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Fungal Meningitis Pathogen Discovers New Appetite for Human Brains

The primary culprit in the recent flare-up caused by tainted steroids, Exserohilum rostratum, is not an especially picky eater. Although the fungus prefers grasses, it will dine on many items - including humans

By Jennifer Frazer | Monday, November 12, 2012 | 9

The nation's ongoing fungal meningitis outbreak has killed 30 and sickened 419 people so far, but the fungus responsible has never wrought such havoc before. The fungus, Exserohilum rostratum, is a plant-eating generalist equipped with a spore-launching mechanism ideal for going airborne, is not an especially picky eater and, although it prefers grasses, will dine on many items - including humans. But just how a pathogen typically associated with the great outdoors got into the three lots of injectable steroids prepared inside an admittedly filthy laboratory - and why only three lots - remains a puzzling mystery.

Exserohilum rostratum **Image: Glenn Roberts**

The errant fungus has been identified in lab samples from 52 of those affected and was similarly found growing in unopened vials of the steroid alleged to have caused the outbreak, according to the U.S. Centers for Disease Control and Prevention. A third recalled lot is still being tested. But E. rostratum is not a household name, even among mycologists.

Glenn Roberts, a retired medical mycologist, says that in his 40 years of experience at the Mayo Clinic in Rochester, Minn., he had seen only one case: a soft-tissue arm wound in an immunocompromised patient. He was shocked when he heard the identity of the pathogen in the epidemic that originated with the New England Compounding Center pharmacy in Framingham, Mass. “I could hardly believe it because it’s just so uncommon,” he says. And yet, outside in the air and on plants, E. rostratum is not so uncommon.

In press reports, it has been described as occurring "on grasses," but that is not the full story. The fungus, which seems to prefer tropical and subtropical environments, has turned up on a wide variety of plant species, says Kurt Leonard, an emeritus professor in the Department of Plant Pathology at the University of Minnesota who retired in 2001 from the U.S. Department of Agriculture's Cereal Disease Lab (then the Cereal Rust Lab).

Early in his career, Leonard untangled the taxonomic mess of similar-looking, but only distantly related, fungi with multicellular dark spores that were causing disease in grains such as corn. He named one new genus he had created - Exserohilum - for the prominent protuberances called hila (the belly buttons of the fungal and botanical world) on its spores.

The modus operandi of one species in this genus - E. rostratum - was to infect a plant and in some cases precipitate tissue death. Plant defenses - which can include induced cell fortification, cell suicide, toxic chemicals, and defensive enzymes and proteins - typically were sufficient to keep the infection in check, but not strong enough to eliminate it. The payoff came when the plant died - the fungus was first in line to feed on its decaying remains. "I think it's just a general weak pathogen of plants," Leonard says, "something that can infect plants while alive and not really do much damage until the leaf senesces."

Leonard found E. rostratum on corn, sorghum and Johnsongrass fairly often, although it was not nearly as common as several more severe corn pathogens. It was an opportunist and would sometimes infect ears and stalks when insects drilled into the plant, creating a convenient landing pad of dying tissue for the fungus.

Most often the fungus shows up on grasses and other monocots - plants often distinguished by flower parts in threes and parallel leaf venation - such as pineapples, bananas and sugarcane, but it has also been found on non-monocots such as grapes and muskmelon. It's a fungus that is not, apparently, very picky about its food. "It's just a really common fungus in the environment that mostly lives on dead and dying plant tissue," Leonard says. There are many such others, and many of them can also occasionally infect animals or people.

Leonard has observed one other intriguing characteristic of E. rostratum in his lab: The fungus can grow from a single spore to a lawn of freshly spore-crowned fungal filaments on a piece of dried leaf in two days flat - faster and more abundantly than any other related species he studied. "This is a fungus very well-adapted to colonizing senescent or dead leaf tissue once conditions are right," Leonard says. "So that would be another reason E. rostratum would be a likely candidate for showing up in a messy lab."

But if the fungus is primarily tropical and subtropical, what was it doing in a place like New England?

In the summer the fungus can probably find ideal growing conditions in places in the northern U.S., Leonard explains, or it may be spread northward by winds. The spores have a static electricity–based ejection system designed to launch them into the air with ease. And plentiful lawn clippings provide an ideal place for the fungus to grow.

Roberts says the group of fungi pigmented with melanin (which includes E. rostratum) - the same molecule that darkens and protects human skin - seem to be generating more human infections for reasons he does not understand. E. rostratum, in addition to causing soft-tissue infections, has also rarely provoked sinus or eye infections, primarily in immunocompromised patients.

Although the identity of the fungus surprised him, Roberts was not surprised by its ability to capitalize on its situation once inside a patient. After the fungus was injected along with the drug into the epidural space - the space between the dura mater, which encloses the spinal fluid and spinal cord, and the inside walls of the vertebrae - the fungus's filaments were able to penetrate the dura mater, enter the spinal fluid and travel straight to the brain, an environment where the immune system has a very difficult time eliminating or even just controlling infection. "Spinal fluid is a great culture medium - one of the best," he says. "The nutrients are there, and the temperature is certainly right."

Those who suffered the worst infections, he speculates, were probably those in whom the needle accidentally penetrated the dura mater, thereby shortening the fungus's deadly path into the spinal fluid. Then, in some fatal cases, the fungal filaments began to grow in the brain, attracting platelets and white and red blood cells to aggregate around the filaments and form a mass that could block a blood vessel and initiate a stroke. Strokes were not implicated in all the fatalities, however, so the mechanism(s) in those other deaths remains unclear.

The fungus's confinement to just three lots of the drug also remains unexplained. If the facility's water or air supplies in general were contaminated, one would expect all lots to be affected. Perhaps something blew in from a nearby recycling center or some other source on one or a few days and not on others, Roberts speculated. Another pathway could be the drug itself: Although the water used for making up the final doses was allegedly sterile, the steroid drug ingredient was not. "Using nonsterile components [for injection] in somebody's spine?" Roberts says. "My goodness, that's terrible."

http://phys.org/news/2012-11-anthropologist-large-differences-gait-early.html

Anthropologist finds large differences in gait of early human ancestors

The walking gait between two of our early ancestors was likely so different that it's doubtful they would have done so together

(Phys.org) - Patricia Ann Kramer, professor of anthropology at the University of Washington, has found that the walking gait between two of our early ancestors was likely so different that it's doubtful they would have done so together, despite being two members of the same species living during roughly the same time period. In her paper published in the American Journal of Physical Anthropology, Kramer outlines how she compared the natural walking speeds of modern humans to those of two members of the Australopithecus afarensis species and found that such large differences existed between two members of our early ancestors that walking together would have been troublesome.

A sculptor's rendering of the hominid Australopithecus afarensis is displayed as part of an exhibition that includes the 3.2 million year old fossilized remains of "Lucy", the most complete example of the species, at the **Houston Museum of Natural Science**

In her study, Kramer compared the bones of Lucy, the famous skeletal remains found in Ethiopia, with those of Kadanuumuu (Big Man in Afar) another member of the A. afarensis species unearthed in 2010, though clearly much larger. Because of their difference in height – Lucy would have been about 3.5 feet tall, Big Man approximately 5 – Kramer wondered if they would have been able to walk around together.

To find out, she enlisted the aid of 36 children and 16 adults who all agreed to have their leg bones measured and then to be tested walking on a treadmill. Scientists know that people have a natural walking gait that is also the optimal speed for conserving energy. For long legged people, a faster gait is optimal, whereas for those with shorter legs, slower is better. In the case of Lucy and Big Man, the difference in the length of leg bones would have been equivalent to the difference in leg bone length between modern children and adults. She used the data from her volunteers' efforts to create a mathematical formula that allowed her to estimate the natural gait of Lucy and Big Man and found them to be 3.4 feet per second, versus 4.4 feet per second. Such a difference would have meant Lucy would have had to walk a lot faster than normal to keep up with Big Man, or Big Man would have had to walk a lot slower for the two of them to walk around together; an idea that seems counterintuitive because it would mean one or the other would have had to walk at a pace that consumed more energy.

Kramer notes that her study includes just two specimens of A. afarensis which are of the opposite gender, and who would have lived some distance from one another. Thus, she suggests it's possible that regional differences

were at play, or that males of the time were simply much larger than females, which likely would have meant they spent most of their time apart, similar to modern chimpanzees.

Could Kadanuumuu (KSD-VP-1/1) and Lucy (AL 288-1) have walked together comfortably? American Journal of Physical Anthropology, DOI: 10.1002/ajpa.22169

Abstract

The estimated lower limb length (0.761–0.793 m) of the partial skeleton of Australopithecus afarensis from Woranso-Mille (KSD-VP-1/1) is outside the previously known range for Australopithecus and within the range of modern humans. The lower limb length of KSD-VP-1/1 is particularly intriguing when juxtaposed against the lower limb length estimate of the other partial skeleton of A. afarensis, AL 288-1 (0.525 m). A sample of 36 children (age, >7 years, trochanteric height = 0.56–0.765 m) and 16 adults (trochanteric height = 0.77–1.00 m) walked at their self-selected slow, preferred, and fast walking velocities, while their oxygen consumption was monitored. Lower limb length and velocity were correlated with slow ($P < 0.001$, $r2 = 0.44$), preferred ($P < 0.001$, $r2 = 0.55$), and fast ($P < 0.001$, $r2 = 0.69$) walking velocity. The relationship between optimal velocity and lower limb length was also determined and lower limb length explained 47% of the variability in optimal velocity. The velocity profile for KSD-VP-1/1 (slow = 0.73–0.75 m/s, preferred = 1.08–1.11 m/s, and fast = 1.48–1.54 m/s) is 36–44% higher than that of AL 288-1 (slow = 0.53 m/s, preferred = 0.78 m/s, and fast = 1.07 m/s). The optimal velocity for AL 288-1 is 1.04 m/s, whereas that for KSD-VP-1/1 is 1.29–1.33 m/s. This degree of lower limb length dimorphism suggests that members of a group would have had to compromise their preferences to walk together or to split into subgroups to walk at their optimal velocity.

http://bit.ly/UNzdab

Early Human Ancestors Ate Grass

Early human ancestors in central Africa 3.5 million years ago ate a diet of mostly tropical grasses and sedges, finds new research.

Analysis by Jennifer Viegas

The study suggests our relatives were mostly plant-eaters before they evolved a taste for meaty flesh. Consider that tidbit while passing around the creamed spinach during Thanksgiving dinner. The study focused on Australopithecus bahrelghazali, which had quite a set of teeth. You can see a reconstruction of this human relative here.

"We found evidence suggesting that early hominins, in central Africa at least, ate a diet mainly comprised of tropical grasses and sedges," co-author Julia Lee-Thorp, a University of Oxford archaeologist, said in a press release. She continued, "No African great apes, including chimpanzees, eat this type of food despite the fact it grows in abundance in tropical and subtropical regions. The only notable exception is the savannah baboon which still forages for these types of plants today. We were surprised to discover that early hominins appear to have consumed more than even the baboons."

She and her colleagues made the determination after studying the fossilised teeth of three A. bahrelghazali individuals -- the first early human relatives excavated at two sites in Chad. The researchers analyzed the carbon isotope ratios in the teeth and found the signature of a diet rich in foods derived from C4 plants.

This indicates our long-gone relatives experienced a shift in their diet relatively early, at least in central Africa. These individuals survived in open landscapes with few trees, so apparently they could exploit not only dense woodland areas but also other environments. Although the area where A. bahrelghazali roamed is now dry and hyper-arid, back in the day it featured a network of shallow lakes with nearby floodplains and wooded grasslands.

While this ancestor of ours clearly had big, impressive teeth, they would not have been able to tackle leaves day after day. The individuals also lacked cow-like guts to break down and digest such food, so the researchers suspect the early hominids probably relied more on the roots, corms and bulbs at the base of the plants.

Given the carbon isotope data, there is a very remote chance that the early hominids ate animals that, in turn, ate the tropical grasses. "But as neither humans nor other primates have diets rich in animal food, and of course the hominins are not equipped as carnivores are with sharp teeth, we can assume that they ate the tropical grasses and the sedges directly," Lee-Thorp said.

The research was published in Proceedings of the National Academy of Sciences.

http://www.sciencedaily.com/releases/2012/11/121112135516.htm

Humans Are Slowly but Surely Losing Intellectual and Emotional Abilities, Article Suggests

Hypothesis suggests that we are losing our intellectual and emotional capabilities because the web of genes endowing us with our brain power is susceptible to mutations which are not being selected against in society

ScienceDaily - Human intelligence and behavior require optimal functioning of a large number of genes, which requires enormous evolutionary pressures to maintain. A provocative hypothesis published in a recent set of Science and Society pieces published in the Cell Press journal Trends in Genetics suggests that we are losing

our intellectual and emotional capabilities because the intricate web of genes endowing us with our brain power is particularly susceptible to mutations and that these mutations are not being selected against in our modern society.
"The development of our intellectual abilities and the optimization of thousands of intelligence genes probably occurred in relatively non-verbal, dispersed groups of peoples before our ancestors emerged from Africa," says the papers' author, Dr. Gerald Crabtree, of Stanford University. In this environment, intelligence was critical for survival, and there was likely to be immense selective pressure acting on the genes required for intellectual development, leading to a peak in human intelligence.
From that point, it's likely that we began to slowly lose ground. With the development of agriculture, came urbanization, which may have weakened the power of selection to weed out mutations leading to intellectual disabilities. Based on calculations of the frequency with which deleterious mutations appear in the human genome and the assumption that 2000 to 5000 genes are required for intellectual ability, Dr. Crabtree estimates that within 3000 years (about 120 generations) we have all sustained two or more mutations harmful to our intellectual or emotional stability. Moreover, recent findings from neuroscience suggest that genes involved in brain function are uniquely susceptible to mutations. Dr. Crabtree argues that the combination of less selective pressure and the large number of easily affected genes is eroding our intellectual and emotional capabilities.
But not to worry. The loss is quite slow, and judging by society's rapid pace of discovery and advancement, future technologies are bound to reveal solutions to the problem. "I think we will know each of the millions of human mutations that can compromise our intellectual function and how each of these mutations interact with each other and other processes as well as environmental influences," says Dr. Crabtree. "At that time, we may be able to magically correct any mutation that has occurred in all cells of any organism at any developmental stage. Thus, the brutish process of natural selection will be unnecessary."

http://www.eurekalert.org/pub_releases/2012-11/msu-afm111312.php

Ancient foot massage technique may ease cancer symptoms

Study offers the strongest evidence yet that reflexology can help cancer patients manage their symptoms and perform daily tasks

EAST LANSING, Mich. - A study led by a Michigan State University researcher offers the strongest evidence yet that reflexology – a type of specialized foot massage practiced since the age of pharaohs – can help cancer patients manage their symptoms and perform daily tasks.
Funded by the National Cancer Institute and published in the latest issue of Oncology Nursing Forum, it is the first large-scale, randomized study of reflexology as a complement to standard cancer treatment, according to lead author Gwen Wyatt, a professor in the College of Nursing. "It's always been assumed that it's a nice comfort measure, but to this point we really have not, in a rigorous way, documented the benefits," Wyatt said. "This is the first step toward moving a complementary therapy from fringe care to mainstream care."
Reflexology is based on the idea that stimulating specific points on the feet can improve the functioning of corresponding organs, glands and other parts of the body.
The study involved 385 women undergoing chemotherapy or hormonal therapy for advanced-stage breast cancer that had spread beyond the breast. The women were assigned randomly to three groups: Some received treatment by a certified reflexologist, others got a foot massage meant to act like a placebo, and the rest had only standard medical treatment and no foot manipulation. Wyatt and colleagues surveyed participants about their symptoms at intake and then checked in with them after five weeks and 11 weeks.
They found that those in the reflexology group experienced significantly less shortness of breath, a common symptom in breast cancer patients. Perhaps as a result of their improved breathing, they also were better able to perform daily tasks such as climbing a flight of stairs, getting dressed or going grocery shopping.
Wyatt said she was surprised to find that reflexology's effects appeared to be primarily physical, not psychological. "We didn't get the change we might have expected with the emotional symptoms like anxiety and depression," she said. "The most significant changes were documented with the physical symptoms."
Also unexpected was the reduced fatigue reported by those who received the "placebo" foot massage, particularly since the reflexology group did not show similarly significant improvement. Wyatt is now researching whether massage similar to reflexology performed by cancer patients' friends and family, as opposed to certified reflexologists, might be a simple and inexpensive treatment option.
Reflexology did not appear to reduce pain or nausea, but Wyatt said that could be because the drugs for combating those symptoms are generally quite effective, so the women may not have reported them to begin with. Although health researchers only recently have begun studying reflexology in a scientifically rigorous way, it's widely practiced in many parts of the world and dates back thousands of years.

