons.

We also found some promising potential interventions for cognitively normal persons and MCI patients. We found that, for example, specific omega-3 fatty acids are likely to decrease cognitive impairment in MCI, and flavonoids (eg, regular intake of at least 8 oz per week of blueberries and strawberries) may delay symptoms.

On the other hand, there is strong evidence that beta-carotene does not decrease the risk for AD in nondemented patients. There is weak to moderately strong evidence against vitamin E as helpful in nondemented persons and MCI patients; however, a recent RCT found that 2000 IU resulted in slower functional decline and decreased caregiver burden.

Although there is insufficient evidence for a low-carbohydrate diet, one small RCT demonstrated cognitive improvements with a very low-carbohydrate diet, as well as beneficial effect on a number of relevant biomarkers. In addition, preliminary evidence suggests that dietary ketosis may lead to cognitive benefits in a subset of AD patients, although further studies are necessary.

Medscape: On the basis of your findings, what would your take-home message be for clinicians?

Dr. Glazer: AD starts in the brain 20-30 years before the first symptoms of memory loss, and several nutritional approaches, as well as other lifestyle interventions, may be among the most appropriate strategies for managing AD risk that we have today. Dietary interventions should be considered in the management of patients at risk for AD, and probably also in the earliest stages. Aside from being low-risk, these strategies may have other health-promoting benefits (eg, prevention of cardiovascular disease and the metabolic syndrome).

Because currently available pharmacologic interventions may have limited efficacy in some patients, it is necessary to take a more comprehensive, multimodal approach toward AD care. Although our conclusions are based on a preliminary review of the evidence, physicians should consider recommending a Mediterranean diet across the spectrum of AD (stages 1-3), specific omega-3 fatty acids for MCI patients, and flavonoids and B vitamins to those with MCI, as well as to those at risk.

There is less robust evidence toward improving clinical outcomes in dementia due to AD, but this may be attributed to use relatively too late to more meaningfully modify the disease process. As such, dietary interventions may be more helpful in normal, preclinical AD and MCI patients, before they begin to develop functional impairment and dementia.

Our group is currently studying the most effective methods to teach people about these brain-healthy dietary strategies in an effort to understand which methods work best. For busy clinicians who do not have the time or who may not be comfortable with nutritional counseling, we would suggest referral to a registered dietitian, or inviting their patients to participate in an online education research study led by investigators at Weill Cornell Medical College (www.AlzU.org), which uses an online AD nutrition tracking system to longitudinally study outcomes.

1. *Glazer H, Greer C, Barrios D, et al. Evidence on diet modification for Alzheimer's disease and mild cognitive impairment. Program and abstracts of the 66th Annual Meeting of the American Academy of Neurology; April 26-May 3, 2014; Philadelphia, Pennsylvania. Abstract P5.224.*

http://www.eurekalert.org/pub_releases/2014-06/du-cnc060314.php

Complex neural circuitry keeps you from biting your tongue

Similar wiring diagram may be used elsewhere in the brain

DURHAM, N.C. -- Eating, like breathing and sleeping, seems to be a rather basic biological task. Yet chewing requires a complex interplay between the tongue and jaw, with the tongue positioning food between the teeth and then moving out of the way every time the jaw clamps down to grind it up. If the act weren't coordinated precisely, the unlucky chewer would end up biting more tongue than burrito.

Duke University researchers have used a sophisticated tracing technique in mice to map the underlying brain circuitry that keeps mealtime relatively painless. The study, which appears June 3 in eLife, could lend insight into a variety of human behaviors, from nighttime teeth grinding to smiling or complex vocalizations.

"Chewing is an activity that you can consciously control, but if you stop paying attention these interconnected neurons in the brain actually do it all for you," said Edward Stanek IV, lead study author and graduate student at Duke University School of Medicine. "We were interested in understanding how this all works, and the first step was figuring out where these neurons reside."

Previous mapping attempts have produced a relatively blurry picture of this chewing control center. Researchers know that the movement of the muscles in the jaw and tongue are governed by special neurons called motoneurons and that these

are in turn controlled by another set of neurons called premotor neurons. But the exact nature of these connections -- which premotor neurons connect to which motoneurons -- has not been defined.

Senior study author Fan Wang, Ph.D., associate professor of neurobiology and a member of the Duke Institute for Brain Sciences, has been mapping neural circuits in mice for many years. Under her guidance, Stanek used a special form of the rabies virus to trace the origins of chewing movements.

The rabies virus works naturally by jumping backwards across neurons until it has infected the entire brain of its victim. For this study, Stanek used a genetically disabled version of rabies that could only jump from the muscles to the motoneurons, and then back to the premotor neurons. The virus also contained a green or red fluorescent tag, which enabled the researchers to see where it landed after it was done jumping.

Stanek injected these fluorescently labeled viruses into two muscles, the tongue-protruding genioglossus muscle and the jaw-closing masseter muscle. He found that a group of premotor neurons simultaneously connect to the motoneurons that regulate jaw opening and those that trigger tongue protrusion. Similarly, he found another group that connects to both motoneurons that regulate jaw closing and those responsible for tongue retraction. The results suggest a simple method for coordinating the movement of the tongue and jaw that usually keeps the tongue safe from injury.

"Using shared premotor neurons to control multiple muscles may be a general feature of the motor system," said Stanek. "For other studies on the rest of the brain, it is important to keep in mind that individual neurons can have effects in multiple downstream areas."

The researchers are interested in using their technique to jump even further back in the mouse brain, eventually mapping the circuitry all the way up to the cortex. But first they plan to delve deeper into the connections between the premotor and motoneurons.

"This is just a small step in understanding the control of these orofacial movements," Stanek said. "We only looked at two muscles and there are at least 10 other muscles active during chewing, drinking, and speech. There is still a lot of work to look at these other muscles, and only then can we get a complete picture of how these all work as a unit to coordinate this behavior," said Stanek.

The research was supported by grants from the National Institutes of Health (NS077986 and DE019440).

CITATION: "Monosynaptic Premotor Circuit Tracing Reveals Neural Substrates for Oro-motor Coordination," Edward Stanek IV, Steven Chang, Jun Takatoh, Bao-Xia Han, and Fan Wang. eLife, June 3, 2014. DOI: 10.7554/eLife.02511

http://elifesciences.org/content/early/2014/04/30/eLife.02511

http://www.eurekalert.org/pub_releases/2014-06/ul-htg060514.php

Healthy tissue grafted to the brains of Huntington's patients also develops the disease

Healthy tissue grafted to the brains of patients with Huntington's disease also developed signs of the illness

Quebec City - A recent study published in Annals of Neurology reports that healthy human tissue grafted to the brains of patients with Huntington's disease in the hopes of treating the neurological disorder also developed signs of the illness, several years after the graft. This discovery will have profound implications on our understanding of the disease and how to treat it, and may also lead to the development of new therapies for neurodegenerative disorders.

Huntington's disease is a hereditary illness that causes the progressive breakdown of nerve cells in the brain, resulting in major motor, cognitive, and psychiatric impairments. It leads to a gradual loss of autonomy and, eventually, to death. The disease typically appears between age 40 and 50. There is no cure and current treatment methods only help control some of the symptoms without slowing down the disease itself.

"Until now, we thought that Huntington's disease was exclusively the result of a genetic mutation within cells, an intrinsic phenomenon that gradually led to the manifestation of the illness," explains Francesca Cicchetti, professor at the Université Laval Faculty of Medicine, researcher at the CHU de Québec Research Center, and lead author of the study. "However, our work shows that the mutant protein at the source of the illness can also spread from sick to healthy cells, which we did not expect."

These findings by Dr. Cicchetti and her colleagues will have profound implications on the understanding of this pathology and how to treat it. It could also lead to the development of new therapies against other more common neurodegenerative disorders of the central nervous system, as well as diseases related to the propagation of pathological proteins, including Parkinson's and Alzheimer's.

In addition to Francesca Cicchetti, the study's coauthors are: Steve Lacroix, Giulia Cisbani, Nicolas Vallières, Martine Saint-Pierre, and Isabelle St-Amour (CHU du Québec Research Center); Diego Mantovani and Ranna Tolouei (Laboratoire de biomatériaux et bioingénierie, Hôpital Saint-François d'Assise-CHU de Québec); Jeremy M. Skepper (Cambridge Advanced Imaging Centre, University of Cambridge); Robert Hauser (Parkinson's Disease and Movement Disorders Center, University of South Florida); Roger Barker (John van Geest Centre for Brain Repair, University of Cambridge); Thomas B. Freeman (Center of Excellence for Aging and Brain Repair at the University of South Florida).

http://www.eurekalert.org/pub_releases/2014-06/cp-nti052914.php

Neurons transplanted into Parkinson's-affected brains appear healthy after 14 years

When transplanted into the midbrains of adult patients with Parkinson's disease, dopamine neurons derived from fetal tissue can remain healthy for many years.

The findings reported in the Cell Press journal Cell Reports on June 5th suggest that transplanted neurons don't degenerate over time as some had suggested and feared they would, which provides further rationale for pursuing stem cells as a source for transplant-ready dopamine neurons, according to the researchers.

"Our findings show a robust expression of dopamine transporters and a lack of abnormal mitochondrial morphology in implanted dopamine neurons for at least 14 years after transplantation," said Ole Isacson of Harvard University and McLean Hospital. "Our data therefore suggest that transplanted dopamine neurons can remain healthy and functional for decades."

The tremors and other motor symptoms that characterize Parkinson's disease result from the loss of dopamine-producing neurons in part of the midbrain. Neuron transplantation can replace failing neurons with healthy ones from a donor source, but there were questions about the health of those transplanted cells over time.

In the new study, Isacson and his colleagues examined dopamine neurons in five patients who had received fetal cell transplantation four to 14 years earlier. Their examination showed normal expression of dopamine transporters. The transplanted dopamine neurons also appeared to remain healthy and functional over time, with no signs of the degeneration characteristic of Parkinson's disease.

Isacson said it is now clear that fetal cell transplantation has been beneficial for patients with Parkinson's disease; some patients have continued to improve clinically for decades without any medication for the disease at all. However, the therapeutic approach - in which a cell suspension derived from fetuses is injected directly into the relevant portion of the brain - has been offered to only a limited number of patients participating in clinical trials.

The researchers say they now hope to advance on alternative sources for dopamine neurons, particularly from induced pluripotent stem cells made from a patient's own cells. "Our findings are extremely encouraging and timely for the field of regenerative medicine and for advancing stem cell-derived dopamine neuron transplantation as a restoration therapy for Parkinson's disease," Isacson said.

Cell Reports, Hallett et al.: "Long-term dopamine transporter expression and normal cellular distribution of mitochondria in dopaminergic neuron transplants in Parkinson's disease patients."

www.eurekalert.org/pub_releases/2014-06/afri-iga060514.php

Is glaucoma a brain disease?

Scientists find that jigsaw effect in glaucoma patients proves it is

Rockville, Md. - Findings from a new study published in Translational Vision Science & Technology (TVST) show the brain, not the eye, controls the cellular process that leads to glaucoma. The results may help develop treatments for one of the world's leading causes of irreversible blindness, as well as contribute to the development of future therapies for preserving brain function in other age-related disorders like Alzheimer's.

In the TVST paper, Refined Data Analysis Provides Clinical Evidence for Central Nervous System Control of Chronic Glaucomatous Neurodegeneration, vision scientists and ophthalmologists describe how they performed a data and symmetry analysis of 47 patients with moderate to severe glaucoma in both eyes. In glaucoma, the loss of vision in each eye appears to be haphazard. Conversely, neural damage within the brain caused by strokes or tumors produces visual field loss that is almost identical for each eye, supporting the idea that the entire degenerative process in glaucoma must occur at random in the individual eye - without brain involvement.

However, the team of investigators discovered during their analysis that as previously disabled optic nerve axons - that can lead to vision loss - recover, the remaining areas of permanent visual loss in one eye coincide with the areas that can still see in the other eye. The team found that the visual field of the two eyes fit together like a jigsaw puzzle, resulting in much better vision with both eyes open than could possibly arise by chance.

"As age and other insults to ocular health take their toll on each eye, discrete bundles of the small axons within the larger optic nerve are sacrificed so the rest of the axons can continue to carry sight information to the brain," explains author William Eric Sponsel, MD, of the University of Texas at San Antonio, Department of Biomedical Engineering. "This quiet intentional sacrifice of some wires to save the rest, when there are decreasing resources to support them all (called apoptosis), is analogous to pruning some of the limbs on a stressed fruit tree so the other branches can continue to bear healthy fruit."

According to the researchers, the cellular process used for pruning small optic nerve axons in glaucoma is "remarkably similar to the apoptotic mechanism that operates in the brains of people afflicted with Alzheimer's disease."

"The extent and statistical strength of the jigsaw effect in conserving the binocular visual field among the clinical population turned out to be remarkably strong," said Sponsel. "The entire phenomenon appears to be under the meticulous control of the brain."

The TVST paper is the first evidence in humans that the brain plays a part in pruning optic nerve axon cells. In a previous study, Failure of Axonal Transport Induces a Spatially Coincident Increase in Astrocyte BDNF Prior to Synapse Loss in a Central Target, a mouse model suggested the possibility that following injury to the optic nerve cells in the eye, the brain controlled a pruning of those cells at its end of the nerve. This ultimately caused the injured cells to die.

"Our basic science work has demonstrated that axons undergo functional deficits in transport at central brain sites well before any structural loss of axons," said David J. Calkins, PhD, of the Vanderbilt Eye Institute and author of the previous study. "Indeed, we found no evidence of actual pruning of axon synapses until much, much later. Similarly, projection neurons in the brain persisted much longer, as well."

"This is consistent with the partial recovery of more diffuse overlapping visual field defects observed by Dr. Sponsel that helped unmask the more permanent interlocking jigsaw patterns once the eyes of his severely affected patients had been surgically stabilized," said Calkins.

Sponsel has already seen how these findings have positively affected surgically stabilized patients who were previously worried about going blind. "When shown the complementarity of their isolated right and left eye visual fields, they become far less perplexed and more reassured," he said. "It would be relatively straightforward to modify existing equipment to allow for the performance of simultaneous binocular visual fields in addition to standard right eye and left eye testing.

Authors of the TVST paper suggest their findings can assist in future research with cellular processes similar to the one used for pruning small optic nerve axons in glaucoma, such as occurs in the brains of individuals affected by Alzheimer's.

"If the brain is actively trying to maintain the best binocular field, and not just producing the jigsaw effect accidentally, that would imply some neuro-protective substance is at work preventing unwanted pruning," said co-author of the TVST paper Ted Maddess, PhD, of the ARC Centre of Excellence in Vision Science, Australian National University. "Since glaucoma has much in common with other important neurodegenerative disorders, our research may say something generally about connections of other nerves within the brain and what controls their maintenance."

http://www.eurekalert.org/pub_releases/2014-06/uoia-amp060514.php

Alcohol may protect trauma patients from later complications

Injured patients with alcohol in their blood show reduced risk for developing cardiac and renal complications

Injured patients who have alcohol in their blood have a reduced risk for developing cardiac and renal complications, according to a study from the University of Illinois at Chicago School of Public Health. Among patients who did develop complications, those with alcohol in their blood were less likely to die.

The study is published in the June issue of the journal Alcohol.

"After an injury, if you are intoxicated there seems to be a substantial protective effect," says UIC injury epidemiologist Lee Friedman, author of the study. "But we don't fully understand why this occurs."

To better understand the link, Friedman looked at medical complications that are associated with dying in the hospital in relation to patient blood alcohol levels. Other studies have demonstrated that up to 64 percent of post-trauma deaths are attributable to a limited set of later complications.

Nearly 85,000 trauma patients with measured blood alcohol levels were included in the retrospective study, which analyzed 10 years of cases at level I and level II trauma units in Illinois. Children under 16 and patients with certain injuries, such as burns and superficial wounds, were excluded from the study.

Patients' blood alcohol content ranged from 0 to 0.5 percent -- a life-threatening amount, more than six times the level of legal impairment in the U.S.

Overall, 3.2 percent of the patients studied died. Mortality was substantially higher for those who developed complications compared to those who did not (10.3 percent versus 2.1 percent). Among those who died, 43.2 percent had at least one complication.

Blood alcohol concentration was associated with a reduced risk of developing any complication, and with fewer complications overall.

In patients who had alcohol in their blood, cardiac complications were reduced by 23.5 percent. Renal complications were reduced by 30 percent.

The study raises important questions for treatment of traumatic injury.

"Even though alcohol is metabolized quickly by the body, it appears the protective benefit lasts long after there should be only trace amounts in the body," said Friedman, who is assistant professor of environmental and occupational health sciences at UIC.

It is unclear, he said, if alcohol's protective effect comes during the initial period after injury, when alcohol is still present in the blood -- or if the benefit comes from alcohol's metabolites, in tandem with the body's compensatory responses to both the alcohol and the injury.

"The current analysis shows there were reductions in medical complications dominating the cardiovascular system and kidneys, which provides clues to solving this interesting and potentially life-saving puzzle," Friedman said.

http://www.eurekalert.org/pub_releases/2014-06/uosc-fts060214.php

Fasting triggers stem cell regeneration of damaged, old immune system

Results are first evidence of natural intervention triggering stem cell-dependent regeneration of organ or system

In the first evidence of a natural intervention triggering stem cell-based regeneration of an organ or system, a study in the June 5 issue of the Cell Press journal Cell Stem Cell shows that cycles of prolonged fasting not only protect against immune system damage - a major side effect of chemotherapy - but also induce immune system regeneration, shifting stem cells from a dormant state to a state of self-renewal.

In both mice and a Phase 1 human clinical trial, long periods of not eating significantly lowered white blood cell counts. In mice, fasting cycles then "flipped a regenerative switch": changing the signaling pathways for hematopoietic stem cells, which are responsible for the generation of blood and immune systems, the research showed.

The study has major implications for healthier aging, in which immune system decline contributes to increased susceptibility to disease as we age. By outlining how prolonged fasting cycles - periods of no food for two to four days at a time over the course of six months - kill older and damaged immune cells and generate new ones, the research also has implications for chemotherapy tolerance and for those with a wide range of immune system deficiencies, including autoimmunity disorders.

"We could not predict that prolonged fasting would have such a remarkable effect in promoting stem cell-based regeneration of the hematopoietic system," said corresponding author Valter Longo, the Edna M. Jones Professor of Gerontology and the Biological Sciences at the USC Davis School of Gerontology, and director of the USC Longevity Institute.

"When you starve, the system tries to save energy, and one of the things it can do to save energy is to recycle a lot of the immune cells that are not needed, especially those that may be damaged," Longo said. "What we started noticing in both our human work and animal work is that the white blood cell count goes down with prolonged fasting. Then when you re-feed, the blood cells come back. So we started thinking, well, where does it come from?"

Prolonged fasting forces the body to use stores of glucose, fat and ketones, but also breaks down a significant portion of white blood cells. Longo likens the effect to lightening a plane of excess cargo.

During each cycle of fasting, this depletion of white blood cells induces changes that trigger stem cell-based regeneration of new immune system cells. In particular, prolonged fasting reduced the enzyme PKA, an effect previously discovered by the Longo team to extend longevity in simple organisms and which has been linked in other research to the regulation of stem cell self-renewal and pluripotency - that is, the potential for one cell to develop into many different cell types. Prolonged fasting also lowered levels of IGF-1, a growth-factor hormone that Longo and others have linked to aging, tumor progression and cancer risk.

"PKA is the key gene that needs to shut down in order for these stem cells to switch into regenerative mode. It gives the 'okay' for stem cells to go ahead and begin proliferating and rebuild the entire system," explained Longo, noting the potential of clinical applications that mimic the effects of prolonged fasting to rejuvenate the immune system. "And the good news is that the body got rid of the parts of the system that might be damaged or old, the inefficient parts, during the fasting. Now, if you start with a system heavily damaged by chemotherapy or aging, fasting cycles can generate, literally, a new immune system."

Prolonged fasting also protected against toxicity in a pilot clinical trial in which a small group of patients fasted for a 72-hour period prior to chemotherapy, extending Longo's influential past research: "While chemotherapy saves lives, it causes significant collateral damage to the immune system. The results of this study suggest that fasting may mitigate some of the harmful effects of chemotherapy," said co-author Tanya Dorff, assistant professor of clinical medicine at the USC Norris Comprehensive Cancer Center and Hospital. "More clinical studies are needed, and any such dietary intervention should be undertaken only under the guidance of a physician."

"We are investigating the possibility that these effects are applicable to many different systems and organs, not just the immune system," said Longo, whose lab is in the process of conducting further research on controlled dietary interventions and stem cell regeneration in both animal and clinical studies.

The study was supported by the National Institute of Aging of the National Institutes of Health (grant numbers: AG20642, AG025135, P01AG34906). The clinical trial was supported by the V Foundation and the National Cancer Institute of the National Institutes of Health (grant number P30CA014089).

Chia Wei-Cheng of USC Davis School of Gerontology was first author of the study. Gregor Adams, Xiaoying Zhou and Ben S. Lam of the USC Broad Center for Regenerative Medicine and Stem Cell Research; Laura Perin and Stefano Da Sacco of the Saban Research Institute at Children's Hospital Los Angeles; Min Wei of the USC Davis School; Mario Mirisola of the University of Palermo; Dorff and David Quinn of the Keck School of Medicine of USC; and John J. Kopchick of Ohio University were co-authors of the study.

http://www.eurekalert.org/pub_releases/2014-06/ucl-oot060414.php

Our own treacherous immune genes can cause cancer after viral infection

Mutations that cause cancer following human papillomavirus infection are caused by a family of genes that normally protect against viral infections, finds new UCL research

HPV (human papillomavirus) infection is widely known to induce cancer. Many of the mutations that cause this virally-induced cancer are caused by a family of genes that normally combats viral infections, finds new UCL (University College London) research.

This raises the possibility of developing drugs to regulate the activity of these genes to prevent HPV-associated cancers from developing and reduce the ability of existing cancers to evolve resistance to treatments.

The research, published in Cell Reports, shows for the first time that genes from the 'APOBEC' family, which help to fight off viral infection, actually cause mutations that lead to HPV-associated cancer. This research was funded by the Rosetrees Trust, a charity dedicated to supporting pioneering medical research, with additional funding from the Debbie Fund and Cancer Research UK.

"Genes from the APOBEC family encode proteins that modify the DNA of invading viruses, causing mutations that prevent the virus from replicating," explains senior author Dr Tim Fenton, of the Tumour Virus team at the UCL Cancer Institute. "We now provide evidence that they can also cause mutations in our own DNA after HPV infection, leading to cancer."

Over 99% of cervical cancers are caused by HPV infection, and HPV is responsible for approximately 26,700 new cases of cancer each year in the United States. Previous genetic studies have shown associations between APOBEC genes and cancer.

"Our results show that after HPV infection, APOBEC genes cause very specific mutations, with very high frequency in a cancer-promoting gene called PIK3CA, thus leading to tumour development," says co-lead author Dr Stephen Henderson of the Bill Lyons Informatics Centre at the UCL Cancer Institute. "It is not clear why HPV infection causes the APOBEC genes to misbehave and mutate PIK3CA. It could be that the body responds to HPV infection with increased ABOBEC activity, simply making 'friendly fire' more likely. Alternatively, there may well be something about the virus that causes the APOBEC response to wrongly target the body's own genes for mutation."

Mutated PIK3CA (p110α) protein is known to play a key role in the development of a range of cancers, so it is a hot target for new drugs. The new research could explain why particular PIK3CA mutation variants are so commonly found in HPV-associated cancers.

"While it is too early to consider targeting APOBEC genes to prevent tumour formation, it is nonetheless fascinating to work out how HPV drives tumour formation through their activity," says co-lead author Ankur Chakravarthy, a PhD student in the Tumour Virus team. The team's aim is now to learn what happens following HPV infection of cells in which APOBEC genes have been deleted.

The research could also inform screening procedures, as there are variants of particular APOBEC genes that are known to affect cancer risk. For example, a variant of the specific gene APOBEC3B is known to approximately double breast cancer risk. This variant is common in East Asia, with approximately 80% of Indonesians carrying the gene variant. By contrast, only around 2% of the African population carry this variant.

"Previous studies have shown that APOBECs cause mutations in a range of cancers but our finding that they mutate key cancer genes implicates them as drivers of tumour development, particularly in HPV-associated cancers. It will be interesting to see whether such APOBEC variants can predict the risk of developing cancer after HPV infections," says Dr Tim Fenton. "If at-risk groups could be identified by genetic testing, this could have important implications for HPV screening and vaccination programmes."

http://www.eurekalert.org/pub_releases/2014-06/vumc-vsd060514.php

Vanderbilt scientists discover that chemical element bromine is essential to human life

Twenty-seven chemical elements are considered to be essential for human life. Now there is a 28th – bromine.

In a paper published Thursday by the journal Cell, Vanderbilt University researchers establish for the first time that bromine, among the 92 naturally-occurring chemical elements in the universe, is the 28th element essential for tissue development in all animals, from primitive sea creatures to humans.

"Without bromine, there are no animals. That's the discovery," said Billy Hudson, Ph.D., the paper's senior author and Elliott V. Newman Professor of Medicine.

The researchers, led by co-first authors Scott McCall, Christopher Cummings, Ph.D., and Gautam (Jay) Bhave, M.D., Ph.D., showed that fruit flies died when bromine was removed from their diet but survived when bromine was restored.

This finding has important implications for human disease. "Multiple patient groups … have been shown to be bromine deficient," said McCall, an M.D./Ph.D. student. Bromine supplementation may improve the health of patients on dialysis or total parenteral nutrition (TPN), for example.

The report is the latest in a series of landmark papers by the Vanderbilt group that have helped define how collagen IV scaffolds undergird the basement membrane of all tissues, including the kidney's filtering units.

Hudson said the foundation for the discovery about bromine goes back 30 years when he was at the University of Kansas Medical School.