"Reflexology comes out of the Chinese tradition and out of Egypt," Wyatt said. "In fact, it's shown in hieroglyphics. It's been around for a very long time."

Wyatt's co-authors include MSU statistics and probability professor Alla Sikorskii and College of Nursing research assistant Mei You, along with colleagues from Northwestern University and the University of Texas Health Science Center at Houston.

http://www.eurekalert.org/pub_releases/2012-11/wuis-loa111312.php

Less of a shock

A novel electrotherapy greatly reduces the energy needed to shock a heart back into rhythm, potentially making implanted defibrillators more acceptable to patients

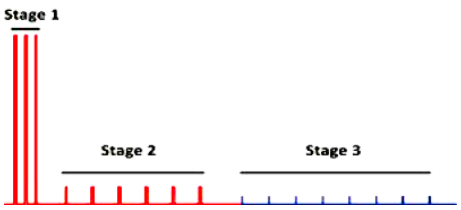
Implantable defibrillators currently on the market apply between 600 and 900 volts to the heart, almost 10 times the voltage from an electric outlet, says Ajit H. Janardhan, MD, PhD, a cardiac electrophysiology fellow at the Washington University's School of Medicine. After being shocked, he says, some patients get post-traumatic stress disorder. Patients may even go so far as to ask their physicians to remove the defibrillator, even though they understand that the device has saved their lives.

The huge shocks are not only unbearably painful, they damage the heart muscle and have been shown in many studies to be associated with increased mortality. In an advance online edition of the Journal of American College of Cardiology, Janardhan and Igor Efimov, PhD, professor of biomedical engineering in the School of Engineering & Applied Science, report on a low-energy defibrillation scheme that significantly reduces the energy needed to re-establish a normal rhythm in the heart's main chambers.

They hope this electrotherapy will be much less painful than shocks from existing implantable defibrillators, and may even fall beneath the threshold at which patients begin to perceive pain. The team has just received a National Institutes of Health grant to develop a prototype low-energy defibrillator for humans and plan to begin clinical trials of the device shortly.

Losing the beat

The lub-dub of the heartbeat begins with an electrical impulse generated by the sinoatrial node, a group of cells on the wall of the right atrium that is the heart's natural pacemaker. Spreading through conductive pathways in the heart, the electrical signal first causes the two upper chambers of the heart (the atria) to contract, and then, a split second later, the two lower chambers (the ventricles), coordinated motions that efficiently pump blood to the rest of the body.



The novel electrotherapy consists of multiple low-voltage shocks within one heart beat, followed by even lower voltage shocks and then by anti-tachycardia pacing. **Efimov et. al**

The synchronized squeezing of a normal heartbeat is called sinus rhythm, after the node that triggers it.

The rhythm can go wrong in many different ways, but the real killer is ventricular tachycardia. Ventricular tachycardia is an abnormal heart rhythm that starts in the ventricles rather than from the sinoatrial node, and that causes the heart to beat at a rate too fast (tachy is Greek for rapid or fast) to efficiently pump blood to the rest of the body.

Moreover, the rapid heartbeat can degenerate precipitously into ventricular fibrillation, or the loss of all rhythm, says Efimov. During ventricular fibrillation the uncoordinated contraction of heart muscle prevents the heart from pumping blood at all, and without immediate intervention, death quickly follows.

Most people who develop ventricular tachycardia and ventricular fibrillation outside the hospital die, says Janardhan, but studies show that if we implant a defibrillator in patients with a weak heart that does not pump as strongly as it should, we can significantly reduce mortality.

Restarting the rhythm

There are really only three therapies for ventricular tachycardia, Efimov says. One is drugs that reduce the likelihood of tachycardia, but drugs are often ineffective. The second is ablation, or the deliberate creation of nonconductive scar tissue within the heart that blocks abnormal conductive patterns and redirects electrical activity to more normal pathways. The major problem with ablation, says Efimov, is recurrence. It's a temporary measure, not a cure. Patients typically need additional treatment within five years.

The third therapy is an implantable cardioverter defibrillator, or ICD. These devices are placed beneath the skin in the chest and monitor the rate and rhythm of the heart. If they detect ventricular tachycardia, they try to break the rhythm by pacing the heart at a rate faster than its intrinsic rate, a strategy anti-tachycardia pacing.

Anti-tachycardia pacing is very low energy, so low that patients may not even sense it. But it is relatively ineffective when the heart is beating 200 time per minute or faster. At these higher rates, the ICS zaps the heart with a strong electrical shock that resets it and, with luck, allows the pacemaker node to restart it with a normal rhythm.

A novel electrotherapy

The scientists knew from earlier experiments that the voltage needed to shut down ventricular tachycardia depended on the timing of the shock. This led them to ask whether a sequence of multiple, closely timed low-voltage shocks might be more effective than a single high-voltage shock, and be less sensitive to timing. Indeed it turned out that if they shocked the heart multiple times they could reduce the peak shock amplitude from well over 200 volts to 20 volts, timing no longer mattered, and the therapy worked even if the ventricular tachycardia was very rapid. Although this electrotherapy involves multiple shocks, the total energy it delivers is still lower than that of a single large shock, roughly 80 times lower.

Why do multiple shocks work better? Arrhythmias generate electrical wave vortices — little electrical tornadoes in the heart — and it is these vortices, or re-entrant circuits, that make the heart beat too fast and prevent it from pumping properly. But immediately after it contracts, heart muscle goes through a refractory, or unresponsive, period during which it does not respond to electrical stimulation. The multiple shocks may do a better job of extinguishing the re-entrant circuits by creating an area of unresponsive muscle into which the re-entrant wavefront –the electrical tornado – crashes, the scientists suggest.

Relocating the Electrodes

Defibrillators now on the market apply shocks between the right ventricle (RV) and an "active can" located above the chest wall, below the collarbone. The shocks are painful in part because they pass through the chest wall muscle and sensory nerves.

The investigators found they could reduce peak shock voltages by an additional 50 percent if they applied shocks between the RV and coronary sinus (CS), a vessel that collects deoxygenated blood from the heart muscle, rather than through the chest wall. Less energy was required because the shocks were confined to the heart itself, and for the same reason they were also less painful. In an earlier paper, Efimov's student Wenwen Li, PhD, now at St. Jude Medical, had reported on a similar strategy for restoring the rhythm of the atria, the two upper chambers of the heart, for a less serious but more common rhythm abnormality.

The team has already developed the first low-energy atrial defibrillator, which will soon be entering clinical trials. They hope for similarly rapid progress with the ventricular defibrillator. "We think this technology can and will be implemented soon," says Janardhan. "There's a lot of cardiac research that may pan out 20 or 30 years from now," he says, "but as a physician I want something that can help my patients now."

http://www.eurekalert.org/pub_releases/2012-11/bc-ero110612.php

Experts report 1 of 2 remaining types of polio virus may be eliminated in Pakistan

But researchers, reporting at ASTMH annual meeting, also cite barriers to complete elimination from a surge of cases in Nigeria to intensifying vaccine refusals in Pakistan

ATLANTA - Polio cases worldwide reached historic lows in 2012, and for the first time there were no new outbreaks beyond countries already harboring the disease, leaving researchers confident that a massive and re-energized international campaign to eradicate polio is on a path to success, according to presentations today at the annual meeting of the American Society of Tropical Medicine and Hygiene (ASTMH).

Globally there were 177 polio cases through October 2012, a drop from 502 during the same period last year. Despite the dramatic drop, polio experts noted challenges in Pakistan posed by parents who refuse to vaccinate their children and in Nigeria where polio cases more than doubled in 2012 and threatened to re-infect currently polio-free countries. Pakistan, Nigeria and Afghanistan are the only countries where polio remains endemic and are the battle grounds of efforts to make polio only the second human disease, after smallpox, to be completely eliminated.

Steven Wassilak, MD, a medical epidemiologist and polio expert at the US Centers for Disease Control and Prevention (CDC), said new data from Pakistan show that of the two types of wild polio virus (WPV1 and WPV3) circulating in the country, the one known as WPV3 - or Type 3 - is close to being eliminated. "There have not been any Type 3 cases reported for six months, which is the longest gap in incidence there to date," he said. "CDC works with Pakistan officials to monitor different chains of transmission over time and Type 3 is now down to only one chain, which is an indication that we are close to breaking the last link of Type 3."

CDC, which has led the effort to establish a global network of laboratories to track and sequence the genome of the wild polio virus, is one of four partners spearheading the Global Polio Eradication Initiative along with Rotary International, UNICEF and the World Health Organization (WHO), with great support from the Bill and Melinda Gates Foundation. In September, the partners along with leaders from Pakistan, Nigeria, and Afghanistan met at United Nations Headquarters in New York to reaffirm the Emergency Action Plan against polio, launched in May. The initiative includes a "surge of human resources" involving 4,000 people who have been deployed to complete the eradication effort.

New Study Finds Vaccine Refusals Remain Barrier to Elimination

At the ASTMH conference, Anita Zaidi, MD, a pediatrician at Aga Khan University in Karachi who serves on Pakistan's National Immunization Technical Advisory Group, is presenting new data showing that while Pakistan has made major progress against polio by expanding immunization campaigns, the remaining challenge is not one easily solved by additional resources.

"We found that in Karachi, a key reason children fail to get immunizations is not due to lack of access, but because their parents refuse to participate," Zaidi said. "That is a big challenge and not something that can be overcome only by expanding immunization campaigns."

In a study published in the Bulletin of the World Health Organization (WHO), Zaidi and her colleagues found that in Karachi, "parent refusal was the most common reason given for the failure of children to participate in two recent polio supplementary immunization activities," accounting for 74 percent of missed immunizations.

While opposition to polio vaccinations has been widely reported in the remote, restive tribal territories in the north, less examined, Zaidi said, has been the situation in the urban environs of Karachi, which her study describes as "the only megacity in the world that has not succeeded in interrupting polio transmission."

Nationwide, Pakistani health officials believe vaccine refusals, driven by false rumors that immunizations cause sterility and are contaminated with HIV, actually are declining. But Zaidi said that recent events in Karachi suggest the intensity of those who remain opposed appears to be rising.

She pointed out that in just the last few months, a health worker in Karachi involved in the polio campaign was killed and the child of another was kidnapped (though later returned unharmed). Zaidi said that she recently participated in a polio seminar in Karachi where concerns about violence prompted the organizers to avoid any publicity. Given the current volatile environment and continued evidence of widespread viral transmission in sewage sampling, Zaidi said that while Pakistan is making considerable progress in reducing polio cases, eradication by 2013 appears unrealistic.

"Efforts should focus on building trust through grassroots efforts using community elders in populations with high vaccine refusal rates," she said. By providing vaccination at mass transit sites such as bus routes used to travel up-country throughout the year, we can at least isolate the viral reservoirs and make sure we avoid what happened last year, which was exporting the virus to China."

In Nigeria, Working With Traditional Leaders, Nomadic Groups

Meanwhile, opposition to immunizations is also a major challenge in Nigeria, where the number of polio cases has risen for the second straight year, said Adamu Nuhu, MD, with Nigeria's National Primary Healthcare Development Agency. He said cases are at least confined to the northern part of the country where opposition to immunizations is rooted in religious or political differences and, as in Pakistan, has been stirred by rumors of vaccine-induced sterilization and HIV infection.

"We are working now with traditional leaders in the north who are respected by local people to change the perceptions of polio immunization and encourage more participation in immunization efforts," Nuhu said. "There is evidence that overall, immunization rates among children at risk are rising to 80 percent."

He described a campaign that is now literally going house-to-house to identify family decision makers and talk with them about the importance of polio immunization. Also, health officials—who are working closely with international partners including the WHO, UNICEF and the CDC—are pursuing new strategies to improve immunization coverage in nomadic groups.

"Even though they are often moving, they have a leadership structure and we can work through these leaders to provide polio immunizations," Nuhu said. "But we understand that you will not be able to immunize any children unless you can reach them on their pastoral routes and camps."

The persistence of polio in Nigeria—in 2012 the country has documented 99 cases—is especially worrisome because cases in Nigeria have spread in the past to Sudan, Chad, and 23 other countries. All these countries are now once again polio-free except for Chad. The CDC reported in October that Chad could interrupt wild polio transmission by the end of this year, but that failure to stop transmission in Nigeria could prompt new outbreaks.

Vaccine Refusal: Lessons from India?

The CDC's Wassilak said the refusal by parents in Pakistan and Nigeria to vaccinate their children presents a challenge to eradication. "Karachi is a densely populated area where you need a high degree of immunity to interrupt polio transmission, so every child counts," he said.

But Wassilak said vaccine refusal is not an insurmountable barrier to global eradication.

"We saw similar problems in India, and while they were not resolved overnight, eventually we saw immunization coverage increase and polio cases halted," he said. "It requires working more closely with community leaders and greater political commitment at all levels, which is what we are seeing in both Pakistan and Nigeria."

India has not reported a polio infection since January 2011 and the entire Southeast Asia Region of WHO could be certified polio-free in 2014 if no new cases arise. Wassilak said advisors from India are now working in Nigeria to share their lessons learned. Meanwhile, the CDC is providing technical support to the global eradication effort, tracking the different types of polio that are circulating in the affected countries and training volunteers to assist in polio vaccination campaigns.

"This urgent international push for polio eradication means that soon no child will be hurt by this disease. The relentless drive to alleviate pain and suffering is the spirit of this Society and permeates every session and every hallway conversation at this meeting," said ASTMH President James W. Kazura, MD, FASTMH. "We will get this done."

http://www.sciencedaily.com/releases/2012/11/121113134228.htm

Natural Product Produced by Marine Algae Shows Promise in Stroke Recovery Treatment

A new study using brevetoxin-2, a compound produced naturally by marine algae, stimulated nerve cell growth and plasticity in cultured mouse neurons.

ScienceDaily - This research advances a potentially new pharmacological treatment to aid recovery of brain function following a stroke or other traumatic brain injury. Stroke is a leading cause of death in the United States with more than 795,000 people suffering a stroke each year, according to the Center for Disease Control. Stroke is a leading cause of serious long-term disability and there is currently no drug treatment for post-stroke rehabilitation.

"Our research suggests that compounds like brevetoxin-2 can augment neuronal plasticity potentially providing a neural repair therapy for stroke recovery. If that outcome can be supported by further studies in animals and subsequently humans, it could have a profound impact on a currently non-treatable condition," said Thomas F. Murray, Ph.D. associate vice president for Health Science Research and professor and chair of the Department of Pharmacology, Creighton School of Medicine. The research team from Creighton University School of Medicine, University of North Carolina Wilmington, and Scripps Institution of Oceanography published their findings in the Nov. 12 online edition of the journal Proceedings of the National Academy of Sciences (PNAS).

The tiny marine dinoflagellate Karenia brevis produces brevetoxin, which in high concentrations is responsible for the harmful algal blooms known as red tides that occur in the waters off the west coast of Florida. The neurotoxin-laden red tide causes respiratory irritation in humans and central nervous system paralysis in fish.

"Brevetoxin is a neurotoxin that is known to activate nerves cells to fire spontaneously," said Dan Baden, Ph.D. He is director of the Center for Marine Science as well as a founding member and Executive Principal of MARBIONC at University of North Carolina Wilmington. "It's a great advancement to show that this naturally occurring ocean compound can stimulate nerve cell growth in cultured mouse cells."

Brevetoxin is one of more than 1,000 ocean organisms cultured at the University of North Carolina Wilmington's MARBIONC facility (Marine Biotechnology in North Carolina) for use in bio-medical research. The bioactive materials from Karenia brevis have been actively studied by Baden since the early 1970s. A clot that restricts blood flow to an area of the brain causes a stroke. Although the dead tissue cannot be revived, the brain can be trained to redirect nerve impulses to living nerve cells nearby.

Recent scientific studies have shown that rewiring of nerve cells following a stroke occurs as a result of heightened plasticity around the brain's damaged cerebral cortex, which is the area of the brain responsible for sensory and cognitive nervous system functions.

This new study showed enhanced neuronal sprouting, the growth of axons or dendrites of a nerve cell as well the formation of new synapses between nerve cells in mouse neurons in a culture dish.

J. George, D. G. Baden, W. H. Gerwick, T. F. Murray. Bidirectional influence of sodium channel activation on NMDA receptor-dependent cerebrocortical neuron structural plasticity. Proceedings of the National Academy of Sciences, 2012; DOI: 10.1073/pnas.1212584109

http://www.sciencedaily.com/releases/2012/11/121113134807.htm

Glutamate Neurotransmission System May Be Involved With Depression Risk

Researchers have found that variants in a group of genes involved in transmission of signals by the neurotransmitter glutamate appear to increase the risk of depression

ScienceDaily - Researchers using a new approach to identifying genes associated with depression have found that variants in a group of genes involved in transmission of signals by the neurotransmitter glutamate appear to increase the risk of depression. The report published in the journal Translational Psychiatry suggests that drugs targeting the glutamate system may help improve the limited success of treatment with current antidepressant drugs.

"Instead of looking at DNA variations one at a time, we looked at grouping of genes in the same biological pathways and found that a set of genes involved in glutamatergic transmisson was associated with the risk of depression," says Jordan Smoller, MD, ScD, director of the Psychiatric and Neurodevelopmental Genetics Unit in the Massachusetts General Hospital (MGH) Department of Psychiatry, senior author of the study. "Our findings are particularly interesting in light of recent studies showing that drugs affecting glutamate transmission can have rapid antidepressant effects."

While the risk of depression clearly runs in families, the genome-wide association studies typically used to identify gene variants that increase disease risk have been unable to find strongly associated genes. The research team -- which includes investigators from the Broad Institute of MIT and Harvard and other research centers in the U.S., Australia and the Netherlands -- adopted a strategy called gene set pathway analysis. Starting with a set of genes that previous studies had implicated in depression, they used an analysis process called text mining to scan the medical literature for information on the biological function of these genes. Based on those findings, they identified 178 biological pathways that included these genes. Only one of those pathways -- the one involved in transmission of neural signals carried by glutamate -- was significantly associated with the risk for depression.

"Glutamate is the excitatory transmitter most widely used by the central nervous system, and several studies in animals and humans have suggested that it may play a role in depression," explains Smoller, an associate professor of Psychiatry at Harvard Medical School. "Most intriguingly, recent studies have found that ketamine -- a drug known to block one glutamate receptor -- appears to have antidepressant effects that are much faster than those of traditional antidepressants, which can take several weeks to become effective. Now additional research needs to confirm these findings and investigate exactly how variation in glutamate function affects brain systems involved in depression."

Phil Hyoun Lee, PhD, of the MGH Psychiatric and Neurodevelopmental Genetics Unit (PNGU) is lead author of the Translational Psychiatry report. Additional co-authors are Roy Perlis, MD, Richie Siburian, Stephen Haddad, MS, Catherine Mayerfeld, and Shaun Purcell, PhD, MGH PNGU; Erroll Rueckert, PhD, MGH Center for Human Genetic Research; Jae-Yoon Jung, PhD, Harvard Medical School; Enda Byrne, PhD, Naomi Wray, PhD, and Nicholas Martin, PhD, Queensland Institute of Medical Research, Australia; Andrew Heath, DPhil, Michele Pergadia, PhD, and Pamela Madden, PhD, Washington University, St. Louis; Dorret Boomsma, PhD, and B.W. Penninx, PhD, VU University, Amsterdam, The Netherlands; and Pamela Sklar, MD, PhD, Mount Sinai School of Medicine, New York. Lee, Perlis, Rueckert, Purcell and Smoller are also affiliated with the Broad Institute. The study was supported by grants from the National Institute of Mental Health.

P H Lee, R H Perlis, J-Y Jung, E M Byrne, E Rueckert, R Siburian, S Haddad, C E Mayerfeld, A C Heath, M L Pergadia, P A F Madden, D I Boomsma, B W Penninx, P Sklar, N G Martin, N R Wray, S M Purcell, J W Smoller. Multi-locus genome-wide association analysis supports the role of glutamatergic synaptic transmission in the etiology of major depressive disorder. Translational Psychiatry, 2012; 2 (11): e184 DOI: 10.1038/tp.2012.95

http://www.sciencedaily.com/releases/2012/11/121113185200.htm

International Action Needed to Ensure the Quality of Medicines and Tackle the Fake Drugs Trade

A global treaty is urgently needed to tackle the deadly trade of substandard and fake medicines, say leading experts in a paper published on bmj.com today.

ScienceDaily - Their call comes just days before 100 World Health Organisation member states hold their first meeting to discuss the problem, and the authors hope it will help to influence the debate and lead to some concrete actions. The lethal meningitis outbreak in the US due to contaminated steroid injections has again highlighted the serious consequences of this global problem. Other recent examples include a heart medicine containing a toxic overdose of a malaria drug, which led to 125 deaths in Pakistan, and a fake cancer medicine containing starch and acetone trafficked to Canada and the US. The extent of harm to patients is still unknown.

Substandard and fake medicines harm and kill patients, write an international group of experts led by Amir Attaran from the University of Ottawa in Canada, with the help of the World Federation of Public Health Associations, International Pharmaceutical Federation and the International Council of Nurses.

In poor countries, the World Health Organisation estimates that over 10% of medicines may be "counterfeit" and, although medicine safety is better in rich countries, fake drugs still cause thousands of adverse reactions and some deaths. In the European Union medicines are now the leading illegitimate product seized at the border, increasing 700% from 2010 to 2011.

Yet despite years of debate, no agreement on how best to tackle this scandal has been reached, they argue. They say that progress on the twin challenges of safeguarding the quality of genuine medicine and criminalising falsified ones "has been held back by controversy over intellectual property rights and confusion over terms."

They believe that to move forward, several challenges must first be overcome. For example, anti-counterfeiting laws must shift from protecting commercial interests to protecting public health interests; there must be clear, internationally agreed definitions for different types of illegitimate medicines; and more transparent surveillance and research is needed to measure the global scale of the problem.
"We argue that tackling the challenges of poor quality, unsafe medicines requires a comprehensive global strategy on which all stakeholders agree," say the authors.
They point to other global treaties, for example on human trafficking or money laundering, that "have helped governments strengthen their laws and cooperate internationally to clamp down on the havens."
They also point out that under today's leading public health treaty -- the Framework Convention on Tobacco Control (FCTC) -- the law is now tougher on fake tobacco than on fake medicines.
They urge WHO to embark on a similar process to that used to create the FCTC, which they believe "avoids unnecessary controversy and can better enable governments, companies, advocates, and the health professions to protect the public's health."
A. Attaran, D. Barry, S. Basheer, R. Bate, D. Benton, J. Chauvin, L. Garrett, I. Kickbusch, J. C. Kohler, K. Midha, P. N. Newton, S. Nishtar, P. Orhii, M. McKee. How to achieve international action on falsified and substandard medicines. BMJ, 2012; 345 (nov13 22): e7381 DOI: 10.1136/bmj.e7381

http://www.eurekalert.org/pub_releases/2012-11/uom-af111312.php

Astronomers find 'homeless' planet wandering through space

The planet does not orbit a star and is the first of its kind to be found

A planet that is not orbiting a star, effectively making it homeless, has been discovered by a team of University of Montreal (UdeM) researchers working with European colleagues and data provided by the Canada-France-Hawaii Telescope (CFHT) and the European Southern Observatory's Very Large Telescope (VLT). "Although theorists had established the existence of this type of very cold and young planet, one had never been observed until today," said Étienne Artigau, an astrophysicist at UdeM. The absence of a shining star in the vicinity of this planet enabled the team to study its atmosphere in great detail. This information will in turn enable astronomers to better understand exoplanets that do orbit stars.

Free-floating planets are planetary-mass objects that have no gravitational link to a star. "Over the past few years, several objects of this type have been identified, but their existence could not be established without scientific confirmation of their age," explained Jonathan Gagné, a doctoral student of physics at UdeM. "Astronomers weren't sure whether to categorize them as planets or as Brown dwarfs. Brown dwarfs are what we could call failed stars, as they never manage to initiate nuclear reactions in their centres."

This closeup of an image captured by the SOFI instrument on ESO's New Technology Telescope at the La Silla Observatory shows the free-floating planet CFBDSIR J214947.2-040308.9 in infrared light. This object, which appears as a faint blue dot at the centre of the picture, is the closest such object to the Solar System. **P. Delorme, European Southern Observatory**

Gagné and Artigau, along with Lison Malo and Loïc Albert, all of whom are astrophysicists with UdeM and the Centre for Research in Astrophysics of Quebec (CRAQ), were able to find this planet with the assistance of French astronomers. Philippe Delorme, of the Laboratoire d'Astrophysique de l'Observatoire de Grenoble, was lead researcher. The planet is in fact called CFBDSIR2149 and appears to be part of a group of very young stars known as the AB Doradus Moving Group. "This group is unique in that it is made up of around thirty starts that all have the same age, have the same composition and that move together through space. It's the link between the planet and AB Doradus that enabled us to deduce its age and classify it as a planet," Malo explained.
First of all, the researchers obtained a series of infrared images of CFBDSIR2149 using the 3.6 metres in diameter CFHT. They then used the full strength of the 8 metres in diameter VLT to deduce its mass, its temperature, and of particular note, its age. The planet was found to be between 50 and 120 million years old, with a temperature of approximately 400 degrees celsius, and a mass four to seven times that of Jupiter. Objects more than 13 times the mass of Jupiter are not considered to be planets but rather Brown dwarfs, as it is this is the minimum amount of mass required for the deuterium at the heart of a star to achieve fusion.
As an aside, it is interesting to note the significance of the finding in terms of the roots of the word "planet." "Planet as a word originates from the Latin word planetus, which originally comes from the Greek words planeta or planêtês, meaning moving or wandering celestial bodies, as opposed to stars which appeared to be in a fixed position in the sky," said Oliver Hernandez, an astrophysicist at UdeM.

In short, this is the first isolated planet – perhaps flung away during its formation – that is not tied by gravity to a star and whose mass, temperature and age meet the relevant criteria. This discovery, which has been sought after for more than a decade, supports theories relating to the formation of stars and planets. Moreover, it supports theories that suggest that these kinds of isolated objects are much more numerous than currently believed.
"This object was discovered during a scan that covered the equivalent of 1000 times the surface of the full moon," Artigau explained. "We observed hundreds of millions of stars and planets, but we only found one homeless planet in our neighbourhood. Now we will be looking for them amongst an astronomical number of sources further afield. It's like looking for a single needle in amongst thousands of haystacks."

http://www.eurekalert.org/pub_releases/2012-11/bmj-gpw111312.php

Give pregnant women vitamin D supplements to ward off MS, say researchers

Risk of MS highest in April and lowest in October, large analysis shows

The risk of developing multiple sclerosis (MS) is highest in the month of April, and lowest in October, indicates an analysis of the available evidence, published online in the Journal of Neurology Neurosurgery and Psychiatry. The findings, which include several populations at latitudes greater than 52 degrees from the equator for the first time, strongly implicate maternal exposure to vitamin D during pregnancy.
They extend previous research and prompt the authors to conclude that there is now a strong case for vitamin D supplementation of pregnant women in countries where ultraviolet light levels are low between October and March.
The researchers compared previously published data on almost 152,000 people with MS with expected birth rates for the disease in bid to find out if there was any link between country of birth and risk of developing multiple sclerosis. At latitudes greater than 52 degrees from the equator, insufficient ultraviolet light of the correct wavelength (290 to 315 nm) reaches the skin between October and March to enable the body to manufacture enough vitamin D during the winter months, say the authors. The analysis indicated a significant excess risk of 5% among those born in April compared with what would be expected. Similarly, the risk of MS was 5 to 7% lower among those born between October and November, the data indicated.
In order to exclude wholly or partially overlapping data, and therefore the potential to skew the data, the authors carried out a further "conservative analysis" in which such studies were left out. This reduced the number of people with MS to just under 78,500 and showed a clear link only between November and a reduced risk of MS. But this result is likely to have been due to the fact that all the excluded studies involved countries more than 52 degrees from the equator, explain the authors. When the same analysis was carried out again, but this time including all those involving people living in countries less than 52 degrees from the equator, the same seasonal trends were apparent. There was a significant increase in risk among those born in April and May and a significantly lower risk among those born in October and November.
No studies from the southern hemisphere were included in the analysis, largely because so few have been carried out, so the results should be viewed in light of that, caution the authors. But they conclude: "Through combining existing datasets for month of birth and subsequent MS risk, this study provides the most robust evidence to date that the month of birth effect is a genuine one." And they go on to say: "This finding, which supports concepts hypothesised some years previously, surely adds weight to the argument for early intervention studies to prevent MS through vitamin D supplementation."
The month of birth effect in multiple sclerosis: systematic review, meta-analysis and effect of latitude Online First doi 10.1136/jnnp-2012-303934

http://www.eurekalert.org/pub_releases/2012-11/uoe-nbq111412.php

New brain gene gives us edge over apes, study suggests

Scientists have taken a step forward in helping to solve one of life's greatest mysteries – what makes us human?

An international team of researchers have discovered a new gene that helps explain how humans evolved evolved from apes. Scientists say the gene – called miR-941 – appears to have played a crucial role in human brain development and may shed light on how we learned to use tools and language.
Researchers say it is the first time that a new gene – carried only by humans and not by apes – has been shown to have a specific function within the human body.
A team at the University of Edinburgh compared the human genome to 11 other species of mammals, including chimpanzees, gorillas, mouse and rat, to find the differences between them. The results, published in Nature Communications, showed that the gene – miR-941 – is unique to humans. The researchers say that it emerged between six and one million years ago, after humans had evolved from apes.

The gene is highly active in two areas of the brain that control our decision making and language abilities. The study suggests it could have a role in the advanced brain functions that make us human.
It is known that most differences between species occur as a result of changes to existing genes, or the duplication and deletion of genes. But scientists say this gene emerged fully functional out of non-coding genetic material, previously termed "junk DNA", in a startlingly brief interval of evolutionary time. Until now, it has been remarkably difficult to see this process in action.
Researcher Dr Martin Taylor, who led the study at the Institute of Genetics and Molecular Medicine at the University of Edinburgh, said the results were significant. He said: "As a species, humans are wonderfully inventive – we are socially and technologically evolving all the time. But this research shows that we are innovating at a genetic level too. This new molecule sprang from nowhere at a time when our species was undergoing dramatic changes: living longer, walking upright, learning how to use tools and how to communicate. We're now hopeful that we will find more new genes that help show what makes us human."
The team worked with scientists in China and Germany. The study was funded by the National Natural Science Foundation of China and the Medical Research Council.

http://www.eurekalert.org/pub_releases/2012-11/mc-gnt111412.php

Gene nearly triples risk of Alzheimer's, global team including Mayo Clinic finds

A gene so powerful it nearly triples the risk of Alzheimer's disease has been discovered by an international team including researchers from Mayo Clinic.