Curiosity about two rare kidney diseases led, in the mid-1980s, to the discovery of two previously unknown proteins that twist around each other to form the triple-helical collagen IV molecule, like cables supporting a bridge. Disease results when these cables are defective or damaged.

Hudson moved to Vanderbilt in 2002.

In 2009, colleagues led by Roberto Vanacore, Ph.D., assistant professor of Medicine, reported in Science magazine the discovery of a novel sulfilimine bond between a sulfur atom and a nitrogen atom that acts like a "fastener" to connect the collagen IV molecules forming scaffolds for cells. A defective bond may trigger the rare auto-immune disease Goodpasture's syndrome. The disorder is named for the late Vanderbilt pathologist and former medical school dean Ernest Goodpasture, M.D., who was best known for his contribution to the development of vaccines.

That discovery led to simple question: how is the bond formed?

In 2012, Bhave, assistant professor of Medicine, Cummings, now a postdoctoral fellow, and Vanacore led the effort that found the answer -- the enzyme peroxidasin.

Conserved across the animal kingdom, peroxidasin also may play a role in disease. An overactive enzyme may lead to excessive deposition of collagen IV and thickening of the basement membrane, which can impair kidney function, they reported in the journal Nature Chemical Biology.

In the current study, to which Vanacore and Andrea Page-McCaw, Ph.D., associate professor of Cell and Developmental Biology, also contributed, the scientists demonstrated the unique and essential role for ionic bromide as a "co-factor," enabling peroxidasin to form the sulfilimine bond. The chemical element bromine is thus "essential for animal development and tissue architecture," they report.

The study was supported in part by National Institutes of Health grants DK018381, DK100094, GM007347, DK097306 and GM073883.

http://www.eurekalert.org/pub_releases/2014-06/hu-anm060514.php

A new model of liver regeneration

Harvard researchers find switch that causes mature liver cells to revert back to stem cell-like state

Harvard Stem Cell Institute scientists at Boston Children's Hospital have new evidence in mice that it may be possible to repair a chronically diseased liver by forcing mature liver cells to revert back to a stem cell-like state.

The researchers, led by Fernando Camargo, PhD, happened upon this discovery while investigating whether a biochemical cascade called Hippo, which controls how big the liver grows, also affects cell fate. The unexpected answer, published in the journal Cell, is that switching off the Hippo-signaling pathway in mature liver cells generates very high rates of dedifferentiation. This means the cells turn back the clock to become stem-cell like again, thus allowing them to give rise to functional progenitor cells that can regenerate a diseased liver.

The liver has been a model of regeneration for decades, and it's well known that mature liver cells can duplicate in response to injury. Even if three-quarters of a liver is surgically removed, duplication alone could return the organ to its normal functioning mass. This new research indicates that there is a second mode of regeneration that may be repairing less radical, but more constant liver damage, and chips away at a long-held theory that there's a pool of stem cells in the liver waiting to be activated.

"I think this study highlights the tremendous plasticity of mature liver cells," said Camargo, who is an associate professor in the Harvard Department of Stem Cell and Regenerative Biology, and based in the Stem Cell Program at Boston Children's Hospital. "It's not that you have a very small population of cells that can be recruited to an injury; almost 80 percent of hepatocytes [liver cells] can undergo this cell fate change."

Much of the work dissecting the biology of these changes and establishing that the dedifferentated cells are functional progenitors was carried out by the Cell paper's first co-authors Dean Yimlamai, MD, PhD, and Constantina Christodoulou, PhD, of Boston Children's Hospital.

The next step, Camargo said, would be to figure out how Hippo's activity changes in cells affected by chronic liver injury or diseases such as hepatitis. In the long term, this work could lead to drugs that manipulate the Hippo activity of mature liver cells inside of patients to spur dedifferentiation and hasten healing.

It might also be possible to control Hippo signaling to grow countless liver progenitor cells in a laboratory dish for transplant, which Camargo's team pursued in the Cell paper using mice born with a genetic liver disease. They cultured healthy liver progenitor cells and transplanted them into the diseased mice. Over a period of three or four months, the transplanted liver cells engrafted and the animals saw improvement of their condition.

"People have been trying to use liver cell transplants for metabolic diseases since the early 90s, but because of the source of cells - discarded livers - they were unsuccessful," Camargo said. "With this unlimited source of cells from a patient, we think that perhaps it's time to think again about doing hepatocyte or progenitor cell transplants in the context of liver genetic disorders."

The observation that mature liver cells dedifferentiate comes after a number of related studies published in the past year from Harvard researchers showing that mature cells in several different internal organs, including the kidneys, adrenal glands, and lungs, are more plastic than we once assumed.

"I think that maybe it is something that people have overlooked because the field has been so stem cell centric," said Camargo, also a Harvard Stem Cell Institute Principal Faculty member. "But I think the bottom line is that the cells that we have in our body are plastic, and understanding pathways that underlie that plasticity could be another way of potentially manipulating regeneration or expanding some kind of cell type for regenerative medicine."

This work was supported by the Harvard Stem Cell Institute, the Stand Up to Cancer-AACR Initiative, the National Institutes of Health, and the Department of Defense.

Cited: Yimlamai et al., Hippo pathway activity influences liver cell fate, Cell (June 5, 2014), http://dx.doi.org/10.1016/j.cell.2014.03.060

http://www.eurekalert.org/pub_releases/2014-06/sjcr-bcp060514.php

Brain circuit problem likely sets stage for the 'voices' that are symptom of schizophrenia

St. Jude Children's Research Hospital scientists report that a disruption in a brain circuit may contribute to the auditory hallucinations of schizophrenia

MEMPHIS, Tenn - St. Jude Children's Research Hospital scientists have identified problems in a connection between brain structures that may predispose individuals to hearing the "voices" that are a common symptom of schizophrenia. The work appears in the June 6 issue of the journal Science.

Researchers linked the problem to a gene deletion. This leads to changes in brain chemistry that reduce the flow of information between two brain structures involved in processing auditory information.

The research marks the first time that a specific circuit in the brain has been linked to the auditory hallucinations, delusions and other psychotic symptoms of schizophrenia. The disease is a chronic, devastating brain disorder that affects about 1 percent of Americans and causes them to struggle with a variety of problems, including thinking, learning and memory.

The disrupted circuit identified in this study solves the mystery of how current antipsychotic drugs ease symptoms and provides a new focus for efforts to develop medications that quiet "voices" but cause fewer side effects.

"We think that reducing the flow of information between these two brain structures that play a central role in processing auditory information sets the stage for stress or other factors to come along and trigger the 'voices' that are the most common psychotic symptom of schizophrenia," said the study's corresponding author Stanislav Zakharenko, M.D., Ph.D., an associate member of the St. Jude Department of Developmental Neurobiology. "These findings also integrate several competing models regarding changes in the brain that lead to this complex disorder."

The work was done in a mouse model of the human genetic disorder 22q11 deletion syndrome. The syndrome occurs when part of chromosome 22 is deleted and individuals are left with one rather than the usual two copies of about 25 genes. About 30 percent of individuals with the deletion syndrome develop schizophrenia, making it one of the strongest risk factors for the disorder. DNA is the blueprint for life. Human DNA is organized into 23 pairs of chromosomes that are found in nearly every cell.

Earlier work from Zakharenko's laboratory linked one of the lost genes, Dgcr8, to brain changes in mice with the deletion syndrome that affect a structure important for learning and memory. They found evidence that the same mechanism was at work in patients with schizophrenia. Dgcr8 carries instructions for making small molecules called microRNAs that help regulate production of different proteins.

For this study, researchers used state-of-the-art tools to link the loss of Dgcr8 to changes that affect a different brain structure, the auditory thalamus. For decades antipsychotic drugs have been known to work by binding to a protein named the D2 dopamine receptor (Drd2). The binding blocks activity of the chemical messenger dopamine. Until now, however, how that quieted the "voices" of schizophrenia was unclear.

Working in mice with and without the 22q11 deletion, researchers showed that the strength of the nerve impulse from neurons in the auditory thalamus was reduced in mice with the deletion compared to normal mice. Electrical activity in other brain regions was not different.

Investigators showed that Drd2 levels were elevated in the auditory thalamus of mice with the deletion, but not in other brain regions. When researchers checked Drd2 levels in tissue from the same structure collected from 26 individuals with and without schizophrenia, scientists reported that protein levels were higher in patients with the disease.

As further evidence of Drd2's role in disrupting signals from the auditory thalamus, researchers tested neurons in the laboratory from different brain regions of mutant and normal mice by adding antipsychotic drugs haloperidol and clozapine. Those drugs work by targeting Drd2. Originally nerve impulses in the mutant neurons were reduced compared to normal mice. But the nerve impulses were almost universally enhanced by antipsychotics in neurons from mutant mice, but only in neurons from the auditory thalamus.

When researchers looked more closely at the missing 22q11 genes, they found that mice that lacked the Dgcr8 responded to a loud noise in a similar manner as

schizophrenia patients. Treatment with haloperidol restored the normal startle response in the mice, just as the drug does in patients.

Studying schizophrenia and other brain disorders advances understanding of normal brain development and the missteps that lead to various catastrophic diseases, including pediatric brain tumors and other problems.

The study's first author is Sungkun Chun, Ph.D., a postdoctoral fellow in Zakharenko's laboratory. The other authors are Joby Westmoreland, Ildar Bayazitov, Donnie Eddins, Amar Pani, Richard Smeyne, Jing Yu and Jay Blundon, all of St. Jude.

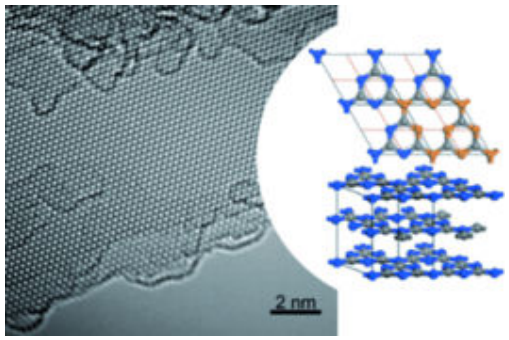
The research was funded in part by grants (MH097742, MH095810, DC012833) from the National Institutes of Health and ALSAC.

http://bit.ly/1kTrMVD

Triazine-based, graphitic carbon nitride as novel two-dimensional semiconductor

Structural analogue of graphene made of carbon and nitrogen that appears to exhibit semiconducting properties

Phys.org - Graphene has been considered a hot candidate for a new generation of silicon-free electronics since the discovery of this two-dimensional form of carbon. However, graphene is not a semiconductor. In the journal Angewandte Chemie, an international team of researchers has now introduced a carbon nitride, a structural analogue of graphene made of carbon and nitrogen that appears to exhibit semiconducting properties.



With a planar, hexagonal, honeycomb structure and freely moving electrons, graphene is, in principle, nothing more than a single-atom layer of graphite. From an electronic point of view, it is a very interesting substance – but it is missing the typical electronic band gap that would make it a semiconductor. This band gap is the difference in energy between the valence band and the conduction band of the electrons. To be effective, this gap must not be too large, so that it allows electrons to easily move from the valence band to the conduction band when excited. Various methods have previously been used to provide graphene with such a band gap. An alternative idea is to make a "graphitic carbon nitride", a material made of carbon and nitrogen, which ought to have properties very similar to graphene. A team of researchers from the University of Liverpool (UK), the University of Ulm (Germany), the Humboldt University in Berlin (Germany), the Aalto University (Finland), University College London (UK), and the Max Planck Institute of Colloids and Interfaces in Potsdam (Germany) has now been able to make such a material for the first time.

Transmission electron microscopy and scanning force microscopy, as well as X-ray crystallographic examinations proved that the thin crystalline films are a triazine-based, graphitic carbon nitride (TGCN). Triazines are six-membered rings containing three carbon and three nitrogen atoms. The new material consists of such triazine rings, with additional nitrogen atoms connecting the rings into groups of three to make a two-dimensional layer. The team led by Andrew I. Cooper and Michael J. Bojdys believes that these layers are not fully planar, but are instead slightly wavy.

TGCN thus has a structure similar to that of graphite, however - as hoped - it is a semiconductor. The films produced consisted of between three and several hundred layers of atoms with a direct band gap between 1.6 and 2.0 eV. During the production process, the layers of TGCN are preferentially deposited onto substrates. The crystallization of TGCN on the surface of insulating quartz offers potential for practically relevant applications. This may be a step on the way to the post-silicon era of electronics.

More information: Algara-Siller, G., Severin, N., Chong, S. Y., Björkman, T., Palgrave, R. G., Laybourn, A., Antonietti, M., Khimyak, Y. Z., Krasheninnikov, A. V., Rabe, J. P., Kaiser, U., Cooper, A. I., Thomas, A. and Bojdys, M. J. (2014), "Triazine-Based, Graphitic Carbon Nitride: a Two-Dimensional Semiconductor." Angew. Chem. Int. Ed.. doi: 10.1002/anie.201402191

http://phys.org/news/2014-06-early-humans-westward-ho-dental.html

Early humans were "Westward Ho," dental records reveal

Early humans, or hominins, stretched further west - into today's Central Africa – t han previously known, according to findings by a research team that included NYU anthropologist Shara Bailey.