JACKSONVILLE, Fla. - It is the most potent genetic risk factor for Alzheimer's identified in the past 20 years. The findings were reported Wednesday in the online edition of the New England Journal of Medicine.
The team included researchers from 44 institutions around the world, including 10 from Mayo Clinic's campuses in Florida and Minnesota. The study was led by John Hardy, Ph.D., a researcher at the Institute of Neurology at University College London and a former professor at Mayo Clinic in Florida.
The researchers used new sequencing techniques to home in on the TREM2 gene. Additional TREM2 sequencing was then performed, in part, by scientist Aleksandra Wojtas in the Mayo Clinic in Florida laboratory of Rosa Rademakers, Ph.D. These studies led to identification of a set of rare variants in TREM2 that occurred more often in 1,092 Alzheimer's disease patients than in a control group of 1,107 healthy people. The most common variant, R47H, was then evaluated in follow-up studies of a large number of Alzheimer's disease patients and controls. Minerva Carrasquillo, Ph.D., a scientist in the Mayo Clinic in Florida laboratory of Steven Younkin, M.D., Ph.D., spearheaded the direct genotyping and analysis of R47H in DNA samples from 1,994 Alzheimer's disease patients and 4,062 "control" participants — individuals verified not to have Alzheimer's. The patients and control participants were evaluated by Mayo Clinic physicians, led by co-authors Dennis Dickson, M.D., Neill Graff-Radford, M.D., and Ronald Petersen, M.D., Ph.D. These follow-up studies showed unequivocally that the R47H variant of TREM2 substantially increases the risk of Alzheimer's disease.
"The TREM2 variant may be rare, but it is potent," Dr. Carrasquillo says. "In our series, it was present in 1.9 percent of the Alzheimer's patients and in only 0.37 percent of the controls. This strong effect rivals that of the well-established genetic variant known as APOE 4, and it was observed both in our study and in the independent study led by deCODE that was published with ours. R47H isn't fully penetrant — meaning that not all people who have the variant will develop Alzheimer's and in those who do, other genes and environmental factors will also play a role — but like APOE 4 it does substantially increase risk."
Dr. Younkin comments: "R47H is the first goldilocks variant to show strong association with Alzheimer's disease." Now being identified using the new sequencing technologies, goldilocks variants are an important type of rare variant so named because they are just right, not too rare and strong enough to show highly significant association in well-powered follow-up genotypic studies like the one performed at Mayo.
"There is a broad consensus that prevention will be the best way to manage Alzheimer's disease," Dr. Younkin says. "In my view, common variants like APOE 4 and goldilocks variants like TREM2 R47H are important because they could be used, in principal, to identify many healthy people at high risk of Alzheimer's disease who would be suitable for prevention trials. Patients whose Alzheimer's disease is driven by high risk genetic variants will frequently transmit these variants to their children. We now know that it takes a long time for the pathology of Alzheimer's disease to produce symptoms, so prevention in children who receive these variants would ideally begin when their elderly parents are diagnosed."
"The variant found in our study identifies a fascinating new Alzheimer's disease gene, TREM2, which is involved in the immune system," Dr. Rademakers says. "This fits well with other evidence linking the immune system to Alzheimer's disease, but additional studies are needed to establish that R47H does, in fact, act by altering immune function. Fortunately, this variant changes an amino acid in TREM2 and that will greatly facilitate the biological studies that follow."

The Mayo Clinic research was funded by the Robert and Clarice Smith and Abigail Van Buren Alzheimer's Disease Research Program, and by the National Institute on Aging through grants awarded to Drs. Petersen, Rademakers and Younkin.

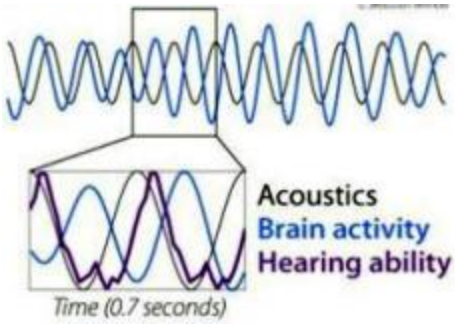
http://www.sciencedaily.com/releases/2012/11/121114113320.htm

Brain Waves Make Waves

Listeners' brain rhythms synchronize with the acoustic stimulus, which causes hearing abilities to "oscillate".

ScienceDaily - Naturally, our brain activity waxes and wanes. When listening, this oscillation synchronizes to the sounds we are hearing. Researchers at the Max Planck Institute for Human Cognitive and Brain Sciences have found that this influences the way we listen. Hearing abilities also oscillate and depend on the exact timing of one's brain rhythms. This discovery that sound, brain, and behaviour are so intimately coupled will help us to learn more about listening abilities in hearing loss.

Our world is full of cyclic phenomena: For example, many people experience their attention span changing over the course of a day. Maybe you yourself are more alert in the morning, others more in the afternoon. Bodily functions cyclically change or "oscillate" with environmental rhythms, like light and dark, and this in turn seems to govern our perception and behaviours. One might conclude that we are slaves to our own circadian rhythms, which in turn are slaves to environmental light-dark cycles.



Listeners' brain rhythms synchronize with the acoustic stimulus, which causes hearing abilities to "oscillate". **Credit: Sebastian Willnow**

A hard-to-prove idea in neuroscience is that such couplings between rhythms in the environment, rhythms in the brain, and our behaviours are also present at much finer time scales. Molly Henry and Jonas Obleser from the Max Planck Research Group "Auditory Cognition" now followed up on this recurrent idea by investigating the listening brain.

This idea holds fascinating implications for the way humans process speech and music: Imagine the melodic contour of a human voice or your favourite piece of music going up and down. If your brain becomes coupled to, or "entrained" by, these melodic changes, Henry and Obleser reasoned, then you might also be better prepared to expect fleeting but important sounds occurring in what the voice is saying, for example, a "d" versus a "t."

The simple "fleeting sound" in the scientists' experiment was a very short and very hard-to-detect silent gap (about one one-hundredth of a second) embedded in a simplified version of a melodic contour, which slowly and cyclically changed its pitch at a rate of three cycles per second (3 Hz).

To be able to track each listener's brain activity on a millisecond basis, Henry and Obleser recorded the electroencephalographic signal from listeners' scalps. First, the authors demonstrated that every listener's brain was "dragged along" (this is what entrainment, a French word, literally means) by the slow cyclic changes in melody; listeners' neural activity waxed and waned. Second, the listeners' ability to discover the fleeting gaps hidden in the melodic changes was by no means constant over time. Instead, it also "oscillated" and was governed by the brain's waxing and waning. The researchers could predict from a listener's slow brain wave whether or not an upcoming gap would be detected or would slip under the radar.

Why is that? "The slow waxings and wanings of brain activity are called neural oscillations. They regulate our ability to process incoming information," Molly Henry explains. Jonas Obleser adds that "from these findings, an important conclusion emerges: All acoustic fluctuations we encounter appear to shape our brain's activity. Apparently, our brain uses these rhythmic fluctuations to be prepared best for processing important upcoming information." The researchers hope to be able to use the brain's coupling to its acoustic environment as a new measure to study the problems of listeners with hearing loss or people who stutter.

Henry, M.J. and Obleser, J. Frequency modulation entrains slow neural oscillations and op-timizes human listening behavior. PNAS, 2012 (in press)

http://www.bbc.co.uk/news/uk-england-sussex-20329103

Sussex hospital pioneers hip operation to cut recovery time

A new way of doing hip operations in Sussex has reduced the amount of time patients spend in hospital by almost a half.

St Richard's Hospital, in Chichester, is using local anaesthetic, instead of general anaesthetic and morphine during surgery. Hospital staff say that some patients who have had full hip replacements are up and walking within hours. Time spent in hospital has fallen from seven days to less than four. The patient is injected with local anaesthetic into their back and affected joint.

'Big change'

Dr Cathryn Eitel, consultant anaesthetist, said: "Patients can expect to be up and about the day they have the surgery. "They will all be able to eat and drink on the day of their surgery and they will no longer have to have fluids attached to them." The operation takes an hour-and-a-half, during which the patient is awake, though can be sedated to make them feel "woozy".

Mark White, senior physiotherapist, said the surgery had been a "big change". "People are feeling less dizzy, less nauseous and having less pain," he said.

St Richard's said other hospitals around the country have also stopped using morphine, but it is the first to develop a specialist system involving operating staff, physios and recovery nurses in the procedure.

http://www.sciencedaily.com/releases/2012/11/121114133923.htm

20-Year-Old Stroke Patient Part of Growing Trend of Strokes in Young People

Michelle Nimmerrichter was only 20 years old when she suffered a stroke that left her in a coma and on a ventilator.

ScienceDaily - She appears to be part of a trend -- a recent study found that strokes are affecting people at younger ages. Nimmerrichter was paralyzed on one side and unresponsive when she arrived at Loyola University Medical Center on May 13th, 2011.

"She was very critically ill," said Dr. Jose Biller, a neurologist who directed her care. Biller is an internationally known expert on strokes in young people, and chair of Loyola's Department of Neurology.

Nimmerrichter has made a remarkable recovery and has returned to college. The main lingering effect is minor dystonia (involuntary muscle contraction) in her right hand. It doesn't stop her from eating her favorite food (cupcakes) or holding her cat, Cupcake.

Biller determined that Nimmerrichter suffered an unusual type of stroke that was triggered by a blood clot in the deep veins in her brain, leading to massive brain swelling. The clot was likely due to two factors: Nimmerrichter's blood has a genetic abnormality that makes it more prone to clotting, and she was taking a hormonal contraceptive, which increases the risk of blood clots.

Biller treated Nimmerrichter with a blood thinner, and medications to relieve pressure in her brain caused by the swelling. She spent two weeks at Loyola and nearly three weeks at a rehabilitation hospital.

Biller said Nimmerrichter's case illustrates the benefits of treating each patient as an individual. "At Loyola, we treat individual patients with stroke, rather than strokes generically," Biller said. "We avoid the temptation of the one-size-fits-all approach, so pervasive with cookbook medicine."

Nimmerrichter's stroke occurred just after she finished her sophomore year at Loyola University Chicago. It was Friday the 13th, and she was home alone at her mother's house in Brookfield. She was discovered by her boyfriend, who came to see her after she failed to respond to a text. He found her laying in bed, staring blankly at the wall.

Her mother, Mari Nimmerrichter, was shocked when she was told her daughter had suffered a stroke. "Most people equate strokes with people who are 50 or 60 or older," she said. But a recent University of Cincinnati College of Medicine study found that strokes among people under age 55 in the greater Cincinnati area increased from 13 percent of all strokes in 1993-94 to 19 percent in 2005. The study was published in Neurology®, the journal of the American Academy of Neurology.

"Strokes can occur at any age," Biller said. "The impact of strokes can be devastating to young adults, their families and society."

Biller is author of the textbook, "Stroke in Children and Young Adults," and a co-author of the American Heart Association's guidelines for management of stroke in infants and children.

The quicker a patient is diagnosed and treated, the better the outcome. "But people don't think that children and young adults can get strokes," Biller said. "So family members often are slow to recognize strokes."

Warning signs of stroke include sudden: Weakness of the face, arm or leg, especially on one side of the body. Numbness or tingling of the face or one side of the body. Confusion or trouble understanding. Trouble speaking. Trouble seeing in one or both eyes. Trouble walking; dizziness; loss of balance or coordination. Severe, unusual headaches.

A stroke kills 32,000 brain cells each second. So if you or a family member experience stroke symptoms, Biller advises, call 911 immediately.

"Every second counts," Biller said. "Time is brain."

http://phys.org/news/2012-11-venomous-aussie-redback-spiders-tokyo.html

Venomous Aussie redback spiders zero in on Tokyo

Australia's venomous redback spider, first sighted in Japan 17 years ago, is crawling toward Tokyo, with at least one of the creepy-crawlies found in a neighbouring city, officials said.

Australia's venomous redback spider, first sighted in Japan 17 years ago, is crawling toward Tokyo, with at least one of the creepy-crawlies found in a neighboring city, officials said Wednesday.

A man in Kawasaki, which borders southern Tokyo, used an insecticide to kill a strange spider and three egg sacs found inside a sprinkler in his garden on Monday, authorities said. City officials later confirmed the insect to be a redback, which the national government has labelled an invasive alien species deemed a menace to the country's ecosystem.

The Environment Ministry said redbacks had been found in 23 of Japan's 47 administrative districts. The district including Kawasaki is the closest the spiders have been detected to Tokyo.

Two female spiders on a 60cm-long slender branch in Kagoshima, Japan in 2002.

"They are presumed to have spread their habitats widely in Japan as they were carried while nestling in cargo, containers, construction materials, automobiles and such" after possibly arriving by sea originally, the ministry said on its website.

"I myself feel that they have come so close," Katsutoshi Oikawa, an environmental official at Tokyo's metropolitan government, told AFP. "We have not yet proactively worked out measures. But we have been responding to individual inquiries from citizens about ways to deal with the spiders."

Redback bites, which inject a potent neurotoxin, have caused numerous deaths in Australia, although an antivenom stocked in hospitals has prevented fatalities more recently.

Redbacks were first spotted in Japan in 1995 around Osaka, a major port where, experts believe, they may have arrived in a container of Australian woodchips used to make paper in Japan.

http://www.eurekalert.org/pub_releases/2012-11/uot-ais111212.php

Archaeologists identify spear tips used in hunting a half-million years ago

Findings suggest hunting with stone-tipped spears began much earlier than previously believed

TORONTO, ON – A University of Toronto-led team of anthropologists has found evidence that human ancestors used stone-tipped weapons for hunting 500,000 years ago – 200,000 years earlier than previously thought.

"This changes the way we think about early human adaptations and capacities before the origin of our own species," says Jayne Wilkins, a PhD candidate in the Department of Anthropology at the University of Toronto and lead author of a new study in Science. "Although both Neandertals and humans used stone-tipped spears, this is the first evidence that the technology originated prior to or near the divergence of these two species," says Wilkins.

Attaching stone points to spears – known as 'hafting' – was an important advance in hunting weaponry for early humans. Hafted tools require more effort and foreplanning to manufacture, but a sharp stone point on the end of a spear can increase its killing power.

This is a ~500,000-year-old point from Kathu Pan 1. Multiple lines of evidence from a University of Toronto-led study indicate that points from Kathu Pan 1 were used as hafted spear tips. Scale bar = 1 cm.

Credit: Jayne Wilkins

Hafted spear tips are common in Stone Age archaeological sites after 300,000 years ago. This new study shows that they were also used in the early Middle Pleistocene, a period associated with Homo heidelbergensis and the last common ancestor of Neandertals and modern humans.

"It now looks like some of the traits that we associate with modern humans and our nearest relatives can be traced further back in our lineage", Wilkins says.

Wilkins and colleagues from Arizona State University and the University of Cape Town examined 500,000-year-old stone points from the South African archaeological site of Kathu Pan 1 and determined that they had functioned as spear tips.

Point function was determined by comparing wear on the ancient points to damage inflicted on modern experimental points used to spear a springbok carcass target with a calibrated crossbow. This method has been used effectively to study weaponry from more recent contexts in the Middle East and southern Africa. The

stone points exhibit certain types of breaks that occur more commonly when they are used to tip spears compared to other uses.
"The archaeological points have damage that is very similar to replica spear points used in our spearing experiment," says Wilkins. "This type of damage is not easily created through other processes."
The findings are reported in the paper "Evidence for Early Hafted Hunting Technology" published in the November 16, 2012 issue of Science. Other authors contributing to the study are Benjamin Schoville from Arizona State University, Kyle Brown of the University of Cape Town, and University of Toronto archaeologist Michael Chazan. Funding for the research was provided by the Social Sciences and Humanities Research Council of Canada, the National Science Foundation, and the Hyde Family Foundation. Logistical support came from the South African Heritage Resources Agency and the McGregor Museum, Kimberley, South Africa.
The points were recovered during 1979-1982 excavations by Peter Beaumont of the McGregor Museum. In 2010, a team directed by Chazan reported that the point-bearing deposits at Kathu Pan 1 dated to ~500,000 years ago using optically stimulated luminescence and U-series/electron spin resonance methods. The dating analyses were carried out by Naomi Porat, Geological Survey of Israel, and Rainer Grün, Australian National University.

http://www.eurekalert.org/pub_releases/2012-11/uomh-wtg111512.php

When the going gets tough, the tough get... more relief from a placebo?

University of Michigan-led brain research may help explain why sham medicines work for some more than others, and could be used to improve tests of new treatments

ANN ARBOR, Mich. - Are you good at coping when life gets tough? Do people call you a straight-shooter? Will you help others without expecting anything in return? Those personality traits might do more than help you win a popularity contest. According to new University of Michigan-led neuroscience research, those qualities also might make you more likely to get pain relief from a placebo – a fake medicine.
And, the researchers show, it's not just your mind telling you the sham drug is working or not. Your brain's own natural painkiller chemicals may actually respond to the pain differently depending on your personality.
If you're more of an angry, hostile type, they find, a placebo won't do much for you.
For the first time, the new findings link specific, established personality traits with an individual's susceptibility to the placebo effect from a sham medicine for pain. The researchers showed a significant link between certain personality traits and how much relief people said they felt when given the placebo – as well as the level of a specific chemical that their brains released. The work, published online today in the journal Neuropsychopharmacology, was done by a team of U-M Medical School researchers and their colleagues at the University of North Carolina and University of Maryland.
The results build on nearly a decade's worth of work on the placebo effect by the team led by Jon-Kar Zubieta, M.D., Ph.D., the Phil Jenkins Professor of Depression in the U-M Department of Psychiatry, a professor in the Department of Radiology and a member of the Molecular and Behavioral Neuroscience Institute.
The findings show that about one-quarter of placebo response was explained by the personality traits of resiliency, straightforwardness, altruism or anger/hostility, as measured on standardized tests. Other personality traits didn't appear to be linked to placebo response. The new results come from a few dozen healthy volunteers, so the experiment must be repeated in larger, more diverse groups to be confirmed.
If confirmed, the findings could help researchers who study new drugs and other treatments – a field where placebo responses can really muddy the results and make it unclear whether the real therapy is working. Perhaps one day researchers will be able to adjust their results to account for the individual placebo responses of volunteers in their clinical trials.
Zubieta notes that the new findings came from a study involving pain, but that it may also apply to how personality influences a person's response to other stress-inducing circumstances.
"We started this study not just looking at measures that might seem more obviously related to placebo responses, such as maybe impulsivity, or reward-seeking, but explored potential associations broadly without a particular hypothesis," he explains. "We ended up finding that the greatest influence came from a series of factors related to individual resiliency, the capacity to withstand and overcome stressors and difficult situations. People with those factors had the greatest ability to take environmental information -- the placebo -- and convert it to a change in biology."
He and his team, including first author, former MBNI postdoctoral fellow and now psychiatry research investigator Marta Peciña, M.D., Ph.D., hope to continue the research in people with depression, and to continue to explore how genetics as well as personality influence placebo response.

He notes that the findings may even have implications for the doctor-patient relationship – for instance, patients who have certain personality traits and placebo-response tendencies may also be more likely to partner with their doctors on their care, and discuss frankly any concerns they have about their response to treatment.
How it was done:
The researchers conducted the study among nearly 50 healthy volunteers, both male and female, between the ages of 19 and 38. They gave each person a battery of standard psychological tests that help identify the strongest personality traits an individual has, and then had them lie down in a brain scanner called a positron emission tomography or PET machine.
They told the volunteers that they were going to experience pain from salt water injected into their jaw muscle, and that a painkiller – actually, a placebo – would be injected at certain times. They asked patients to rate how much relief they expected to get before the experiment began. Then, during the 20-minute period when volunteers received salt water and/or "painkiller", they asked them repeatedly to say how effective they though the painkiller was.
Meanwhile, the PET scanner made images of volunteers' brains, allowing the researchers to see how much of the natural painkillers called endogenous opioids, were released in certain areas of each person's brain under painful or "painkiller" conditions. They also drew blood from some of the patients during the experiment, and measured levels of a stress-induced chemical called cortisol.
After the tests, the researchers performed sophisticated statistical analysis to determine how personality traits influenced pain ratings, brain chemical response and cortisol levels. Although the cortisol levels did not seem to be influenced by personality traits and the placebo effect, the endogenous opioid activation elicited by the placebo, as well as patient-rated pain intensity levels, were.
In addition to Zubieta and Peciña, the team included Hamdan Azhar, M.S. and Tingting Lu, M.S., of MBNI and the U-M School of Public Health's Biostatistics program, MBNI research investigator Tiffany M. Love, Ph.D., Barbara L. Fredrickson, Ph.D. of UNC, and Christian S. Stohler, D.M.D. of U. Maryland. The research was supported by grants from the National Institutes of Health, including R01 AT 001415, R01 DA 016423 and R01 DA 022520, and by the Phil F. Jenkins Foundation.
Volunteers are needed for other studies by this team; visit www.umclinicalstudies.org and search for Zubieta.

http://www.eurekalert.org/pub_releases/2012-11/uol-mrw111512.php

Meteorites reveal warm water existed on Mars

Hydrothermal fractures around Martian impact craters may have been a habitable environment for microbial life

New research by the University of Leicester and The Open University into evidence of water on Mars, sufficiently warm enough to support life, has been published this week in the journal Earth and Planetary Science Letters. The study determined that water temperatures on the Red Planet ranged from 50°C to 150°C. Microbes on Earth can live in similar waters, for example in the volcanic thermal springs at Yellowstone Park, the scientists behind the research point out. The research is based on detailed scrutiny of Mars meteorites on Earth using powerful microscopes in the University of Leicester Department of Physics and Astronomy. This was followed-up by computer modeling work at The Open University.
Dr John Bridges, Reader in Planetary Science in the University of Leicester Space Research Centre and Lead Author, said: "Rovers on Mars – the Mars Exploration rovers Spirit and Opportunity, and the Mars Science Laboratory rover Curiosity – are studying rocks to find out about the geologic history of the Red Planet. Some of the most interesting questions are what we can find out about water, how much there was and what temperature it might have had.
"While the orbiters and rovers are studying the minerals on Mars, we also have meteorites from Mars here on Earth. They come in three different groups, the shergottites, the nakhlites and the chassignites. Of most interest for the question of water on Mars are the nakhlites, because this group of Martian meteorites contains small veins, which are filled with minerals formed by the action of water near the surface of Mars."
Dr. Bridges and his group studied those alteration minerals in great detail. Altogether eight nakhlite Martian meteorites are known, and all have small but significant differences between them and in their alteration minerals.
Lafayette is one of them; and the most complete succession of newly formed minerals can be found in its veins (see figure). Careful investigations of the minerals with an electron microscope and a transmission electron microscope have revealed that the first newly formed mineral to grow along the walls of the vein was iron carbonate. The carbonate would have been formed by CO2-rich water around 150°C. When the water cooled to 50°C, it would have formed the clay minerals, which were then followed by an amorphous phase that has the same composition as the clay.
Microbes use the reactions during mineral formation to gain energy and elements essential for their survival.

Dr Bridges added: "The mineralogical details we see tell us that there had been high carbon dioxide pressure in the veins to form the carbonates. Conditions then changed to less carbon dioxide in the fluid and clay minerals formed. We have a good understanding of the conditions minerals form in but to get to the details, chemical models are needed."
Dr Susanne Schwenzer, Postdoctoral Research Associate in the Department of Physical Sciences at The Open University who previously studied Martian meteorite compositions, said: "Until John's study was finished, I used the findings from orbiters around Mars, and modelled each of the new minerals individually. Those orbiters have found clays on the surface of Mars, but the spatial resolution is very different from the detailed study achieved in the nakhlites. Before we had the detailed study of the nakhlite meteorites, we did not know that carbonates are forming first, followed by the clays. Therefore I was very excited to see the details of the new mineralogical study."
By combining data from both universities, researchers were able to predict water conditions on Mars. Initially, the water was around 150°C and contained a lot of CO2, forming the carbonates, then cooled to about 50°C, thus forming the clays.
"The driving force heating the water might have been an impact into the Martian surface." Dr. Bridges explains. "And you only have to look at a map of Mars to see how numerous those are on the Martian surface," Dr. Schwenzer adds.
1. Bridges J.C. and Schwenzer S.P. The nakhlite hydrothermal brine on Mars. Earth and Planetary Science Letters 359 (2012) 117.

http://www.eurekalert.org/pub_releases/2012-11/eic-hhf111512.php

Hubble helps find candidate for most distant object in the universe yet observed

Astronomers have found what is probably the most distant galaxy yet seen in the Universe

By combining the power of the NASA/ESA Hubble Space Telescope, NASA's Spitzer Space Telescope and one of nature's zoom lenses, astronomers have found what is probably the most distant galaxy yet seen in the Universe. The object offers a peek back into a time when the Universe was only 3 percent of its present age of 13.7 billion years.
We see the newly discovered galaxy, named MACS0647-JD, as it was 420 million years after the Big Bang. Its light has travelled for 13.3 billion years to reach Earth, which corresponds to a redshift of approximately 11 [1]. This is the latest discovery from the Cluster Lensing And Supernova survey with Hubble (CLASH) [2], which uses massive galaxy clusters as cosmic telescopes to magnify distant galaxies behind them, an effect called gravitational lensing.
"While one occasionally expects to find an extremely distant galaxy using the tremendous power of gravitational lensing, this latest discovery has outstripped even my expectations of what would be possible with the CLASH program," said Rychard Bouwens (Leiden University, Netherlands), a co-author of the study. "The science output in this regard has been incredible."
Along the way, 8 billion years into its journey, the galaxy's light took a detour along multiple paths around the massive galaxy cluster MACS J0647.7+7015. Due to the gravitational lensing, the team observed three magnified images of MACS0647-JD with Hubble. The cluster's gravity boosted the light from the faraway galaxy, making the images appear far brighter than they otherwise would, although they still appear as tiny dots in Hubble's portrait.
"This cluster does what no manmade telescope can do," said Marc Postman (Space Telescope Science Institute, USA), leader of the CLASH team. "Without the magnification, it would require a Herculean effort to observe this galaxy."
The object is so small it may be in the first stages of galaxy formation, with analysis showing the galaxy is less than 600 light-years across. For comparison the Milky Way is 150 000 light-years across. The estimated mass of this baby galaxy is roughly equal to 100 million or a billion suns, or 0.1 - 1 percent the mass of our Milky Way's stars.
"This object may be one of many building blocks of a galaxy," explained Dan Coe (Space Telescope Science Institute), lead author of the study. "Over the next 13 billion years, it may have dozens, hundreds, or even thousands of merging events with other galaxies and galaxy fragments."
The team spent months systematically ruling out all other alternative explanations for the object's identity before concluding that it is the distance record holder. This was important, as nearby objects (such as red stars, brown dwarfs and old or dusty galaxies) can mimic the appearance of an extremely distant galaxy and must be carefully excluded.
The area around the galaxy was observed by Hubble through 17 filters -- spanning near-ultraviolet to near-infrared wavelengths -- with the galaxy appearing only in the two reddest filters. This was consistent with a

highly redshifted galaxy, but did not fully exclude other possibilities. Images of the galaxy at longer infrared wavelengths taken by Spitzer were more conclusive, however: if the object were intrinsically red, it would appear bright in these images. Instead, the galaxy was barely detected, if at all.
MACS0647-JD may be too far away for any current telescope to confirm the distance with spectroscopy [3]. Nevertheless, all the evidence points towards the fledgling galaxy being the new distance record holder.
The galaxy will almost certainly be a prime target for the James Webb Space Telescope, scheduled for launch in 2018, which will be able to conduct spectroscopy to make a definitive measurement of its distance and study its properties in more detail.

[1] Redshift is a consequence of the expansion of space over cosmic time, which stretches the wavelength of light. This has the consequence of making a distant object appear redder than it really is. Objects with a higher redshift have had their light stretched more, and are more distant. The previous candidate for the most distant object observed has a redshift of 10.3 ([heic1103][1]); confirmations of several objects with redshifts between 7 and 9 have been reported using spectroscopy, which gives more robust results (see for example [eso1041][2]). This newly discovered galaxy's redshift has been calculated as being approximately 10.8, with a 95% confidence that it lies between 10.3 and 11.3.

[2] The new distance champion is the second remote galaxy uncovered in the CLASH survey, a multiwavelength census of 25 hefty galaxy clusters with Hubble's Advanced Camera for Surveys (ACS) and Wide Field Camera 3 (WFC3) instruments. Earlier this year, the CLASH team announced the discovery of a galaxy that existed when the universe was about 490 million years old (redshift 9.6), 70 million years later than the new record-breaking galaxy. So far, the survey has completed observations for 20 of the 25 clusters.

[3] Redshift can only be precisely measured using spectroscopy, in which an object's light is dispersed and its colour probed in detail. However, it can be estimated by comparing images made of an object through different coloured filters, a method called photometric redshift. The galaxy will only be visible in some of the filters (having been redshifted out of some filters altogether), and the redshift is derived from the bluest filter in which the galaxy is visible. Photometric redshifts, as used in this study, provide less certainty, but they can be calculated for objects much fainter than is possible with spectroscopic redshifts. For this reason, the paper refers to the object as a 'candidate z≈11 galaxy'

[1]: http://www.spacetelescope.org/news/heic1103[2]: http://www.eso.org/public/news/eso1041

The international team of astronomers in this study consists of:

Dan Coe (Space Telescope Science Institute, Baltimore, USA), Adi Zitrin (Institut fur Theoretische Astrophysik, Heidelberg, Germany), Mauricio Carrasco (Institut fur Theoretische Astrophysik, Heidelberg, Germany and Pontifica Universidad Catolica de Chile, Santiago, Chile), Xinwen Shu (University of Science and Technology of China, Hefei, China), Wei Zheng (Johns Hopkins University, Baltimore, USA), Marc Postman (Space Telescope Science Institute, Baltimore, USA), Larry Bradley (Space Telescope Science Institute, Baltimore, USA), Anton Koekemoer (Space Telescope Science Institute, Baltimore, USA), Rychard Bouwens (Leiden University, Netherlands), Tom Broadhurst (University of the Basque Country, Bilbao, Spain and Ikerbasque Basque Foundation for Science, Bilbao, Spain) Anna Monna (Universitatssternwarter Munchen, Munich, Germany), Ole Host (University College London, London, UK and Dark Cosmology Centre, Niels Bohr Institute, University of Copenhagen, Denmark), Leonidas A. Moustakas (Jet Propulsion Laboratory, California Institute of Technology, La Cañada Flintridge, USA), Holland Ford (Johns Hopkins University, Baltimore, USA), John Moustakas (Siena College, Loudonville, USA), Arjen van der Wel (Max Planck Institute for AStronomy, Heidelberg, Germany), Megan Donahue (Michigan State University, East Lansing, USA), Steven A. Rodney (Johns Hopkins University, Baltimore, USA), Narciso Bentez (Instituto de Astrofisica de Andalucia, Granada, Spain), Stephanie Jouvel (University College London, London, UK and Institut de Cincies de l'Espai, Bellaterra (Barcelona), Spain), Stella Seitz (Universitatssternwarte Munchen, Munich, Germany and Max Planck Institute for Extraterrestrial Physics, Garching, Germany), Daniel D. Kelson (Carnegie Observatories, Pasadena, USA), and Piero Rosati (European Southern Observatory, Garching, Germany)

The research is presented in a paper entitled "CLASH: Three Strongly Lensed Images of a Candidate z≈11 Galaxy" to be published in the December 20 issue of the Astrophysical Journal.

http://www.eurekalert.org/pub_releases/2012-11/uoc--rrp111512.php

Researchers report potential new treatment to stop Alzheimer's disease

Molecular 'tweezers' break up toxic aggregations of proteins in mouse model

Last March, researchers at UCLA reported the development of a molecular compound called CLR01 that prevented toxic proteins associated with Parkinson's disease from binding together and killing the brain's neurons.

Building on those findings, they have now turned their attention to Alzheimer's disease, which is thought to be caused by a similar toxic aggregation or clumping, but with different proteins, especially amyloid-beta and tau. And what they've found is encouraging. Using the same compound, which they've dubbed a "molecular tweezer," in a living mouse model of Alzheimer's, the researchers demonstrated for the first time that the compound safely crossed the blood–brain barrier, cleared the existing amyloid-beta and tau aggregates, and also proved to be protective to the neurons' synapses — another target of the disease — which allow cells to communicate with one another. The report appears in the current online edition of the journal Brain.