The results, which appeared in the journal PLOS ONE, expand the range of early hominins significantly farther west and suggest that they made use of a wide range of geographic locations and likely ecological conditions. They also reveal a need for a shift in our paradigm about where to search for early hominins.

"While the eastern branch of the Rift Valley is an important place for early human evolution, this find suggests additional results may come from farther west than we once thought," says Bailey.

The team's conclusions are based on the discovery of a molar in the western, or Albertine, branch of the East African Rift, which had previously yielded several discoveries of more recent fossil humans.

The tooth was originally unearthed in the 1950s in what is now the Democratic Republic of Congo. But its age had not been confirmed. Moreover, previous scholarship had limited early hominins to the Rift Valley's eastern branch - a few hundred miles separate the eastern and western branches - with the assumption that this discovery was of a more recent fossil human.

Advances in technology helped prompt the researchers to more closely examine the 60-year-old find.

Bailey, whose expertise centers on teeth, conducted a comparative analysis of the molar with those from early hominins discovered in other regions. She focused on its enamel surface while Matthew Skinner, an anthropologist at University College London, examined its underlying structure.

Their analysis revealed a remarkable consistency in dimensions and enamel thickness with previously discovered molars of east and southern African early hominins. Moreover, the structure was quite distinct from the teeth of Homo sapiens - modern humans.

http://bit.ly/1j9wWxa

Decline of monarch butterflies linked to modern agriculture

Loss of milkweed plants in the midwest reduces caterpillar survival.

by Kate Prengaman - June 5 2014, 11:21pm TST

The massive migration of monarch butterflies is amazing - the insects go from grazing on milkweed plants as caterpillars in the midwest to spending winters in Mexico. But Monarch populations have been on the decline for some time, with a variety of factors being considered: lost habitat in Mexico, damage from pesticides, or climate change.

Conservation strategy for a species that traverses thousands of miles is complicated business, so a team of scientists from the University of Guelph decided to sort out which factors were the most responsible for the monarch's population declines - changes at the breeding grounds, the wintering sites, or climate changes.

Monarch butterfly in the butterfly house at Palmitos Park in Gran Canaria.
William Warby

Their conclusions suggest that we can't blame deforestation in Mexico for this environmental problem. The monarchs are suffering from a lack of milkweed, the only plant the caterpillars eat. In fact, a model built by the researchers suggested that monarch populations were four times more sensitive to the loss of milkweed on their breeding grounds than the loss of the forested habitat in which they spend the winters.

The spatial model was built using population dynamics data, which incorporated locations and life stages, known survival rates at different stages, and standard reproductive success. It used this data to predict how various changes in the system, from climate to habitat, would affect the insects' complex lives.

Using their model, the scientists found a 21 percent decline in milkweed abundance between 1995 and 2013. The largest declines, in the midwest, line up with the largest declines in butterfly population.

The monarchs depend on milkweed - it's the plants' chemical defenses that give the butterflies their infamous unpalatability. The adults only lay their eggs on milkweed to give the larvae a strong start in life, so the researchers say the plants' decreasing abundance has implications across the life cycle.

Milkweed is disappearing, they write, because of the increasingly intensive land use of agriculture; although the study didn't do primary research on this connection, it has been demonstrated by others. Milkweed is still common in nature preserves, gardens, and along roadways, but for farmers, it's a weed. In the corn belt, agricultural land is being used more intensely, which means fewer buffers and borders of natural plants between the fields, and more powerful herbicides to reduce the number of weeds. The invention of herbicide-tolerant corn and soybeans has made growing more efficient, since it allows farmers to spray and kill off everything else, but it's bad news for milkweed and monarchs.

This doesn't come as a surprise to everyone - Chip Taylor, of Monarch Watch and the University of Kansas, talks about how shifts in the agricultural practices in the midwest have reduced milkweed and, therefore, caterpillars. But many thought that habitat protections in Mexico would solve the problem.

Efforts to protect the wintering habitat in Mexico are important too, the scientists write, because the forest cover protects the insects as they huddle together in million-monarch masses to survive the cold. But those protections aren't enough.

Taylor offers a simple suggestion, and the Guelph scientists back him up: if we want monarchs, we need to plant more milkweed, perhaps in gardens and along roadsides. But to stop the decline, the scientists say we also need to preserve undeveloped lands in the corn belt, like parks and prairies, where milkweed can feed caterpillars that grow into beautiful butterflies.

Journal of Animal Ecology, 2014. DOI not yet available.

http://www.medscape.com/viewarticle/825908

ID Update 2014: New and Emerging Threats

Hi, everyone. I am John Bartlett, at the American College of Physicians Internal Medicine 2014 meeting in Orlando, Florida, giving an update on infectious diseases.

John G. Bartlett, MD

I am going to move to the topic of our epidemic of epidemics. We have broken a lot of records lately.

We had a record number of Lyme disease cases. It was not necessarily a big increase in Lyme disease because the Centers for Disease Control and Prevention (CDC) used a different method to find cases and it went up 10-fold.[1] But it is pretty amazing: 300,000 cases in 2013.

Epidemic of Epidemics: Records

- **Lyme disease 2013:** 300,000 cases; a 10-fold increase
- **West Nile virus 2012:** 5674 cases
- **Coccidiomycosis 1998-2011:** 10-fold increase, with 98% in California and Arizona
- **Malaria 2011:** record 1925 cases
- **Measles 2013:** 189 cases, the most in 18 years
- **Pertussis 2013:** 41,800 cases and 18 deaths
- **Meningococcal meningitis 2013:** 3 outbreaks
- **Norovirus 2013:** 20 million cases
- **Ebola 2013-2014:** 134 cases and 84 deaths in Guinea
- **Chikungunya 2013:** 26 Caribbean travelers
- **"Hartland virus"2012-2014:** Tick-borne, 8 cases Tennessee and Missouri
- **Mumps 2013-2014:** 116 cases Ohio
- **Measles 2014:** 25 cases in New York City
- **"Polio-like virus" 2014:** 25 children in California (? Enterovirus 68)

For West Nile virus, 61% of the 5600 cases were neuroinvasive cases, with a bill of almost $1 billion to take care of those patients.[2] That is a big one.

The rates of coccidioidomycosis have gone up by 10-fold in 10 years.[3] Some of that is probably due to an increase in reporting. If you have a patient from California or Arizona or someone who has traveled there, this becomes a diagnostic possibility that you have to think about.

We set a record for malaria in 2011.[4] They were mostly caused by *Plasmodium falciparum* -- 61% of the cases.

Acute, Potentially Lethal Respiratory Tract Infection (RTI) Viruses in Travelers

When to suspect:

MERS-Co-V	Influenza A H7N9
• Unexplained severe lower RTI, with travel to Arabian Peninsula or neighboring countries within past 14 days	• Unexplained severe lower RTI, with travel to China within 10 days prior to onset of symptoms
• Diagnosis: Molecular test from CDC in most state labs	• Diagnosis: Molecular test from CDC available in most state labs
• Infection control: Strict precautions	• Infection control : Strict precautions
• Treatment: None; possibly interferon/ribavirin	• Treatment: Neuraminidase inhibitor (oseltamivir)

Measles outbreaks are all over the place. Measles and pertussis are a little bit different. Measles reflects the absence of vaccination, and pertussis generally reflects an inadequate vaccination as a result of changes in the pertussis vaccine strategies or recommendations.

We had 3 outbreaks of meningococcal meningitis in 2013 and another one in 2014. Two of these epidemics were in Los Angeles and New York,[5] in gay men, and there were 2 college outbreaks.[6] Norovirus had 20 million cases.

Ebola virus had a lot this year. These are recent but not updated data.

Chikungunya is very interesting. There was a big epidemic in the Caribbean and in travelers to that area.[7] Perhaps more important, we expect chikungunya and dengue fever to be endemic in the United States, certainly in Florida and some of the border states of Mexico, and that is worrisome for the future. Neither one of those are pretty diseases. Chikungunya is a mosquito-borne disease and it can cause arthralgia and arthritis that last for a very long time.

Influenza H5N1/H7N9

- **Risks**: Poultry contact; limited human-transmission; no "sustained" human-h transmission shown to date.
- Needs **hemagglutinin (HA) mutation** t promote binding to human epithelial c This will be the mutation likely to prod global pandemic.
- **Only a few flu strains caused pandem** past 95 years: H1N1, H2N2, and H3N2

More recent is the tick-borne "Heartland virus" in Tennessee and Missouri. I know anything about this until I read about it.

Mumps and measles had recent cases in Ohio and New York City.

A polio-like virus, which I think is probably enterovirus 68, seems to have qu a bit in recent months, but there were 25 cases in kids who look like they are disabled.

Of course, the big one is influenza. H5N1 and H7N9 are ready to explode. W they need is the hemagglutination mutation that will promote pathogenicity i human epithelial cells, and if that takes place, we are in real trouble. Don't fo the previous histories of influenza and what it can do. Remember that oseltar a good drug. It is certainly not a great drug, and we had a huge problem of resistance in 2006. It doesn't take much to develop oseltamivir resistance.

A warning about Middle East respiratory syndrome coronavirus (MERS-CoV influenza H7N9: If you have a patient who has a serious lower respiratory tra infection and has traveled to the Arabian peninsula or neighboring countries past 14 days, think about MERS-CoV.

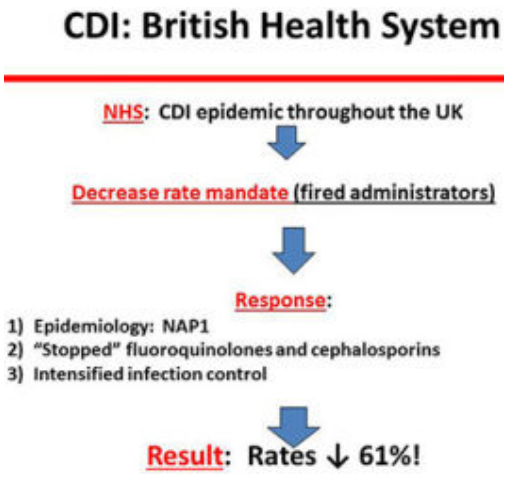
The molecular diagnostic test for it is available in most state laboratories. No treatment is available at present, but interferon/ribavirin is possible. It is highly lethal. The same can be said to some extent for influenza A H7N9, which is in China. Travelers to China within the past 10 days who have a serious unexplained respiratory illness should be suspected to have H7N9 and have a respiratory molecular test don virus is sensitive to oseltamivir. I can't tell you how good it is, but it is sensit

New *Clostridium difficile* Infection (Epidemic Strain: NAP1(027)

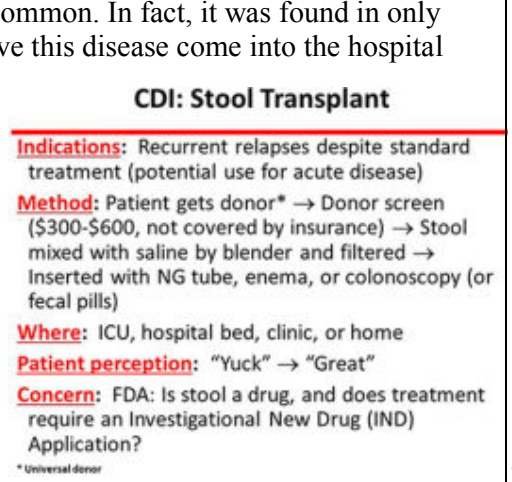
- Resistant to fluoroquinolones
- ? More virulent and relapse
- Hospital epidemics
- Epidemic strain – Europe, US, Canada
- Now, 027 and 078 are most worrisome
- Clinicians rarely know strain

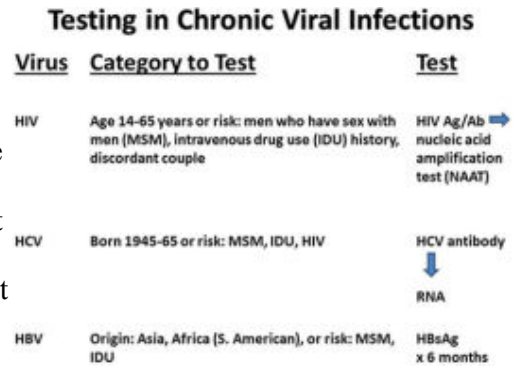
Clostridium difficile is a pet of mine. It got legs in the early 2000s as a result of the epidemic of the NAP1 strain.[8] Its epidemiologic explosion was probably a result of resistance to fluoroquinolones (and how we fed it with our use of those drugs) rather than its virulence. Nevertheless, that epidemic traveled across Europe and North America and accounted for much of what we have seen.

More recently, there have been some very interesting epidemiology studies in the UK, in part because the country demanded a reduction in cases. They have managed to reduce *C difficile* infection (CDI) by 61%, and they claim that most of this is the result of restricting the use of fluoroquinolones and, to some extent, cephalosporins.[9] They also showed that our epidemiologic concepts of how you get CDI are quite different from traditional teaching. They showed this with molecular sequencing, which has shown that patient-to-patient transmission is not common. In fact, it was found in only 23% of cases. Most of the patients who have this disease come into the hospital already colonized with it.