"This is the first demonstration that molecular tweezers work in a mammalian animal model," said Gal Bitan, an associate professor of neurology at UCLA and the senior author of the study. "Importantly, no signs of

toxicity were observed in the treated mice. The efficacy and toxicity results support the mechanism of this molecular tweezer and suggest these are promising compounds for developing disease-modifying therapies for Alzheimer's disease, Parkinson's and other disorders."
Molecular tweezers are complex molecular compounds capable of binding to other proteins. Shaped like the letter "C," these compounds wrap around chains of lysine, a basic amino acid that is a constituent of most proteins. Bitan and his colleagues, including Aida Attar, first author of the study and a graduate student in Bitan's lab, have been working with a particular molecular tweezer called CLR01.
In collaboration with scientists at the Università Cattolica in Rome, the researchers, working first in cell cultures, found that CLR01 effectively inhibited a process known as synaptotoxicity, in which clumps of toxic amyloid damage or destroy a neuron's synapses.
Even though synapses in transgenic mice with Alzheimer's may shut down and the mice may lose their memory, upon treatment, they form new synapses and regain their learning and memory abilities.
"For humans, unfortunately, the situation is more problematic because the neurons gradually die in Alzheimer's disease," Bitan said. "That's why we must start treating as early as possible. The good news is that the molecular tweezers appear to have a high safety margin, so they may be suitable for prophylactic treatment starting long before the onset of the disease."
Next, using a radioactive "label," the researchers were able to confirm that the compound had crossed the mouse's blood–brain barrier and was effective in clearing the brain of amyloid-beta and tau aggregates.
"This work shows that molecular tweezers do a number of things — they help to ameliorate multiple pathologic features of Alzheimer's, including amyloid plaques, neurofibrillary tangles and brain inflammation, and our cell culture experiments demonstrated that molecular tweezers block the toxic effect of amyloid-beta on synaptic integrity and communication," Bitan said.
"We call these unique tweezers 'process-specific,' rather than the common protein-specific inhibitors," he added, meaning the compound only attacks the targeted toxic aggregates and not normal body processes. "That's a big deal, because it helps confirm evidence that the molecular tweezers can be used safely, ultimately supporting their development as a therapy for humans."
The next step, Bitan hopes, is to confirm that the tweezers improve memory and not just brain pathology. The researchers say they are working on this question and already have encouraging preliminary data.
There were multiple authors on the study in addition to Bitan and Attar. Please see the study for the complete list.
The work was supported by the UCLA Jim Easton Consortium for Alzheimer's Drug Discovery and Biomarker Development; American Health Assistance Foundation grant A2008-350; RJG Foundation grant 20095024; a Cure Alzheimer's Fund grant; individual pre-doctoral National Research Service Award 1F31AG037283; National Institute of Health grant R01AG021975; and a Veteran's Administration Merit Award.

http://nyti.ms/XSy7KL

New Infection, Not Relapse, Brings Back Lyme Symptoms, Study Says

When people who have been treated for Lyme disease recover but later come down with its symptoms again, is the illness a relapse or a new infection?

By DENISE GRADY

The question has lingered for years. Now, a new study finds that repeat symptoms are from new infections, not from relapses.
The results challenge the notion, strongly held by some patients and advocacy groups, that Lyme disease, a bacterial infection, has a tendency to resist the usual antibiotic treatment and turn into a chronic illness that requires months or even years of antibiotic therapy.
The conclusion that new symptoms come from new infections is based on genetically fingerprinting the Lyme bacteria in people who have had the illness more than once, and finding that the fingerprints do not match. The result means that different episodes of Lyme in each patient were caused by different strains of the bacteria, and could not have been relapses.
The study, by researchers at the University of Pennsylvania and New York Medical College, in Valhalla, was published online on Wednesday in The New England Journal of Medicine.
An estimated 20,000 to 30,000 cases of Lyme disease occur each year in the United States. The disease is caused by a bacterium, Borrelia burgdorferi, that is carried by deer ticks. It often begins with an expanding zone of red skin — a symptom called erythema migrans — around the tick bite, but sometimes in other areas too. Fever, headaches, fatigue and aches and pains often follow.
Untreated, the disease can cause heart and neurological problems and arthritis, with symptoms that can come and go for years. Advanced cases that have gone months or years before being treated are most likely to result in persistent arthritis.

But when the disease is detected earlier, treatment with an antibiotic, usually two to four weeks of doxycycline, can get rid of the bacteria, according to infectious disease experts. Even advanced cases can be cleared by the drugs, doctors say, though an extra month or so of treatment may be needed. Symptoms like pain and fatigue can linger even after the bacteria are gone, possibly because the infection caused abnormalities in the immune system.

However, some doctors, patients and advocacy groups think that the bacteria themselves can somehow hang on despite treatment, even in cases caught early, and cause a chronic infection that requires long-term treatment with antibiotics. In some cases, people with unexplained pain, fatigue and cognitive problems have been told they had chronic Lyme disease even though blood tests found no evidence of the infection.

Several controlled studies have found that long-term antibiotics did not help people who had already been treated for Lyme disease but had such lingering problems. Despite the data, the belief has hung on that Lyme disease bacteria can cause a chronic infection even after treatment.

The researchers who conducted the new study wanted to test that idea by finding out whether people who had repeated bouts of the disease were actually having relapses. They identified 17 patients who had erythema migrans — the rash — more than once between 1991 and 2011. Most had it twice, at least a year apart, but a few patients had it three times and one had four cases. Many had other symptoms as well, and more than half had signs of widespread systemic infection. All were treated, and recovered fully.

Lyme bacteria were grown from skin or blood samples taken from the patients when they had the rash, and the researchers analyzed a bacterial gene that varies from one strain to another. For each patient, they compared the genes from different cases of the rash. The genotypes did not match, which the researchers said proved that each rash represented a new infection, not a relapse.

In an editorial accompanying the article, Dr. Allen C. Steere, a Harvard professor who was the first to identify Lyme disease, said the new study supported previous research suggesting that new infections, not relapses, were the cause of new symptoms in people who had taken antibiotics to treat earlier cases of the disease.

Dr. Steere acknowledged that symptoms, sometimes disabling ones, do linger for months after treatment in as many as 10 percent of patients. Doctors do not know why. But, Dr. Steere said, “antibiotics are not the answer.”

http://arstechnica.com/science/2012/11/remodeled-enzyme-converts-carbon-dioxide-into-methane/

Remodeled enzyme converts carbon dioxide into methane

Its structure could provide clues that improve other catalysts.

by Melissae Fellet - Nov 16 2012, 1:30am TST

An engineered enzyme is the first single biological catalyst that converts carbon dioxide into a renewable form of energy: methane. Surprisingly, the same enzyme can use carbon dioxide to make an important ingredient in plastics as well.

Recycling carbon dioxide by turning it into fuels like methanol (CH_3OH) or methane (CH_4) might be one way to slow the CO_2 accumulation in our atmosphere. But that's quite a challenge, because CO_2 is a pretty inert molecule and doesn't readily participate in chemical reactions. So chemists have developed metal-containing catalysts to assist in the reduction reactions that convert it to methane and other carbon-containing small molecules. Alternatively, bacteria can use CO_2 to make methane, but they use a series of proteins to catalyze the transformation.

Lance Seefeldt at Utah State University and his colleagues study a bacterial enzyme called a nitrogenase, which reduces nitrogen gas (N_2) to ammonia (NH_3) with the help of a cluster of iron and molybdenum atoms buried inside the protein. The reduction of carbon dioxide to methane requires a transfer of eight electrons, just as ammonia production does, so the scientists wondered if an altered version of this enzyme could accept and reduce carbon dioxide.

They changed two amino acids in one subunit of this protein. The altered nitrogenase converts carbon dioxide to methane for 20 minutes and then slows down. The enzyme’s reaction rate and the number of reactions it catalyzes are comparable to similar soluble metal catalysts.

But the real surprise to Seefeldt was that the enzyme triggered a more complex reaction: it combined two molecules, carbon dioxide and acetylene, to form propylene, a three-carbon ingredient in many plastics. That particular reaction is new for any catalyst, inorganic or biological, he says.

The scientists want to test other versions of the enzyme to see if it can use CO_2 to build other kinds of molecules, too. Enzymes are used as biocatalysts to make some chemicals on an industrial scale, but that’s not Seefeldt’s ultimate goal in engineering this enzyme. He wants to extend this enzyme’s catalytic ability to better understand how the protein works.

Lessons about how the binding site environment helps catalyze a particular reaction might translate into clues that help other scientists build better catalysts for the production of methane and other commercially relevant chemicals. This altered enzyme won't solve our carbon dioxide or energy problems on its own. But its structure, or that of its yet-to-be-found mutant cousins, might provide some useful hints that do help us address those issues by recycling CO2 through chemistry.

Proc. Natl. Acad. Sci., 2012. DOI: 10.1073/pnas.1213159109 (About DOIs).

http://www.sciencenews.org/view/generic/id/346435/title/Ebola_may_go_airborne

Ebola may go airborne

Infected pigs can transmit virus to primates without contact, study finds

By Tina Hesman Saey

The Ebola virus can spread through the air from pigs to macaques, a new study suggests.

Transmission of the virus — which causes an often fatal hemorrhagic fever in people and primates — was thought to require direct contact with body fluids from an infected animal or person. But in the new study, published online November 15 in Scientific Reports, piglets infected with Ebola passed the virus to macaques housed in the same room even though the animals never touched.

"The evidence that the virus got from a pig to a monkey through a respiratory route is good," says Glenn Marsh, a molecular virologist at the Commonwealth Scientific and Industrial Research Organization's Animal Health Laboratory in Geelong, Australia. Marsh was not involved in the new study but has investigated Ebola and other viruses in pigs.

Although pigs transmitted Ebola in the laboratory, there is still no evidence that anyone has been sickened from contact with infected pigs in Africa, where the virus occurs naturally, or that the virus passes through the air under normal conditions, says study coauthor Gary Kobinger, an infectious disease researcher at the University of Manitoba in Winnipeg, Canada. "It's definitely not an efficient route of transmission."

Only 13 of the more than 2,200 human cases of Ebola documented since the virus was discovered in 1976 cannot be traced to direct contact with an infected person, animal or body fluid, he notes. If Ebola were able to spread easily through the air, many more cases might result.

The new study raises questions about whether humans can also transmit Ebola by respiratory routes, says Pierre Formenty, of the World Health Organization's Control of Epidemic Diseases Unit. That is something that will have to be investigated in future outbreaks, he says.

Kobinger became interested in Ebola in pigs after investigating an outbreak in 2007 in the Democratic Republic of the Congo. Villagers mentioned that some pigs had gotten sick and died early in the outbreak. At the time, there was no evidence that Ebola could infect pigs. Kobinger and his colleagues have since demonstrated that the virus causes disease in pigs in the lab, but no cases have been confirmed in livestock.

"This is all story-telling. Nobody has isolated virus or even detected antibodies from pigs in Africa," Kobinger says.

But other researchers discovered that pigs on farms in the Philippines could contract a form of the virus known as Reston Ebola. The Reston strain causes disease in macaques but has not been shown to make people sick. Some pig farmers in the Philippines have antibodies in their blood against Reston Ebola, indicating that infected pigs may have exposed farmers to the virus.

Kobinger wanted to know whether pigs could also pass along the form of Ebola found in Africa. Working in a lab designed to contain the most dangerous pathogens, Kobinger and his colleagues infected piglets with the strain known as Zaire Ebola. The piglets were housed next to four cynomolgus macaques, primates often used as stand-ins for humans. A barrier prevented the animals from coming into direct contact with each other.

After about a week living next to infected piglets, two of the macaques fell ill with Ebola. Those two animals were in cages in the path of air flowing from the pigs' enclosure. It took several more days for the other two macaques to develop the disease.

While the finding could indicate that the virus spread through the air, the researchers can't rule out that virus may have infected the macaques via water droplets scattered while cleaning the pig cage.

No one is blaming pigs for Ebola outbreaks in Africa now, but Kobinger says the growing pig industry on the continent might want to take a few simple steps to protect their animals. Keeping fruit trees, which attract fruit bats that carry Ebola, away from pig farms is one such measure.

Ebola viruses related to the African strains have been found in orangutans in Indonesia, raising the possibility that other unknown Ebola-like viruses could spill over into pigs and then humans, Marsh says. "That's concerning."

H.M. Weingartl et al. Transmission of Ebola virus from pigs to non-human primates. Scientific Reports. DOI: 10.1038/srep00811. ***[Go to]***

http://www.eurekalert.org/pub_releases/2012-11/bidm-gde111612.php

Gene distinguishes early birds from night owls and helps predict time of death

Common gene variant helps determine the time you will wake up each day -- and the time of day you are likely to die

BOSTON – Many of the body's processes follow a natural daily rhythm or so-called circadian clock. There are certain times of the day when a person is most alert, when blood pressure is highest, and when the heart is most efficient. Several rare gene mutations have been found that can adjust this clock in humans, responsible for entire families in which people wake up at 3 a.m. or 4 a.m. and cannot stay up much after 8 at night. Now new research has, for the first time, identified a common gene variant that affects virtually the entire population, and which is responsible for up to an hour a day of your tendency to be an early riser or night owl.

Furthermore, this new discovery not only demonstrates this common polymorphism influences the rhythms of people's day-to-day lives -- it also finds this genetic variant helps determine the time of day a person is most likely to die.

The surprising findings, which appear in the November 2012 issue of the Annals of Neurology, could help with scheduling shift work and planning medical treatments, as well as in monitoring the conditions of vulnerable patients.

"The internal 'biological clock' regulates many aspects of human biology and behavior, such as preferred sleep times, times of peak cognitive performance, and the timing of many physiological processes. It also influences the timing of acute medical events like stroke and heart attack," says first author Andrew Lim, MD, who conducted the work as a postdoctoral fellow in the Department of Neurology at Beth Israel Deaconess Medical Center (BIDMC).

"Previous work in twins and families had suggested that the lateness or earliness of one's clock may be inherited and animal experiments had suggested that the lateness or earliness of the biological clock may be influenced by specific genes," adds Lim, who is currently an Assistant Professor in the Division of Neurology at the University of Toronto.

The work originated several years ago while Lim was working in the laboratory of BIDMC Chief of Neurology Clifford Saper, MD, PhD. Lim and the other lab members were studying why older people have trouble sleeping and had joined a research project based at Rush University in Chicago involving 1,200 people who signed on as healthy 65-year-olds and would receive annual neurological and psychiatric examinations.

The cohort's original intent was to determine if there were identifiable precursors to the development of Parkinson's disease or Alzheimer's disease. As part of the research the subjects were undergoing various sleep-wake analyses using a wristband called an actigraph, which provides a reliable record of an individual's pattern of activity. Additionally, in order to provide the scientists with information on sleep-wake patterns within a year of death, the participants had agreed to donate their brains after they died.

But the investigation took a new turn when Lim learned that the same group of subjects had also had their DNA genotyped. Teaming up with investigators from Brigham and Women's Hospital (BWH), Lim and his colleagues compared the wake-sleep behavior of these individuals with their genotypes. These findings were later verified in a group of young volunteers.

They soon discovered a single nucleotide near a gene called "Period 1" that varied between two groups that differed in their wake-sleep behavior. At this particular site in the genome, 60 percent of individuals have the nucleotide base termed adenine (A) and 40 percent have the nucleotide base termed guanine (G). Because we have two sets of chromosomes, in any given individual, there's about a 36 percent chance of having two As, a 16 percent chance of having two Gs, and a 48 percent chance of having a mixture of A and G at this site.

"This particular genotype affects the sleep-wake pattern of virtually everyone walking around, and it is a fairly profound effect so that the people who have the A-A genotype wake up about an hour earlier than the people who have the G-G genotype, and the A-Gs wake up almost exactly in the middle," explains Saper, who is also the James Jackson Putnam Professor of Neurology and Neuroscience at Harvard Medical School. Also, expression of the Period 1 gene was lower in the brains and white blood cells of people with the G-G genotype than in people with the A-A genotype, but only in the daytime, which is when the gene is normally expressed.

This discovery marks the biggest contribution of a single genotype in a large population to determine the time of day when people wake up or go to sleep. But could the variant also affect other aspects of the body's circadian rhythm?

"Virtually all physiological processes have a circadian rhythm, meaning that they occur predominantly at certain parts of the day. There's even a circadian rhythm of death, so that in the general population people tend

on average to be most likely to die in the morning hours. Sometime around 11 am is the average time," says Saper.
When the investigators went back and looked at the people in the study (many of whom had enrolled more than 15 years ago at age 65) who had died, they found that this same genotype predicted six hours of the variation in the time of death: those with the AA or AG genotype died just before 11 a.m., like most of the population, but those with the GG genotype on average died at just before 6 p.m.
"So there is really a gene that predicts the time of day that you'll die. Not the date, fortunately, but the time of day," says Saper.
Lim says that additional work is needed to determine the mechanisms by which this and other gene variants influence the body's biological clock. In addition to helping people optimize their schedules, the research could eventually lead to novel therapies to treat disturbances of this clock as seen in jet lag or shift work.
"Also, working out which causes of death are influenced by gene variants like the one we identified may eventually lead to rational timed interventions—such as taking heart medications at particular times depending on which version of the gene variant one carries—to provide protection during an individuals' period of greatest risk," says Lim. The potential clinical applications may be as diverse as the many processes that the circadian clock controls.
In addition to Lim and Saper, study coauthors include Anne-Marie Chang, PhD, Joshua M. Shulman, MD, PhD, Towfique Raj, PhD, Lori B. Chibnik, PhD, Sean W. Can, PhD, Katherine Rothamel, BS, Christophe Benoist, PhD, Amanda J. Myers, PhD, Charles A. Czeisler, MD, PhD, Aron S. Buchman, MD, David A Bennett, MD, Jeanne F. Duffy, PhD, and Philip L. De Jager, MD, PhD.
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http://www.sciencedaily.com/releases/2012/11/121116124644.htm

Reconsidering Cancer's Bad Guy

This new research stands on the shoulders of many years of work on S100A4 in its deadlier role in cancer progression.

ScienceDaily - Researchers at the University of Copenhagen have found that a protein, known for causing cancer cells to spread around the body, is also one of the molecules that trigger repair processes in the brain. These findings are the subject of a paper, published this week in Nature Communications. They point the way to new avenues of research into degenerative brain diseases like Alzheimer's.
How to repair brain injuries is a fundamental question facing brain researchers. Scientists have been familiar with the protein S100A4 for some time as a factor in metastasis, or how cancer spreads. However it's the first time the protein has been shown to play a role in brain protection and repair.
"This protein is not normally in the brain, only when there's trauma or degeneration. When we deleted the protein in mice we discovered that their brains were less protected and able to resist injury. We also discovered that S100A4 works by activating signalling pathways inside neurons," says Postdoc Oksana Dmytriyeva, who worked on the research in a team at the Protein Laboratory in the Department of Neuroscience and Pharmacology at the University of Copenhagen.

The villain turns out to be the hero

This research stands on the shoulders of many years of work on S100A4 in its deadlier role in cancer progression. The discovery represents a significant development for the new Neuro-Oncology Group that moved to the University of Copenhagen's Protein Laboratory Group from the Danish Cancer Society in October.
"We were surprised to find this protein in this role, as we thought it was purely a cancer protein. We are very excited about it and we're looking forward to continuing our research in a practical direction. We hope that the findings will eventually benefit people who need treatment for neurodegenerative disorders like Alzheimer's disease, although obviously we have a long way to go before we get to that point," says Oksana Dmytriyeva.
The scientific paper The metastasis-promoting S100A4 protein confers neuroprotection in brain injury can be found online in the journal Nature Communications.
Oksana Dmytriyeva, Stanislava Pankratova, Sylwia Owczarek, Katrin Sonn, Vladislav Soroka, Christina M. Ridley, Alexander Marsolais, Marcos Lopez-Hoyos, Noona Ambartsumian, Eugene Lukanidin, Elisabeth Bock, Vladimir Berezin, Darya Kiryushko. The metastasis-promoting S100A4 protein confers neuroprotection in brain injury. Nature Communications, 2012; 3: 1197 DOI: 10.1038/ncomms2202

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http://news.discovery.com/human/strep-throat-treatment-121116.html#mkcpgn=rssnws1

Strep Throat: New Guidelines, Mysteries Remain

Every season, strep throat cases spike and now there is new advice for medical professionals treating patients.

By Sheila Eldred

It's the time of year when parents may dig through children's backpacks to find the dreaded note: "A case of strep throat has been identified in your child's classroom." Fall strep season is in full swing.

"Someone once said, strep infections are an occupational disease of schoolchildren," said Dr. Ed Kaplan, a professor at the University of Minnesota who leads the World Health Organization Collaborating Center for Reference and Research on Streptococci.

Strep throat (technically group A. streptococcus) is one of the most scientifically confounding -- and studied -- bacterial infections. While there are many unknowns, treatment hasn't changed much since you last sucked down syrupy bubblegum-pink medicine: A 10-day course of penicillin is still the gold standard. In fact, unlike other bacteria, there's never been a clinical isolate of group A strep that has resisted it, Kaplan said.

"It's still as exquisitely sensitive to penicillin as it was 50 years ago," Kaplan said. "How many times have you seen or read or heard people talking about the evolution of(bacteria resisting antiobiotics)? For reasons not fully understood, that hasn't happened with strep."

This fall, new guidelines from the Infectious Diseases Society of America (IDSA) suggest a slightly different course of action for adults: Doctors are encouraged to skip the culture and make treatment decision based solely on the rapid test. A new study found that while the rapid test is not 100 percent accurate (it failed 6 percent of the time in the researchers' examination of 20,000 patients), there were no complications from the missed cases. The cost of a backup test costs $113 at the Cleveland Clinic where the study was done. Although the risk of complication is low, strep can lead to scarlet fever, rheumatic fever, and acute nephritis.

Still, some doctors prefer to skip the rapid test, possibly starting antibiotics depending on a patient's symptoms, and wait for the results of the culture.

"If the rapid test is negative, the sensitivity varies from 50 to 95 percent -- I have seen negatives that are false negatives" Kaplan said.

Complicating the issue of diagnosis is one of strep's puzzles: Some kids seem to be "healthy carriers" of strep, meaning that doctors sometimes see asymptomatic patients whose cultures turn up positive.

"Usually they stop having symptoms if they're healthy carriers, but people can carry it for a long time -- 10 months or more," said Dr. Chris Van Beneden, an epidemiologist at the U.S. Centers for Disease Control and Prevention who helped develop the new IDSA guidelines. "It's one of those things where it's hard to do the studies because people won't tolerate being swabbed over and over."

Kaplan said he's seen people carry the infection for two years with no symptoms.

That's not the only puzzle: One of the most controversial associations with strep is the obsessive compulsive/tic disorder known as PANDAS (Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections). The condition is described as a combination of tics, obsessions, compulsions and other symptoms. While it has made headlines since researchers began studying it in the 1990s, there is still not a definitive connection between the disorder and strep.

"While there are a lot of hypotheses, the data to support those hypotheses is incomplete at best," Kaplan said. "There's been a lot published, but we don't know the true reason for this disease or what causes it."

Scientists do know that there are about 130 different types of Group A Strep. And while there's some evidence of a degree of resistance after exposure, strep isn't limited to a single type per season.

Worst case scenario: several people in your family or in your kid's classroom contract strep and recover, only to wake up a week or two later with another sore throat. Doctors call it the ping-pong effect.

"If it gets so bad, I've had the experience of parents banding together to say this has got to stop, and that's when the school will contact the health department," Kaplan said. "If it looks like an outbreak, they can decide what to do. For example, they could culture everybody in the room and treat those who are positive."

What can parents do?

To lessen the risk of exposure, remind kids to wash hands and cover their cough. Strep is spread by coughing and sneezing."

To ward off reinfection, replace toothbrushes when the patient is no longer contagious, but before finishing antibiotics. Same goes for washing pillowcases.

To help you or your child's stomach handle the antibiotics, current thinking in the alternative medicine world recommends a course of probiotics to counteract the destruction of good bacteria going on in your gut.

http://bit.ly/U9TnIZ

Protein's destructive journey in brain may cause Parkinson's

Clumps of alpha-synuclein move through dopamine-producing cells, mouse study finds

By Laura Sanders

The insidious spread of an abnormal protein may be behind Parkinson's disease, a study in mice suggests. A harmful version of the protein crawls through the brains of healthy mice, killing brain cells and damaging the animals' balance and coordination, researchers report in the Nov. 16 Science.

If a similar process happens in humans, the results could eventually point to ways to stop Parkinson's destruction in the brain. "I really think that this model will increase our ability to come up with Parkinson's disease therapies," says study coauthor Virginia Lee of the University of Pennsylvania Perelman School of Medicine in Philadelphia.

The new study targets a hallmark of Parkinson's disease — clumps of a protein called alpha-synuclein. The clumps, called Lewy bodies, pile up inside nerve cells in the brain and cause trouble, particularly in cells that make dopamine, a chemical messenger that helps control movement. Death of these dopamine-producing cells leads to the characteristic tremors and muscle rigidity seen in people with Parkinson's.

Lee and her team injected alpha-synuclein into the brains of healthy mice. After 30 days, the protein had spread to connected brain regions, suggesting that rouge alpha-synuclein moves from cell to cell, the scientists found. Months later, the spreading was even more extensive.

Alpha-synuclein appeared to colonize several areas of the otherwise healthy brain, but the protein was particularly prominent in nerve cells that make dopamine. After six months, Lewy bodies were found inside these cells. As a result, fewer cells survived, and the ones that did churned out less dopamine.

"The real thing here, the novelty, is that the aggregate form can spread from one brain region to another and one cell to another, and cause cell death and disease," says neuroscientist Patrik Brundin of Van Andel Research Institute in Grand Rapids, Mich.

Overall these animals didn't show big movement problems, but researchers did find subtle deficits six months after the alpha-synuclein injection. The mice were worse at balancing on a turning rod and couldn't cling to a wire cage for as long as mice that had been injected with saline.

Scientists don't know whether such cell-to-cell transmission happens in people, because it's impossible to do similar studies on humans. But some clues come from the brain of a woman with Parkinson's who received stem cell transplants in an effort to replenish her missing neurons. Fourteen years after the procedure, Lewy bodies were found in these previously healthy transplanted cells, raising the possibility that alpha-synuclein had spread there from the rest of the brain.

Neurologist and neuroscientist Ted Dawson of Johns Hopkins University School of Medicine cautions that it's still not clear that the alpha-synuclein spreading has to happen for the protein to cause damage. "I think transmission occurs," he says. The real question, he adds, is whether that transmission is important to the disease. "I don't think anyone's answered that in a convincing way."

If scientists could understand how alpha-synuclein travels from cell to cell in the brain, they could potentially stop the spread, and potentially with it, the disease. Antibodies that slurp up alpha-synuclein as it leaves a cell might prove beneficial, says Dawson.

http://www.bbc.co.uk/news/magazine-20324304

Is the toilet seat really the dirtiest place in the home?

The toilet seat has acquired an unfair reputation as the dirtiest item in the average household. But scientists say there are far filthier places in our house, some of them where we least expect.

By Charlotte Pritchard BBC News

Would you chop your vegetables on your toilet seat? I think pretty much all of us would say No. But maybe we should think again.

Dr Chuck Gerba, professor of microbiology at the University of Arizona, studies how diseases are transferred through the environment. This involves swabbing household items and measuring how many bacteria - and what sort - develop. He particularly looks for faecal bacteria such as E.coli and staphylococcus aureus.

His studies have found that on the average toilet seat there are 50 bacteria per square inch.

"It's one of the cleanest things you'll run across in terms of micro-organisms," he says. "It's our gold standard - there are not many things cleaner than a toilet seat when it comes to germs."

We should be more worried about other household items, it seems. "Usually there are about 200 times more faecal bacteria on the average cutting board than on a toilet seat," he says.

In the kitchen it doesn't necessarily get there through actual contact with faeces. It comes via raw meat products or the viscera from inside of the animal, where a lot of the faecal bacteria originate.

Would Gerba be more inclined to chop his vegetables on a toilet seat then? "It would seem a safer place," he says. "Not that I would recommend it, but you might treat your cutting board a bit more like you do your toilet seat." It's because we all fear the dirtiness of the toilet seat so much that we regularly clean it, so perhaps this is the course of action we need to take with our chopping boards.

But the filthiest culprit in our homes is the kitchen sponge or cloth. According to Gerba, there are about 10 million bacteria per square inch on a sponge, and a million on a dishcloth. In other words, a kitchen sponge is 200,000 times dirtier than a toilet seat, and a dishcloth is 20,000 times dirtier.

This is the same the world over. "Always the dirtiest thing by far is the kitchen sponge," says John Oxford, professor of virology at the University of London and chair of the Hygiene Council - an international body that compares hygiene standards across the world. Its latest study examines samples from homes in nine different countries, and finds that 21% of "visibly clean" kitchen cloths actually have high levels of contamination. The cloths also fail the bacterial test which looks for E.coli.

The study identifies faecal bacteria in other places around the home, and this varies from one country to another. Saudi Arabia has the dirtiest fridges, with 95% of the fridges in the study failing the bacteriology test for E.coli. And in South Africa, the dirtiest item is the seal in the bath, with almost two-thirds with unsatisfactory levels of E.coli and 40% for mould.

Household hygiene
2010 Hygiene in the Home Study tested 180 homes in Australia, Canada, Germany, India, Malaysia, Saudi Arabia, South Africa, UK and US
Bathroom seals caused most concern, with 70% failing bacterial tests
Fridge interiors came second - more than 40% of homes failed tests on bacteria and mould build-up
Kitchen towels were found to be unsatisfactory or unacceptably dirty in 36% of homes
Cleanest surface tested was pushchair with only 6% failing bacterial tests

"It's always a bit delicate which countries are the worst," says Oxford. "We found that countries like Australia and particularly Canada are high up on the hygiene list... Countries near the bottom are fairly routinely, unfortunately, India and Malaysia."

What about away from our homes? Gerba says the office is particularly bad. "Many people don't realise they're talking dirty every time they pick up their phone, because they never clean it. "The average desktop has 400 times more bacteria than on a toilet seat."

Beware the supermarket too. "Shopping trolleys are really bad," warns Gerba. What's more, about half of reusable shopping bags have faecal bacteria in them. "Some people have more faecal bacteria in their grocery bag than in their underwear, because they at least wash that."

So what does this actually mean for us in terms of health risks? "These numbers of bacteria, particularly for E.coli, are huge," says Oxford. "E.coli is an indicator bacterium. It may not itself cause horrible disease, but it indicates faeces is around and that might contain other organisms like salmonella and shigella which really are virulently pathogenic."

But we all touch these perhaps startlingly dirty things every day, and on the whole we don't get constantly ill. "We're jolly lucky that as we've evolved over two million years, we have a whole set of genes whose only function is to get the immune system in action," says Oxford.

"All of us, in all these countries we have gone to, rely on Lady Luck too much, keeping our fingers crossed or sitting on our hands. In a modern scientific society, what we want is people to realise there's a problem here and take action."

Disclaimer: Charlotte Pritchard and the BBC do not recommend chopping any sort of food on your toilet seat.

http://bit.ly/U9UxnS

Twitter shows language evolves in cities

WHERE do new words come from? On Twitter at least, they often begin life in cities with large African American populations before spreading more widely, according to a study of the language used on the social network.

17 November 2012 by Jim Giles

Jacob Eisenstein at the Georgia Institute of Technology in Atlanta and colleagues examined 30 million tweets sent from US locations between December 2009 and May 2011. Several new terms spread during this period, including "bruh", an alternative spelling of "bro" or "brother", which first arose in a few south-east cities before eventually hopping to parts of California. Residents of Cleveland, Ohio, were the first to use "ctfu", an abbreviation of "cracking the fuck up", usage that has since spread into Pennsylvania (arxiv.org/abs/1210.5268).

After collecting the data, the team built a mathematical model that captures the large-scale flow of new words between cities. The model revealed that cities with big African American populations tend to lead the way in linguistic innovation. The team is still working on a more detailed analysis and says it is too early to say which cities are the most influential.