Stool transplantation is hot in the field of infectious diseases. The first one was done in 1958, but now we have a surge of papers and a surge of enthusiasm. Bottom line: Stool transplant by Infectious Diseases Society of America and US Food and Drug Administration (FDA) guidance in 2013/2014 -- indications for stool transplant are relapses times 3 or more, also for acute disease. There the data are good but limited. You can transplant the stool at a hospital, in a clinic, or at home. You can put it in by endoscopy, enema, nasogastric tube, or by feeding capsules. Who selects the donor? It is usually the patient or a "universal donor" who is screened, but there are some other sources, such as a group in Canada that is isolating components of the microbiome. OpenBiome is a group of students who offer stool from universal donors for $250. The screening tests are expensive. Donors must be screened for hepatitis, HIV, enteric pathogens, and so forth, and it is not covered by insurance. Patients need to know that.





Some of us are a little worried about putting stool from Joe into Sally because the microbiome is turning out to play a rather prominent role in health. We don't really know the long-term consequences of this. We have no red light so far, but it is something to keep in mind. Finally, the FDA has announced that stool is a drug and therefore requires a treatment Investigational New Drug Application. They say that you need patient consent. We always have it anyway. You have to do the donor screen. And they also say that either the patient or the doctor needs to know the source of the stool, which is something we don't necessarily always know.

Finally, I will mention 2 chronic viral infections. The one to emphasize is the second one on the list, which is hepatitis C. Hepatitis C has undergone a revolutionary change from no treatment to very poorly tolerated treatment to miraculous treatment for cure. This will be the first chronic viral infection that is *cured*. The light is at the end of the tunnel and it is coming very fast with FDA approval. These drugs promise to cure more than 90% of patients with 10-16 weeks of an oral pill once daily. It is just amazing.

What is your responsibility? Most people watching this will not be doing hepatitis C treatment. It is a real specialty in medicine. What primary care needs to do is to find the cases. At risk are people who were born between 1945 and 1965 -- the the baby boomers. They account for 75% of cases and most of them don't know it.

If you have those patients, you need to test hepatitis C antibody and then reflex to hepatitis C RNA. Of course, you also screen anybody who has the standard risk factors.

I might also mention HIV, which continues to be a chronic disease that is treatable. One thing to mention is that the testing is about to change again to a test that will detect antigen as well as antibody. I just want to make the audience aware of that.

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http://www.bbc.com/news/science-environment-27688511

Traces of another world found on the Moon

Researchers have found evidence of the world that crashed into the Earth billions of years ago to form the Moon.

By Pallab Ghosh Science correspondent, BBC News

Analysis of lunar rock brought back by Apollo astronauts shows traces of the "planet" called Theia. The researchers claim that their discovery confirms the theory that the Moon was created by just such a cataclysmic collision. The study has been published in the journal Science.

The accepted theory since the 1980s is that the Moon arose as a result of a collision between the Earth and Theia 4.5 billion years ago. Theia was named after a goddess in Greek mythology who was said to be the mother of Selene, goddess of the Moon. It is thought to have disintegrated on impact with the resulting debris mingling with that from the Earth and coalescing into the Moon.

It is the simplest explanation, and fits in well with computer simulations. The main drawback with the theory is that no-one had found any evidence of Theia in lunar rock samples.

Earlier analyses had shown Moon rock to have originated entirely from the Earth whereas computer simulations had shown that the Moon ought to have been mostly derived from Theia.

Alien origin

Now a more refined analysis of Moon rock has found evidence of material thought to have an alien origin.

According to the lead researcher, Dr Daniel Herwartz, from the University of Goettingen, no-one has found definitive evidence for the collision theory, until now.

"It was getting to the stage where some people were suggesting that the collision had not taken place," he told BBC News. "But we have now discovered small differences between the Earth and the Moon. This confirms the giant impact hypothesis." But the difference, some say, could be explained by material absorbed by the Earth after the Moon formed.

And Prof Alex Halliday of Oxford University, is among many scientists who are surprised that the difference between the Theian material found in the Moon rock and the Earth is so small.

"What you are looking for is a much bigger difference, because that is what the rest of the Solar System looks like based on meteorite measurements," he said.

Dr Herwartz measured the difference in what is called the isotopic composition of the oxygen contained in rocks on Earth and Moon rock. This is the ratio of different forms of oxygen.

Studies of meteorites from Mars and the outer solar system show that these ratios are markedly different - rather like a fingerprint. So Prof Halliday and others are puzzled by the fact that the fingerprints of Earth and Theia seem almost identical.

Similar composition

One possibility is that Theia was formed very close to the Earth and so had a similar composition. If that was the case, it raises the possibility that the assumption that each planet in the current Solar System has a markedly different fingerprint needs to be revisited, according to Prof Halliday.

"It raises the question of how well the meteorites from Mars and the asteroid belt in the outer Solar System are representative of the inner Solar System? We do not have samples from Mercury or Venus.

"They may well be similar to the Earth. If that is the case then all the arguments over the similarities of the Earth and the Moon fall away," he told BBC News.

Dr Mahesh Anand from the Open University described the research as "exciting" but noted that the data was from just three lunar rock samples.

"We have to be cautious about the representativeness of these rocks of the entire Moon, and so further analysis of a variety of lunar rocks is required for further confirmation," he said.

Other theories have been proposed to explain why the composition of the Earth and Moon are so similar: one is that the Earth spun much faster before impact, another is that Theia was much larger than current models suggest.

An alternative, controversial, theory proposed by Prof Rob de Meijer of Groningen University in the Netherlands is that the Earth's crust and mantle was blown into space by an accumulation of nuclear material 2,900km (1,800 miles) below the surface. It was this debris that clumped together to form the Moon.

He told BBC News that the new finding - demonstrating that there was a difference in the composition of the Earth and the Moon - did not change his view.

"The difference is too small," he said. "We don't know how the Moon was formed. What we need are manned missions to the Moon and a search for rocks deeper under the lunar surface, that have not been polluted by meteorite impacts and the solar wind."

http://www.eurekalert.org/pub_releases/2014-06/ggph-acm060514.php

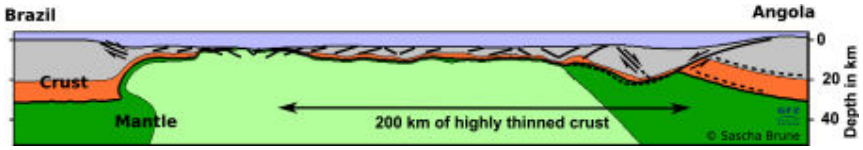
Asymmetric continental margins and the slow birth of an ocean

When South America split from Africa 150 to 120 million years ago, the South Atlantic formed and separated Brazil from Angola.

The continental margins formed through this separation are surprisingly different. Along offshore Angola 200 km wide, very thin slivers of continental crust have been detected, whereas the Brazilian counterpart margin features an abrupt transition between continental and oceanic crust.

For decades, geoscientists have struggled to explain not only why the amount of thinning and the geometries of opposite rifted continental margin are not symmetric, but also why wide margins are often underlain by highly thinned continental crust. Now geoscientists from the German Research Centre for Geosciences (GFZ), the University of Sydney and the University of London have found an explanation, published in the current issue of 'Nature Communications'. Using high-resolution computer models and geological data from the South Atlantic margins, they discovered that the centre of the rift, where the continental crust gets actively thinned through faulting, does not stay fixed during continental break-up, but migrates laterally.

"We could show that rifts are capable of moving sideways over hundreds of kilometres", says Dr Sascha Brune of the GFZ. "During rift migration, the crust on one side of the rift is weakened by hot upwelling material in Earth's mantle, whereas the other side is slightly stronger as the crust there is colder. New faults form only on the warm, weak rift side, while those of the strong side become inactive." This leads to a sideways motion of the rift system, which is equivalent with conveying crustal material from the South American plate to the African plate. These transferred crustal blocks are strongly extended by the rift and finally constitute the enigmatic thin crustal slivers of the African margin.



***"Asymmetry of the South Atlantic continental margins. Shown is a model cross section for the South Atlantic, shortly after the separation of Africa and South America 120 million years ago.* (Image: Sascha Brune, German Research Centre for Geosciences GFZ)"**

Such a relocation of a rift takes its time: during the formation of the present-day Angolan and Brazilian margins, the rift centre migrated more than 200 km westward. This delayed continental break-up and the generation of oceanic crust by up to 20 million years. The new models reveal that extension velocity plays a crucial role in understanding the widths of South Atlantic margins: faster crustal extension leads to longer rift migration and hence to more pronounced asymmetry of the generated continental margins.

Rifts constitute an important tectonic element of our planet. They are responsible for the shape of today's continents, and their activity still continues at present. Illustrating a new aspect of plate tectonic theory, this study shows that during continental break-up, large amounts of material can be conveyed from one side of the plate boundary to the other, a process that has not been yet accounted for. The new models and analyses provide an important stepping-stone toward a comprehensive understanding of rift processes and continental margin formation.

Brune, S./Heine, C./Marta Pérez-Gussinyé, M./Sobolev, S.: "Rift migration explains continental margin asymmetry and crustal hyper-extension", Nature Communications. 5:4014 doi: 10.1038/ncomms5014 (2014), 06.06.2014

http://phys.org/news/2014-06-na-ion-batteries-effective-alternative-li-ion.html

New process designed to make Na-ion batteries an effective alternative to Li-ion

Researchers develop affordable battery alternative

Phys.org - As the demand for rechargeable lithium-ion (Li-ion) batteries has grown, the battery industry has found itself facing a problem of supply-and-demand. Lithium is not an abundant element, and most lithium deposits are found in only a handful of countries. Both problems make its long-term availability and cost uncertain. In a paper published in the June 4 issue of Nature Communications, University of Maryland professors Chunsheng Wang and John Cumings explain how a modified version of a Li-ion battery anode could allow manufacturers to replace the lithium with a more common element.

Sodium (Na), an earth-abundant and inexpensive element, shares many properties with lithium, but so far has not been able to replace it. The best strategies for creating Li-ion batteries often can't be adapted for use in Na-ion batteries, rendering them a laboratory curiosity and keeping them out of the market.

The main problem is the atom's size. Sodium ions are larger than lithium ions, which limits the kinds of materials that can be used in a Na-ion battery anode, the component into which the positively charged ions flow. Graphite (a form of pure carbon) is among the most superior options, and is also the most common in Li-ion batteries. When creating graphite anodes, lithium ions are easily electrochemically intercalated (embedded) into its layered structure, but for sodium ions it's a tight squeeze, and the result is a battery with sluggish performance and low capacity.

The solution, Wang and Cumings have discovered, is to increase the space between the individual layers of carbon that make up the graphite. Their team starts with graphite oxide, a common industrial material formed by exposing graphite to an aggressively corrosive solution that stuffs oxygen between its layers. The oxygen atoms bond with each carbon layer, pushing and holding them apart. However, the resulting material is inevitably "overstuffed," leaving no room for sodium ions to get in. To make the material suitable for use in Na-ion batteries, some of the oxygen must be removed.

The solution to this second problem was developed by the paper's first author, Department of Chemical and Biomolecular Engineering (ChBE) graduate student Yang Wen. Wen heats the expanded, oxidized graphite to high temperatures and floods it with argon gas, causing it to decompose. In this process, oxygen bonded to carbon breaks away in the form of either carbon monoxide (CO) or carbon dioxide (CO2) gas, which is caught up and removed by the argon gas flow. Wen's key discovery is the precise combination of temperature and duration for the reaction. Her technique ensures that enough oxygen atoms have been removed to let the sodium ions in, but enough are left behind to prevent the expanded graphite from collapsing. The process may be likened to jacking up every floor of a multi-storey building to accommodate taller tenants, and then removing excess scaffolding until only the required support beams remain.

After testing the material both in experimental batteries and in a transmission electron microscope for realtime observations, the team found that Na-ion battery anodes manufactured with the expanded graphite had good energy density and retained 73 percent capacity after 2000 charge/discharge cycles.

"Expanded graphite is already commercially available," explains Wang, an associate professor of ChBE, "but industry uses a different method to make it. If they follow Yang's procedure, they can use it to make expanded graphite suitable for sodium-ion batteries." However, he adds, "they won't be as powerful as lithium-ion batteries. You'll need more of them to get the same amount of power, but the cost is so much lower it will make up for it."

Cumings, an associate professor from the Department of Materials Science and Engineering, agrees. "Sodium-ion batteries are also heavier, so for now they're not suitable for most vehicles and airplanes. But for something like building or grid-level power storage–where they're just going to sit there–the fact that you get more kilowatt hours per dollar becomes a strong selling point."

Paper: ww.nature.com/ncomms/2014/140604/ncomms5033/full/ncomms5033.html

http://phys.org/news/2014-06-evidence-speedy-core-formation-solar.html

Researchers find evidence of speedy core formation in solar system planetesimals

Evidence of faster than thought core formation of planetesimals in our solar system

Phys.org - A combined team of researchers from Germany, Switzerland and the U.S. has found evidence of faster than thought core formation of planetesimals in our solar system. In their paper published in the journal Science, the team describes how they came up with a new approach to using tungsten isotope dating in a way that overcame the problem of cosmic rays affecting accuracy. Tim Elliot offers a Perspective piece in the same issue delving further into the work by the team and explains how the new findings are likely to lead to better dating for planetary development in general.

Scientists believe approximately 4.6 billion years ago, our solar system was little more than a star surrounded by a molecular cloud. That cloud eventually coalesced into a proto-planetary disk which eventually coalesced further into planetesimals. Planets and moons and other bodies in the solar system came about as a result. But, one thing that has puzzled space scientists was the rate at which the cores of the planetesimals formed, or put another way, how soon after the formation of solar system, did the cores start to form? To come up with a good approximation, the researchers looked to existing iron meteorites - they are believed to been the creative force behind core formation.

To determine the age of five existing iron meteorites, the researchers used tungsten radioactive isotope dating, an approach used before. Such prior efforts were hobbled in their accuracy, however, by the impact of cosmic rays over time. To get around that problem, the researchers used platinum isotope compositions. Doing so allowed the researchers to calculate that core formation of early planetesimals likely began as early as 100,000 years to 300,000 years after the formation of the solar system.

These findings help explain why the materials that made up the bodies currently in our solar system weren't blown away by the sun - previous estimates suggested core

formation took up to twenty million years, enough time to push such materials beyond our stars' gravitational pull. With such a short formation time, however, the cores of the developing planetesimals would have formed before they were pushed too far out, allowing them to be captured by the tug of the sun's gravity.

More information: Protracted core formation and rapid accretion of protoplanets, Science 6 June 2014: Vol. 344 no. 6188 pp. 1150-1154 DOI: 10.1126/science.1251766

ABSTRACT

Understanding core formation in meteorite parent bodies is critical for constraining the fundamental processes of protoplanet accretion and differentiation within the solar protoplanetary disk. We report variations of 5 to 20 parts per million in 182W, resulting from the decay of now-extinct 182Hf, among five magmatic iron meteorite groups. These 182W variations indicate that core formation occurred over an interval of ~1 million years and may have involved an early segregation of Fe-FeS and a later segregation of Fe melts. Despite this protracted interval of core formation, the iron meteorite parent bodies probably accreted concurrently ~0.1 to 0.3 million years after the formation of Ca-Al–rich inclusions. Variations in volatile contents among these bodies, therefore, did not result from accretion at different times from an incompletely condensed solar nebula but must reflect local processes within the nebula.

http://bit.ly/1uExaDP

Body that formed the Moon came from a different neighborhood

The body that smacked into Earth has a distinctive elemental signature.

by Shannon Palus June 7 2014, 12:37am TST

The giant impact hypothesis goes like this: 4.5 billion years ago, a Mars-sized body named Theia slammed into the Earth. The collision launched magma - some from Theia and some from Earth - into orbit around our planet. The magma condensed and cooled into the rocky sphere that we see in the sky, our Moon.

This scenario - explored through collision models - handily explains the way that our Moon spins, its small core, and its lack of water. It is the most widely accepted scientific response to the question of how the Moon came to be hung in our sky.

But the giant impact hypothesis has suffered from one major problem: multiple analyses of lunar rocks suggest that the moon is made up of the same material as Earth. Collision models peg the moon at 70-90 percent material from Theia, and most bodies in our Solar System have very different compositions.

But based on examinations of lunar rocks, it's as though the big collision never happened. It is "maybe the last major problem" with the giant impact hypothesis, says geochemist Daniel Herwartz, who also thinks he has solved it. In a paper published in Science on Thursday, he reported that the Moon does, in fact, contain a tell-tale sign of alien material.

To Herwartz - or anyone studying lunar rocks with the intention of figuring out their origin - all rocks have a sort of cosmic address label. Since ratios of isotopes (versions of an element with different numbers of neutrons) on the Earth, Mars, and asteroids are unique, we know that our young Solar System was "isotopically heterogeneous." The isotopes in a rock reveal exactly where in the Solar System it formed.

Differences in oxygen isotope levels are particularly dramatic. According to previous readings of moon-rock oxygen, the difference between a key oxygen isotope measurement - the ratio between three variations of oxygen, specifically - appeared to be just 3 parts per million higher on the Moon than on Earth, a difference so small as to be negligible. Against the evidence from collision models, the Moon's address label suggested that it was made mostly or entirely of Earth.

Herwartz thought that maybe the difference was more than statistical variance. He took samples from the Apollo 11, 12, and 16 missions (lunar samples that fell to Earth were too contaminated). Using a high-precision method published earlier this year, he released the oxygen by heating it in a container with fluorine gas, purified it, and then measured the isotope ratios in a gas mass spectrometer.

On this re-evaluation, he found that the ratio between to oxygen isotopes on the Moon was, in fact, different: 12 parts per million higher on the Moon than on Earth. This difference confirms that the Moon is not made of material that formed in the same region as Earth, and, most importantly, that it's not merely a chunk of Earth.

The isotope difference is still not terribly large - Mars and the Earth differ by a factor of 300 ppm, for example. But that suggests Theia probably formed in a region of the solar system near Earth.

As for how much of the Moon is Theia and how much is Earth - that's still a mystery. After colliding with Earth, Theia ceased to exist as an independent body. Collision models peg the ratio at 70 percent to 90 percent. Herwartz suspects that it's closer to 50/50, but that's just an informed guess at this point.

The details may be fuzzy, but as Herwartz said in a statement: "we can now be reasonably sure that the giant collision took place."

Science, 2014. DOI: 10.1126/science.1251117 (About DOIs).

http://bit.ly/1o2sU1n

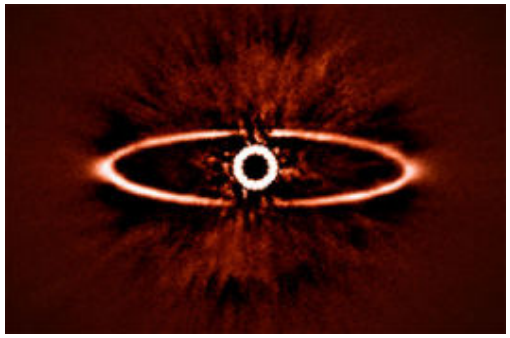
Awesome Exoplanet Imager Begins Hunt for Alien Worlds

A new instrument attached to one of the most powerful telescopes in the world has been switched on and acquired its 'first light' images of alien star systems and Saturn's moon Titan.

Jun 4, 2014 12:35 PM ET // by Ian O'Neill

The Spectro-Polarimetric High-contrast Exoplanet REsearch (or SPHIRES) instrument has been recently installed at the ESO's Very Large Telescope's already impressive suite of sophisticated instrumentation. The VLT is located in the ultra-dry high-altitude climes of the Atacama Desert in Chile.

In the observation above, an 'Eye of Sauron'-like dust ring surrounding the star HR 4796A in the southern constellation of Centaurus, a testament to the sheer power of the multiple technique SPHIRES will use to acquire precision views of directly-imaged exoplanets.



The biggest problem with trying to directly image a world orbiting close to its parent star is that of glare; stars are many magnitudes brighter that the reflected light from its orbiting exoplanet, so how the heck are you supposed to gain enough contrast between the bright star and exoplanet to resolve the two?

This infrared image shows the dust ring around the nearby star HR 4796A in the southern constellation of Centaurus. **ESO**

The SPHIRES instrument is using a combination of three sophisticated techniques to remove a star's glare and zero-in on its exoplanetary targets.

The first technique, known as adaptive optics, is employed by the VLT itself. By firing a laser into the Earth's atmosphere during the observation, a gauge on the turbulence in the upper atmospheric gases can be measured and the effects of which can be removed from the imagery. Any blurriness caused by our thick atmosphere can be adjusted for.

Next up is a precision coronograph inside the instrument that blocks the light from the target star. By doing this, any glare can be removed and any exoplanet in orbit may be bright enough to spot.

But the third technique, which really teases out any exoplanet signal, is the detection of different polarizations of light from the star system. The polarization of infrared light being generated by the star and the infrared glow from the exoplanet are very subtle. SPHIRES can differentiate between the two, thereby further boosting the observation's contrast.

"SPHERE is a very complex instrument. Thanks to the hard work of the many people who were involved in its design, construction and installation it has already exceeded our expectations. Wonderful!" said Jean-Luc Beuzit, of the Institut de Planétologie et d'Astrophysique de Grenoble, France and Principal Investigator of SPHERE, in an ESO press release.

The speed and sheer power of SPHIRES will be an obvious boon to astronomers zooming in on distant exoplanets, aiding our understanding of these strange new worlds.

http://phys.org/news/2014-06-air-roof-tiles-titanium-dioxide.html

Cleaning air with roof tiles: Titanium dioxide coating removes 97 percent of smog-causing nitrogen oxide

Students created a roof tile coating that breaks down smog-causing nitrogen oxides

A team of University of California, Riverside's Bourns College of Engineering students created a roof tile coating that when applied to an average-sized residential roof breaks down the same amount of smog-causing nitrogen oxides per year as a car driven 11,000 miles. They calculated 21 tons of nitrogen oxides would be eliminated daily if tiles on one million roofs were coated with their titanium dioxide mixture. They also calculated it would cost only about $5 for enough titanium dioxide to coat an average-sized residential roof.

That would have a significant impact in Southern California, where 500 tons of nitrogen oxides are emitted daily in the South Coast Air Quality Management District coverage area, which includes all of Orange County and the urban portions of Los Angeles, Riverside and San Bernardino counties.

Last month, the research by the UC Riverside team – Carlos Espinoza, Louis Lancaster, Chun-Yu "Jimmy" Liang, Kelly McCoy, Jessica Moncayo and Edwin Rodriguez – received an honorable mention award for phase two of an Environmental Protection Agency student design competition.

A UC Riverside student team who worked on the project last year received $15,000 as a phase one winner of EPA's P3 (People, Prosperity and the Planet) competition. That team consisted of William Lichtenberg, Duc Nguyen, Calvin Cao, Vincent Chen and Espinoza (an undergraduate then who is now a graduate student at UC Riverside). Both teams were advised by David Cocker, a professor of chemical and environmental engineering, and Kawai Tam, a lecturer at the Bourns College of Engineering.

Nitrogen oxides are formed when certain fuels are burned at high temperatures. Nitrogen oxides then react with volatile organic compounds in the presence of sunlight to create smog. Currently, there are other roofing tiles on the market that help reduce pollution from nitrogen oxides. However, there is little data about claims that they reduce smog.

The students set out to change that. They coated two identical off-the-shelf clay tiles with different amounts of titanium dioxide, a common compound found in everything from paint to food to cosmetics. The tiles were then placed inside a miniature atmospheric chamber that the students built out of wood, Teflon and PVC piping.

The chamber was connected to a source of nitrogen oxides and a device that reads concentrations of nitrogen oxides. They used ultraviolet light to simulate sunlight, which activates the titanium dioxide and allows it to break down the nitrogen oxides.

They found the titanium dioxide coated tiles removed between 88 percent and 97 percent of the nitrogen oxides. They also found there wasn't much of a difference in nitrogen oxide removal when different amounts of the coating were applied, despite one having about 12 times as much titanium dioxide coating. There wasn't much of a difference because surface area, not the amount of coating, is the important factor.

The current team of students, all of whom are set to graduate in June, are hopeful a new team of students will continue with this project and test other variables.

For example, they want to see what happens when they add their titanium dioxide to exterior paint. They are also considering looking at applying the coating to concrete, walls or dividers along freeways. Other questions include how long the coating will last when applied and what impact changing the color of coating, which is currently white, would have.

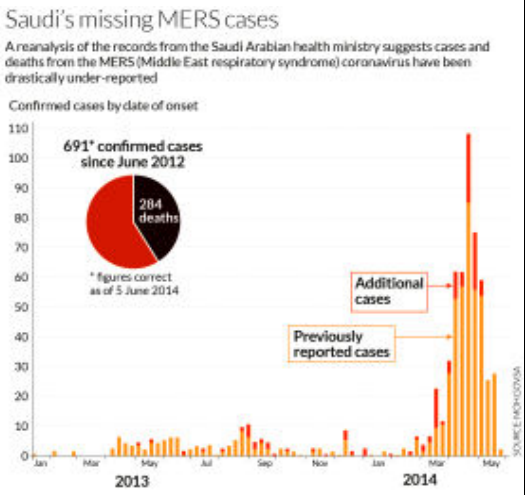
http://bit.ly/1kWVitz

More than 100 missing Saudi MERS cases come to light

Talk about keeping things quiet. The disease-tracking world was rocked today by an announcement from Saudi Arabia's health ministry.

19:30 04 June 2014 by Debora MacKenzie

Apparently the country has had 691 cases of Middle East respiratory syndrome (MERS) since January 2013, not the 575 it had reported until yesterday. Worse, 41 per cent of the total cases have resulted in death, when it was previously thought to be 33 per cent.



The admission comes the day after the Saudi health minister, himself a replacement for a minster sacked in April, dismissed Ziad Memish as the country's top medical official for MERS. Memish has been criticised for being slow to release data on the infection.

The ministry has not yet said how the new cases were found, but they could have emerged from a search of hospital records. Or over-stretched labs might have finally tested a backlog of samples. Most of those infections were picked up in hospitals, 28 per cent of them by health workers, from people already infected. The original source of infection is still unknown, although camels are suspected. Systematic comparisons of people with and without MERS are needed to track down the source of the virus, say epidemiologists, but these have not yet been done.

The Saudi ministry says it will be able to better track the infection in future, using an electronic case-reporting system, more and better testing labs, and a "robust countrywide courier system" to get specimens to them.

http://bit.ly/1kIVvWi

Sugar Substitute Turns Out to Be Potent Insecticide

Researchers have just discovered that erythritol, the main component of the popular sweetener Truvia®, kills insects.

Jun 4, 2014 05:00 PM ET // by Jennifer Viegas

The study, published in the latest PLoS ONE, suggests that the popular sugar substitute could be an effective and human-safe insecticide. No other known sweeteners currently on the market exhibit these toxic effects on insects, according to the authors.

Scientists are always on the lookout for potent bug killers that won't harm people, so it was surprising that the common sweetener does the deadly job so well.

"I feel like this is the simplest, most straightforward work I've ever done, but it's potentially the most important thing I've ever worked on," senior author Sean O'Donnell, a Drexel University professor of biology and biodiversity, was quoted as saying in a press release.

Another researcher who worked on the project was ninth grader Simon D. Kaschock-Marenda. Three years ago, he questioned why both of his parents had stopped eating white sugar when trying to eat healthier.

"He asked if he could test the effects of different sugars and sugar substitutes on fly health and longevity for his science fair, and I said, 'Sure!'" recalled Daniel Marenda, Simon's father who is also a co-author of the study.

The father and son duo went to a local supermarket and bought every type of sugar and sugar substitute that they could. They raised "baby" flies (supplied by Marenda's lab) on the various compounds to see what would happen.

"After six days of testing these flies in our house, he (Simon) came back to me and said, 'Dad, all the flies in the Truvia® vials are dead,'" Marenda said. "To which I responded, 'OK…we must have screwed up somehow. Let's repeat the experiment!'"

They did, and determined that flies raised on food containing Truvia® lived for only 5.8 days on average, compared to 38.6 to 50.6 days for flies raised on control and experimental foods without Truvia®. Flies raised on food containing Truvia® also showed noticeable motor impairments prior to their deaths.

"Indeed what we found is that the main component of Truvia®, the sugar erythritol, appears to have pretty potent insecticidal activity in our flies," Marenda said.
Erythritol is a naturally occurring sugar alcohol that is present in small amounts in many fruits. It has been tested in humans at high doses and these studies have concluded that it's safe for humans to consume. As a result, it has been designated as a generally recognized safe food additive by the U.S. Food and Drug Administration since 2001 and is also approved as a food additive in many other countries.
The scientists determined that stevia plant extract, which is also in Truvia®, had no ill effect on the flies. Only erythritol really did a number on them.
"We are not going to see the planet sprayed with erythritol and the chances for widespread crop application are slim," O'Donnell said. "But on a small scale, in places where insects will come to a bait, consume it and die, this could be huge."
The researchers next hope to find out if the sweetener kills other insect pests, such as termites, cockroaches, bed bugs and ants.

http://www.scientificamerican.com/article/are-human-pheromones-real/

Are Human Pheromones Real?

Scientists are still unraveling nature's secret olfactory signals

May 1, 2014 |By Daisy Yuhas

Strange as it may sound, some scientists suspect that the humble armpit could be sending all kinds of signals from casual flirtation to sounding the alarm. That's because the body's secretions, some stinky and others below the threshold your nose can detect, may be rife with chemical messages called pheromones. Yet despite half a century of research into these subtle cues, we have yet to find direct evidence of their existence in humans.

What Are Pheromones?

Humans and other animals have an olfactory system designed to detect and discriminate between thousands of chemical compounds. For more than 50 years, scientists have been aware of the fact that certain insects and animals can release chemical compounds - often as oils or sweat - and that other creatures can detect and respond to these compounds, which allows for a form of silent, purely chemical communication.
Although the exact definition has been debated and redefined several times, pheromones are generally recognized as single or small sets of compounds that transmit signals between organisms of the same species. They are typically just one part of the larger potpourri of odorants emitted from an insect or animal, and some pheromones do not have a discernable scent.
Since pheromones were first defined in 1959, scientists have found many examples of pheromonal communication. The most striking of these signals elicits an immediate behavioral response. For example, the female silk moth releases a trail of the molecule bombykol, which unerringly draws males from the moment they encounter it. Slower-acting pheromones can affect the recipient's reproductive physiology, as when the alpha-farnesene molecule in male mouse urine accelerates puberty in young female mice.
Some researchers have proposed a third group of pheromones called "signalers" that simply transmit information such as an individual's social status or health. Mice can select appropriate mates based on odor cues, deriving information in part from unique proteins associated with a mouse's genetics.

The Trouble with Humans

So far, scientists have had some success in demonstrating that exposure to body odor can elicit responses in other humans. As in rodent research, human sweat and secretions can affect the reproductive readiness of other humans. Since the 1970s researchers have observed changes in a woman's menstrual cycle when she is exposed to the sweat of other women. In 2011 a Florida State University group demonstrated that the scent of ovulating women could cause testosterone levels to increase in men.
But there is no evidence of a consistent and strong behavioral response to any human-produced chemical cue. "Maybe once upon a time we could react more viscerally," says chemist George Preti of the Monell Chemical Senses Center. Today, however, our reactions seem to be much subtler - and harder to detect - than those of a silk moth. This subtlety has led researchers to propose another kind of chemical messenger, known as a "modulator" pheromone, that affects the mood or mental state of the recipient. In an example of this type, researchers at Stony Brook University found in 2009 that sniffing the sweat of first-time parachute jumpers could increase a person's ability to discriminate between ambiguous emotional expressions. The implication is that chemicals in the jumper's sweat might constitute an alarm signal, which puts the recipient on high alert and makes them more attentive to details.
Yet to demonstrate definitively that pheromones are at work, researchers need to point to the molecules responsible, which they have not yet done. To date, scientists have collected evidence for possible pheromone effects but have not definitively identified a single human pheromone.

A Signature Scent

As the hunt for human pheromones continues, scientists have also investigated other potential explanations for the subtle effects of smells. Consider, for example, the finding that human infants will crawl toward the odor of their mother's breast. Baby rabbits are known to begin nursing when exposed to a specific pheromone from a lactating mother rabbit. Yet the human infants might simply be attracted to a

mother's so-called odor print, or unique personal scent. Odor prints are influenced by diet, environment, health and genetics. They consist of far too many compounds to be described as pheromones themselves.

The failure to identify human pheromones has not stopped some enterprising individuals from trying to make a profit from love potions purporting to contain pheromones. In reality, these products often use pig pheromones. "They don't have any history in the biomedical literature - they just fell out of the sky," says olfactory neuroscientist Charles Wysocki, also of Monell. For now, the idea of perfumes and potions based on human pheromonal communication just doesn't pass the sniff test.

http://www.eurekalert.org/pub_releases/2014-06/gi-rat060514.php

Researchers at the Gladstone Institutes find novel approach to reactivate latent HIV

New study published in Science

SAN FRANCISCO - A team of scientists at the Gladstone Institutes has identified a new way to make latent HIV reveal itself, which could help overcome one of the biggest obstacles to finding a cure for HIV infection. They discovered that increasing the random activity, or noise, associated with HIV gene expression–without increasing the average level of gene expression–can reactivate latent HIV. Their findings were published today in the journal Science.

When HIV infects an immune cell, it inserts its genetic material into the DNA of the infected cell. In most cases, the immune cell's machinery makes copies of the viral genetic material, a process known as transcription. This eventually leads to the production–or expression–of all the components needed to make more viruses. The new viruses are released from the infected cell and spread the infection to other immune cells in the body.

In some cases, however, HIV expression goes into a holding pattern and the virus enters a latent state within the infected immune cell. This means that a small percentage of HIV hides in infected cells, beyond the reach of even the most potent drugs. As a result, we cannot completely eliminate HIV from the body, and people with HIV infection have to take antiretroviral drugs (ARVs) for the rest of their lives.

"Understanding how to reactivate latent HIV is one of the major challenges we must overcome in order to find a cure for HIV," said Leor Weinberger, PhD, Associate Investigator in the Gladstone Institute of Virology and Immunology and senior author of the study. Roy Dar, PhD, the lead author of the study, added, "If we can make the virus show itself, we can then use ARVs to eliminate it. This so-called 'shock and kill' approach holds great promise, but to date it has unfortunately shown only limited success."

One of the properties of latency that makes it so difficult to address is that it is random–or stochastic–in nature. Random fluctuations in transcription are unavoidable and a general aspect of life at the single-cell level and lead to "noise" around the average level of gene expression. HIV happens to have exceptionally noisy gene expression. Scientists have previously identified compounds that can reactivate HIV by activating transcription, but these compounds are not very effective, in part because of the noisiness of HIV transcription.

In this study, the team tested the counter-intuitive notion that compounds that increase noise in gene expression could work together with transcriptional activators to increase overall levels of HIV reactivation. The concept borrows from other fields of science such as chemistry, where theoretical arguments long ago argued that increased fluctuations can increase the efficiency of reactions.

First, they screened a library of 1,600 compounds using a specialized cell line that produces a green fluorescent protein (GFP) when gene expression is activated. The team identified 85 small molecules that increased noise without changing average GFP gene expression levels. They then combined these newly identified noise enhancers with known transcription activators in a cell line that serves as a model for HIV latency.

They found that while the noise enhancers could not cause reactivation on their own, 75 percent of them could synergize with activators and increase viral reactivation relative to activator alone. In fact, some noise enhancers doubled reactivation levels when combined with activators. Furthermore, they found a direct correlation between noise enhancement and the degree of reactivation synergy; the greater the noise, the greater the effect on reactivation. For the first time, these results show that expression noise and reactivation of latent HIV are directly related, and identify new candidates for the "shock and kill" approach to treating latent HIV infection.

Strategies to reverse HIV latency will likely require multiple rounds of treatment, and these new results suggest that noise-enhancing compounds may allow each round of treatment to be more effective at getting HIV to reveal itself. Additional screens for noise-enhancing activity may identify compounds that synergize with activators even better and are more efficient at reactivating the virus in order to eliminate it for good.

"The implications for using noise also extend far beyond HIV reactivation, since random cellular activity contributes to a wide range of processes, from antibiotic persistence to cancer metastasis", said Dr. Weinberger. "Thus, this approach could represent a new tool for drug discovery across multiple fields."

http://www.eurekalert.org/pub_releases/2014-06/ucl-oat060514.php

Our ability to identify the source of pain varies across the body

A new UCL study defines for the first time how our ability to identify where it hurts, called 'spatial acuity,' varies across the body, being most sensitive at the forehead and fingertips

"Where does it hurt?" is the first question asked to any person in pain. A new UCL study defines for the first time how our ability to identify where it hurts, called "spatial acuity", varies across the body, being most sensitive at the forehead and fingertips.

Using lasers to cause pain to 26 healthy volunteers without any touch, the researchers produced the first systematic map of how acuity for pain is distributed across the body. The work is published in the journal Annals of Neurology and was funded by the Wellcome Trust.

With the exception of the hairless skin on the hands, spatial acuity improves towards the centre of the body whereas the acuity for touch is best at the extremities. This spatial pattern was highly consistent across all participants.

The experiment was also conducted on a rare patient lacking a sense of touch, but who normally feels pain. The results for this patient were consistent with those for healthy volunteers, proving that acuity for pain does not require a functioning sense of touch.

"Acuity for touch has been known for more than a century, and tested daily in neurology to assess the state of sensory nerves on the body. It is striking that until now nobody had done the same for pain," says lead author Dr Flavia Mancini of the UCL Institute of Cognitive Neuroscience. "If you try to test pain with a physical object like a needle, you are also stimulating touch. This clouds the results, like taking an eye test wearing sunglasses. Using a specially-calibrated laser, we stimulate only the pain nerves in the upper layer of skin and not the deeper cells that sense touch."

Volunteers were blindfolded and had specially-calibrated pairs of lasers targeted at various parts of their body. These lasers cause a brief sensation of pinprick pain. Sometimes only one laser would be activated, and sometimes both would be, unknown to participants. They were asked whether they felt one 'sting' or two, at varying distances between the two beams. The researchers recorded the minimum distance between the beams at which people were able to accurately say whether it was one sting or two.

"This measure tells us how precisely people can locate the source of pain on different parts of their body," explains senior author Dr Giandomenico Iannetti of the UCL Department of Neuroscience, Physiology and Pharmacology. "Touch and pain are mediated by different sensory systems. While tactile acuity has been well studied, pain acuity has been largely ignored, beyond the common textbook assertion that pain has lower acuity than touch. We found the opposite: acuity for touch and pain are actually very similar. The main difference is in their gradients across the body. For example, pain acuity across the arm is much higher at the shoulder than at the wrist, whereas the opposite is true for touch."

Acuity for both touch and pain normally correlates with the density of the relevant nerve fibres in each part of the body. However, the fingertips remain highly sensitive despite having a low density of pain-sensing nerve cells.

"The high pain acuity of the fingertips is something of a mystery that requires further investigation," says Dr Mancini. "This may be because people regularly use their fingertips, and so the central nervous system may learn to process the information accurately."

The findings have important implications for the assessment of both acute and chronic pain. Dr Roman Cregg of the UCL Centre for Anaesthesia, who was not involved in the research, is a clinical expert who treats patients with chronic pain. "Chronic pain affects around 10 million people in the UK each year according to the British Pain Society, but we still have no reliable, reproducible way to test patients' pain acuity," says Dr Cregg. "This method offers an exciting, non-invasive way to test the state of pain networks across the body. Chronic pain is often caused by damaged nerves, but this is incredibly difficult to monitor and to treat. The laser method may enable us to monitor nerve damage across the body, offering a quantitative way to see if a condition is getting better or worse. I am excited at the prospect of taking this into the clinic, and now hope to work with Drs Mancini and Iannetti to translate their study to the chronic pain setting."

http://www.bbc.com/news/health-27730752

Does female fertility 'drop off a cliff'?

TV presenter Kirstie Allsopp has urged women to put off higher education and a career in favour of having children because their "fertility falls off a cliff".

By Philippa Roxby Health reporter, BBC News

In a recent interview with Jeremy Paxman on BBC Newsnight, Ms Allsopp, who met her husband when she was 32 and then had two children, said: "Nature is not with you and I. Nature is not a feminist." She also encouraged women to be "more honest" with one another about their biological clock, saying the topic was still "taboo". So what is the truth about the female fertility window?

Guidance from the National Institute for Health and Care Excellence (NICE), which was updated in 2013, is pretty encouraging.

It says that "over 80% of couples in the general population will conceive within one year if the woman is aged under 40 years", if they have regular sexual intercourse and do not use contraception.

NICE guidelines go on to state that "of those who do not conceive in the first year, about half will do so in the second year". That leaves around 10% of women - the percentage said to be affected by infertility in the UK. We know, of course, that female fertility declines with age, but is there really a dramatic drop-off at a certain point?

No rule

Mr Yacoub Khalaf, head of assisted reproduction at Guy's and St Thomas' Hospital in London, says it is not quite as simple as that. "It is tempting to want a black and white answer, but biology doesn't work that way. "Some women find it difficult to conceive in their late 20s, while others don't have a problem into their 40s."

So there is no rule, but science tells us that a woman's body does gradually change and there is nothing that can be done to alter that process.

Mr Khalaf explains that from the age of 35, the rate of depletion of the follicles in the ovaries speeds up, and from the age of 40 they start to deplete even faster.

These follicles are important because they house the eggs which will develop and mature before finally being released during ovulation. Hence, the quality and quantity of a women's eggs also begins declining sharply from around the age of 35 onwards.

Disappearing eggs

It is a very different story at birth. Baby girls are born with a finite number of eggs, which can number around one million. By the time of their first period, however, only 400,000 eggs will be left and they continue to decline in number throughout adulthood at a rate of approximately 1,000 eggs each month.

"They are much more accessible in the early years," Mr Khalaf says, referring to women's eggs in their mid-20s. "I would rather have women trying for a baby as soon as they can because they will have healthy eggs, a healthy pregnancy and the energy to enjoy their baby."

But he recognises that the realities of life mean this is not often possible. Higher education, career, finding Mr Right - all mean that women may not start thinking about having children until well into their 30s. By that time, it is possible they may run into problems.

Seek help

Infertility Network UK advises women and their partners not to be complacent about fertility problems and to seek help from their GP. This is because there may be gynaecological disorders which women are unaware of which could come to light. These include polyps or fibroids, endometriosis or pelvic adhesions, which can be treated to maximise fertility. As women reach their 40s, their risk of miscarrying increases to nearly a third of all pregnancies. The chance of giving birth to a baby with Down's Syndrome also rises significantly.

Alison McTavish, nurse manager at the University of Aberdeen's assisted reproduction unit, says when women are already on the "slippery slope" between 30 and 34 years old they mistakenly look to IVF as being a solution. "This sometimes gives them false hope - we're not that good though. "We tend to always talk about IVF success rates, but we don't say it's unsuccessful for most women."

After 40, there is a 5% chance of a woman becoming pregnant without IVF, increasing to 10% with the help of IVF, she says.

Fertility experts agree that the female fertility window has not changed much over the decades. The menopause still occurs in the same age range as it did for our mothers and grandmothers. What has changed though is the male sperm count, which has been decreasing over the years. The reasons for this are not known, although there are theories, so perhaps women should remember to keep check on their partner's fertility as well as their own.

http://www.eurekalert.org/pub_releases/2014-06/uoc--ttu060614.php

Targeting tumors using silver nanoparticles

A new platform developed at UCSB increases the efficiency of drug delivery and allows excess particles to be washed away

Santa Barbara, Calif. - Scientists at UC Santa Barbara have designed a nanoparticle that has a couple of unique - and important - properties. Spherical in shape and silver in composition, it is encased in a shell coated with a peptide that enables it to target tumor cells. What's more, the shell is etchable so those nanoparticles that don't hit their target can be broken down and eliminated. The research findings appear today in the journal Nature Materials.

The core of the nanoparticle employs a phenomenon called plasmonics. In plasmonics, nanostructured metals such as gold and silver resonate in light and concentrate the electromagnetic field near the surface. In this way, fluorescent dyes are enhanced, appearing about tenfold brighter than their natural state when no metal is present. When the core is etched, the enhancement goes away and the particle becomes dim.

UCSB's Ruoslahti Research Laboratory also developed a simple etching technique using biocompatible chemicals to rapidly disassemble and remove the silver nanoparticles outside living cells. This method leaves only the intact nanoparticles for imaging or quantification, thus revealing which cells have been targeted and how much each cell internalized.

"The disassembly is an interesting concept for creating drugs that respond to a certain stimulus," said Gary Braun, a postdoctoral associate in the Ruoslahti Lab in the Department of Molecular, Cellular and Developmental Biology (MCDB) and at Sanford-Burnham Medical Research Institute. "It also minimizes the off-target

toxicity by breaking down the excess nanoparticles so they can then be cleared through the kidneys."

This method for removing nanoparticles unable to penetrate target cells is unique. "By focusing on the nanoparticles that actually got into cells," Braun said, "we can then understand which cells were targeted and study the tissue transport pathways in more detail."

Some drugs are able to pass through the cell membrane on their own, but many drugs, especially RNA and DNA genetic drugs, are charged molecules that are blocked by the membrane. These drugs must be taken in through endocytosis, the process by which cells absorb molecules by engulfing them.

"This typically requires a nanoparticle carrier to protect the drug and carry it into the cell," Braun said. "And that's what we measured: the internalization of a carrier via endocytosis."

Because the nanoparticle has a core shell structure, the researchers can vary its exterior coating and compare the efficiency of tumor targeting and internalization. Switching out the surface agent enables the targeting of different diseases - or organisms in the case of bacteria - through the use of different target receptors. According to Braun, this should turn into a way to optimize drug delivery where the core is a drug-containing vehicle.

"These new nanoparticles have some remarkable properties that have already proven useful as a tool in our work that relates to targeted drug delivery into tumors," said Erkki Ruoslahti, adjunct distinguished professor in UCSB's Center for Nanomedicine and MCDB department and at Sanford-Burnham Medical Research Institute. "They also have potential applications in combating infections. Dangerous infections caused by bacteria that are resistant to all antibiotics are getting more common, and new approaches to deal with this problem are desperately needed. Silver is a locally used antibacterial agent and our targeting technology may make it possible to use silver nanoparticles in treating infections anywhere in the body."

http://www.eurekalert.org/pub_releases/2014-06/uoma-srr060614.php

Study reveals rats show regret, a cognitive behavior once thought to be uniquely human

Research findings recently published in Nature Neuroscience

New research from the Department of Neuroscience at the University of Minnesota reveals that rats show regret, a cognitive behavior once thought to be uniquely and fundamentally human. Research findings were recently published in Nature Neuroscience.

To measure the cognitive behavior of regret, A. David Redish, Ph.D., a professor of neuroscience in the University of Minnesota Department of Neuroscience, and Adam Steiner, a graduate student in the Graduate Program in Neuroscience, who led the study, started from the definitions of regret that economists and psychologists have identified in the past.

"Regret is the recognition that you made a mistake, that if you had done something else, you would have been better off," said Redish. "The difficult part of this study was separating regret from disappointment, which is when things aren't as good as you would have hoped. The key to distinguishing between the two was letting the rats choose what to do."

Redish and Steiner developed a new task that asked rats how long they were willing to wait for certain foods. "It's like waiting in line at a restaurant," said Redish. "If the line is too long at the Chinese food restaurant, then you give up and go to the Indian food restaurant across the street."

In this task, which they named "Restaurant Row," the rat is presented with a series of food options but has limited time at each "restaurant."

Research findings show rats were willing to wait longer for certain flavors, implying they had individual preferences. Because they could measure the rats' individual preferences, Steiner and Redish could measure good deals and bad deals. Sometimes, the rats skipped a good deal and found themselves facing a bad deal.

"In humans, a part of the brain called the orbitofrontal cortex is active during regret. We found in rats that recognized they had made a mistake, indicators in the orbitofrontal cortex represented the missed opportunity. Interestingly, the rat's orbitofrontal cortex represented what the rat should have done, not the missed reward. This makes sense because you don't regret the thing you didn't get, you regret the thing you didn't do," said Redish.

Redish adds that results from Restaurant Row allow neuroscientists to ask additional questions to better understand why humans do things the way they do. By building upon this animal model of regret, Redish believes future research could help us understand how regret affects the decisions we make.

http://www.bbc.com/news/science-environment-27720617

Male faces 'buttressed against punches' by evolution

A new theory suggests that our male ancestors evolved beefy facial features as a defence against fist fights.

By Jonathan Webb Science reporter, BBC News

The bones most commonly broken in human punch-ups also gained the most strength in early "hominin" evolution. They are also the bones that show most divergence between males and females. The paper, in the journal Biological

Reviews, argues that the reinforcements evolved amid fighting over females and resources, suggesting that violence drove key evolutionary changes.

Fossil records show that the australopiths, immediate predecessors of the human genus Homo, had strikingly robust facial structures.

For many years, this extra strength was seen as an adaptation to a tough diet including nuts, seeds and grasses. But more recent findings, examining the wear pattern and carbon isotopes in australopith teeth, have cast some doubt on this "feeding hypothesis".

"In fact, [the australopith] boisei, the 'nutcracker man', was probably eating fruit," said Prof David Carrier, the new theory's lead author and an evolutionary biologist at the University of Utah.

Protective armour

Instead of diet, Prof Carrier and his co-author, physician Dr Michael Morgan, propose that violent competition demanded the development of these facial fortifications: what they call the "protective buttressing hypothesis".

In support of their proposal, Carrier and Morgan offer data from modern humans fighting. Several studies from hospital emergency wards, including one from the Bristol Royal Infirmary, show that faces are particularly vulnerable to violent injuries.

"Jaws are one of the most frequent bones to break - and it's not the end of the world now, because we have surgeons, we have modern medicine," Prof Carrier explained. "But four million years ago, if you broke your jaw, it was probably a fatal injury. You wouldn't be able to chew food... You'd just starve to death."

The jaw, cheek, eye and nose structures that most commonly come to grief in modern fist fights were also the most protected by evolutionary changes seen in the australopiths.

Furthermore, these are the bones that show the most differences between men and women, as well as between our male and female forebears. That is how you would expect defensive armour to evolve, Prof Carrier points out.

"In humans and in great apes in general... it's males that are most likely to get into fights, and it's also males that are most likely to get injured," he told BBC News.

Long-running debate

Interestingly, the evolutionary descendents of the australopiths - including humans - have displayed less and less facial buttressing.

This is consistent, according to Prof Carrier, with a decreasing need for protection: "Our arms and upper body are not nearly as strong as they were in the australopiths," he explained. "There's a temporal correlation."

The facial buttressing idea builds on a previous observation by Prof Carrier and Dr Morgan that the early hominins were the first primates to evolve a hand shape compatible with making a fist - and thus, throwing a punch.

That earlier paper attracted criticism from some other researchers, and Prof Carrier expects this new contribution may also prove controversial. He says that debate about the role of violence in human evolution is not new.

"[Our paper] does address this debate of whether our past was violent or peaceful," he told the BBC. "That's an argument that's been going on for a very long time."

"The historical record goes back a short time, the archaeological record goes back a few tens of thousands years more... But the anatomy holds clues to what selection was important, what behaviours were important, and so it gives us information about the very distant past."