Researchers have tracked the diffusion of words like "cool" and "uptight" from black communities to mainstream use in the past. "We have thousands of examples," says Eisenstein. Their data cannot shed light on why the flow is in this direction, but he notes that language is just one cultural area in which traditions have spread outwards from African American communities.
The team also found that cities that are economically and ethnically similar - rather than geographically close to one another - are more likely to share new words. "Their results indicate that birds of a feather tweet together," says John Nerbonne, a linguist at the University of Groningen in the Netherlands.
Eisenstein says he is looking into whether neologisms now spread more rapidly because of Twitter and other social networks. He is also interested in exploring whether social media is accelerating the evolution of language more generally, something that could be done by analysing everything from blog posts to Facebook entries. It's not like the old days, he says, when the spread of a word relied on people travelling to new areas.

http://www.japantimes.co.jp/text/nn20121118a8.html

Cesium in trout 110 times over limit

A trout caught in Fukushima Prefecture contained 11,400 becquerels of cesium per kilogram

Kyodo - A mountain trout caught in the Niida River in Fukushima Prefecture contained 11,400 becquerels of radioactive cesium per kilogram, more than 110 times above the government limit for food products, a survey by the Environment Ministry showed.
Presenting its findings Friday on cesium in fish and insects in rivers, lakes and sea in Fukushima, the ministry said it also detected 4,400 becquerels of radioactive cesium in a smallmouth bass and 3,000 becquerels in a catfish caught at the Mano Dam in Iitate.
The maximum threshold for food items is 100 becquerels per kilogram.
It is only the second time the ministry has conducted such a survey, after undertaking a study between December and this February. The first data were published in July.
"Like the previous survey, concentrations (of cesium) tended to be higher in rivers and lakes than in the sea. We want to grasp the extent of pollution by continuously conducting the survey," a ministry official said.

http://www.eurekalert.org/pub_releases/2012-11/niob-rbs111512.php

Research breakthrough selectively represses the immune system

NIH-funded scientists develop new treatment to combat autoimmune disease in mouse model

In a mouse model of multiple sclerosis (MS), researchers funded by the National Institutes of Health have developed innovative technology to selectively inhibit the part of the immune system responsible for attacking myelin—the insulating material that encases nerve fibers and facilitates electrical communication between brain cells.
Autoimmune disorders occur when T-cells—a type of white blood cell within the immune system—mistake the body's own tissues for a foreign substance and attack them. Current treatment for autoimmune disorders involves the use of immunosuppressant drugs which tamp down the overall activity of the immune system. However, these medications leave patients susceptible to infections and increase their risk of cancer as the immune system's normal ability to identify and destroy aberrant cells within the body is compromised.
Supported by the National Institute of Biomedical Imaging and Bioengineering (NIBIB) at NIH, Drs. Stephen Miller and Lonnie Shea at Northwestern University, Evanston, teamed up with researchers at the University of Sydney, and the Myelin Repair Foundation in Saratoga, Calif. to come up with a novel way of repressing only the part of the immune system that causes autoimmune disorders while leaving the rest of the system intact.
The new research takes advantage of a natural safeguard employed by the body to prevent autoreactive T-cells—which recognize and have the potential to attack the body's healthy tissues—from becoming active. They report their results in the Nov. 18 online edition of Nature Biotechnology.
"We're trying to do something that interfaces with the natural processes in the body," said Shea. "The body has natural mechanisms for shutting down an immune response that is inappropriate, and we're really just looking to tap into that."
One of these natural mechanisms involves the ongoing clearance of apoptotic, or dying, cells from the body. When a cell dies, it releases chemicals that attract specific cells of the immune system called macrophages. These macrophages gobble up the dying cell and deliver it to the spleen where it presents self-antigens—tiny portions of proteins from the dying cell—to a pool of T-cells. In order to prevent autoreactive T-cells from being activated, macrophages initiate the repression of any T-cells capable of binding to the self-antigens.
Dr. Miller was the first to demonstrate that by coupling a specific self-antigen such as myelin to apoptotic cells, one could tap into this natural mechanism to suppress T-cells that would normally attack the myelin. The lab spent decades demonstrating that they could generate antigen-specific immune suppression in various animal

models of autoimmune diseases. Recently, they initiated a preliminary clinical trial with collaborators in Germany to test the safety of injecting the antigen-bound apoptotic cells into patients with MS. While the trial successfully demonstrated that the injections were safe, it also highlighted a key problem with using cells as a vehicle for antigen delivery:

"Cellular therapy is extremely expensive as it needs to be carried out in a large medical center that has the capability to isolate patient's white blood cells under sterile conditions and to re-infuse those antigen-coupled cells back into the patients," said Miller. "It's a costly, difficult, and time-consuming procedure."

Thus began a collaboration with Dr. Shea, a bioengineer at Northwestern University, to discuss the possibility of developing a surrogate for the apoptotic cells. After trying out various formulations, his lab successfully linked the desired antigens to microscopic, biodegradable particles which they predicted would be taken up by circulating macrophages similar to apoptotic cells.

Much to their amazement, when tested by the Miller lab, the antigen-bound particles were just as good, if not better, at inducing T-cell tolerance in animal models of autoimmune disorders.

Using their myelin-bound particles, the researchers were able to both prevent the initiation of MS in their mouse model as well as inhibit its progression when injected immediately following the first sign of clinical symptoms.

The research team is now hoping to begin phase I clinical trials using this new technology. The material that makes up the particles has already been approved by the U.S. Food and Drug Administration and is currently used in resorbable sutures as well as in clinical trials to deliver anti-cancer agents. Miller believes that the proven safety record of these particles along with their ability to be easily produced using good manufacturing practices will make it easier to translate their discovery into clinical use.

"I think we've come up with a very potent way to induce tolerance that can be easily translated into clinical practice. We're doing everything we can now to take this forward," said Miller.

In addition to its potential use for the treatment of MS, the researchers have shown in the lab that their therapy can induce tolerance for other autoimmune diseases such as type I diabetes and specific food allergies. They also speculate that transplant patients could benefit from the treatment which has the potential to retract the body's natural immune response against a transplanted organ. Dr. Christine Kelley, NIBIB director of the Division of Science and Technology, points to the unique collaboration between scientists and engineers that made this advance a reality.

"This discovery is testimony to the importance of multidisciplinary research efforts in healthcare," said Kelley. "The combined expertise of these immunology and bioengineering researchers has resulted in a valuable new perspective on treating autoimmune disorders."

In addition to a grant from NIBIB (R01-EB013198-02), the research was also supported by NIH's National Institute of Neurological Disorders and Stroke (NS026543), the Myelin Repair Foundation, and the Juvenile Diabetes Research Foundation (17-2011-343).

http://www.bbc.co.uk/news/health-20365355

Nose cell transplant enables paralysed dogs to walk

Scientists have reversed paralysis in dogs after injecting them with cells grown from the lining of their nose.

The pets had all suffered spinal injuries which prevented them from using their back legs. The Cambridge University team is cautiously optimistic the technique could eventually have a role in the treatment of human patients. The study is the first to test the transplant in "real-life" injuries rather than laboratory animals.

In the study, funded by the Medical Research Council and published in the neurology journal Brain, the dogs had olfactory ensheathing cells from the lining of their nose removed. These were grown and expanded for several weeks in the laboratory.

Treadmill

Of 34 pet dogs on the proof of concept trial, 23 had the cells transplanted into the injury site - the rest were injected with a neutral fluid. Many of the dogs that received the transplant showed considerable improvement and were able to walk on a treadmill with the support of a harness. None of the control group regained use of its back legs. The research was a collaboration between the MRC's Regenerative Medicine Centre and Cambridge University's Veterinary School.

Professor Robin Franklin, a regeneration biologist at the Wellcome Trust-MRC Stem Cell Institute and report co-author, said: 'Our findings are extremely exciting because they show for the first time that transplanting these types of cell into a severely damaged spinal cord can bring about significant improvement.

"We're confident that the technique might be able to restore at least a small amount of movement in human patients with spinal cord injuries but that's a long way from saying they might be able to regain all lost function.

'

Prof Franklin said the procedure might be used alongside drug treatments to promote nerve fibre regeneration and bioengineering to substitute damaged neural networks.

Partial repair

The researchers say the transplanted cells regenerated nerve fibres across the damaged region of the spinal cord. This enabled the dogs to regain the use of their back legs and coordinate movement with their front limbs.

The new nerve connections did not occur over the long distances required to connect the brain to the spinal cord. The MRC scientists say in humans this would be vital for spinal injury patients who had lost sexual function and bowel and bladder control.

Olfactory ensheathing cells
The only part of the body where nerve fibres continue to grow in adults is the olfactory system.
Found in the at the back of the nasal cavity, olfactory ensheathing cells (OEC) surround the receptor neurons that both enable us to smell and convey these signals to the brain.
The nerve cells need constant replacement which is promoted by the OECs.
For decades scientists have thought OECs might be useful in spinal cord repair. Initial trials using OECs in humans have suggested the procedure is safe.

Prof Geoffrey Raisman, chair of Neural Regeneration at University College London, who discovered olfactory ensheathing cells in 1985 said: "This is not a cure for spinal cord injury in humans - that could still be a long way off. But this is the most encouraging advance for some years and is a significant step on the road towards it."

He said the clinical benefits were still limited: "This procedure has enabled an injured dog to step with its hind legs, but the much harder range of higher functions lost in spinal cord injury - hand function, bladder function, temperature regulation, for example - are yet more complicated and still a long way away."

Jasper, a 10-year-old dachshund, is one of the dogs which took part in the trial.

His owner May Hay told me: "Before the treatment we used to have to wheel Jasper round on a trolley because his back legs were useless. Now he whizzes around the house and garden and is able to keep up with the other dogs. It's wonderful." Jasper can be seen in the ***video at the top of the page*** before and after his treatment.

http://www.eurekalert.org/pub_releases/2012-11/w-isd111412.php

Inpatient sleeping drug quadrupled fall risk

A drug commonly prescribed to help patients sleep in hospitals has been associated with an increased risk of falls, according to a study published in the Journal of Hospital Medicine.

U.S. sleep specialists from the Mayo Clinic found that the fall rate among the 4,962 patients who took zolpidem during their hospital stay was more than four times as high as the 11,358 who did not take the drug.

They also found that the risk posed by the drug was greater than the risks posed by factors such as age, cognitive impairment, delirium or insomnia, regardless of the dosage used.

"Ensuring that people get enough sleep during their hospital stay is very important, but it can also prove very challenging," says the Clinic's Chief Patient Safety Officer Dr. Timothy I. Morgenthaler, who specializes in sleep disorders and pulmonary and critical care.

"Patient falls are also a significant patient safety issue in hospitals and one that has been quite difficult to tackle, despite considerable efforts. That is why it is one of the target aims of the U.S. Department of Health and Human Services Partnership for Patients project."

"Discovering that zolpidem, which is commonly used in hospitals, is a significant risk factor for patient falls provides us with additional knowledge to help tackle this problem."

Key findings of the study include:

Just under 39 percent of eligible admissions during 2010 were prescribed zolpidem (16,320 patients) but 88 percent of the prescriptions were issued on an "as needed basis."

Zolpidem was administered to 30.4 percent of patients who were prescribed it and to 11.8 percent of all Mayo Clinic admissions in 2010.

Just over three percent of the patients on zolpidem fell during their in-patient hospital stay, compared with 0.7 percent of the patients who did not take zolpidem.

Zolipdem use continued to be associated with an increased fall risk when other key factors, including health, length of hospital stay and assessed fall risk, were taken into consideration.

"Our hospitals have an overall fall rate of about 2.5 per 1000 patient days, which is lower than many national benchmarks. However, we have not been able to significantly reduce this rate in recent years. Now, we calculate that for every 55 patients who received zolpidem, there was one additional fall that may have been avoided by not administering the drug," says Dr. Morgenthaler.

"As a result of our study, we are now phasing out zolpidem and moving toward sleep enhancement techniques that are not based on drugs and which we believe are safer and probably as effective."

http://www.eurekalert.org/pub_releases/2012-11/tu-dkf111312.php

Decreased kidney function leads to decreased cognitive functioning

Decreased kidney function is associated with decreased cognitive functioning in areas such as global cognitive ability, abstract reasoning and verbal memory

Decreased kidney function is associated with decreased cognitive functioning in areas such as global cognitive ability, abstract reasoning and verbal memory, according to a study led by Temple University. This is the first study describing change in multiple domains of cognitive functioning in order to determine which specific abilities are most affected in individuals with impaired renal function.

Researchers from Temple, University of Maine and University of Maryland examined longitudinal data, five years apart, from 590 people. They wanted to see how much kidney function had changed over that time period, and whether it was associated with how much cognitive functioning had changed. They were interested in the overall change, but also in specific abilities such as abstract reasoning and verbal memory.

"The brain and kidney are both organs that are affected by the cardiovascular systems," said the study's lead author, Adam Davey, associate professor of public health in Temple's College of Health Professions and Social Work. "They are both affected by things like blood pressure and hypertension, so it is natural to expect that changes in one organ are going to be linked with changes in another."

What the researchers found was the greater a person's decrease in renal functioning, the greater the decrease in overall cognitive functioning, particularly abstract reasoning and verbal memory.

"Those two tracked together, so this study provides us with evidence that the rate of cognitive decline is associated with deterioration in kidney function" said Davey.

Davey said that this information emphasizes two important points: the importance of diagnosing and managing chronic kidney disease and the extent of decrease in cognitive functioning.

"As we get older, our kidney function tends to decrease naturally, so if there's an extra issue involved in renal function like chronic kidney disease, we need to know about it as soon as possible," he said. "That is something that needs to be managed, just like you would manage hypertension."

Davey also noted that the decrease in cognitive functioning found in the study—when compared to people with dementia or cognitive impairment—is not so great that it would interfere with people being able to assist in their treatment of kidney disease.

"Patients are still going to be able to take their medicine on time and without assistance, as well as understand the information that their physician is sharing with them about their disease," he said.

In addition to Davey, researchers in this study included Merrill Elias, Michael Robbins and Gregory Dore of the University of Maine's Department of Psychology and School of Biomedical Sciences and Stephen L. Seliger of the University of Maryland's School of Medicine.

The researchers published their findings, "Decline in Renal Functioning is Associated with Longitudinal Decline in Global Cognitive Functioning, Abstract Reasoning, and Verbal Memory," in the journal Nephrology, Dialysis and Transplantation.

The study was funded by grants from the National Heart, Lung and Blood Institute and the National Institute on Aging at the National Institutes of Health to Temple University and the University of Maine.

http://bit.ly/U7xoAw

Fraud fighter: 'Faked research is endemic in China'

Shi-min Fang tells us how risking his life and libel writs to expose scientific misconduct in his native China has just won him the inaugural Maddox prize

19 November 2012 by Jon White

You've just won the inaugural Maddox prize, awarded for your continuing work exposing scientific misconduct in China despite the threats you face. How does that feel?

I am thrilled and honoured. There are many people who are supporting me and fighting with me, so I consider this award as an acknowledgement of all our efforts, not just mine.

What prompted you to start challenging dubious pseudoscientific claims in China?

In 1998, after eight years studying in the US, I returned to China and was shocked to see it was deluged with pseudosciences, superstitions and scientific misconduct.

What action did you decide to take?

I had created a Chinese website called New Threads in 1994 when I was a graduate student at Michigan State University as a forum for sharing Chinese classics and literature. From 2000, I started to publish articles on the site fighting scientific misconduct and fraud. Eventually, New Threads became a flagship for those fighting pseudoscience, misconduct, fraud and corruption among the Chinese science community.

Are dubious claims a big problem in China?

The majority of cases exposed are plagiarism, the exaggeration of academic credentials and faked research papers, which are endemic in China.

Tell me about some of them.

A typical case was the nucleic acid "nutrition" scheme - supplements promoted to boost energy levels in the tired, pregnant and old. It involved more than a dozen Chinese biochemists and was the first that brought wide media coverage, both domestically and internationally. New Threads has exposed more than 1000 cases of scientific fraud.

Why is science fraud such a problem in China?

It is the result of interactions between totalitarianism, the lack of freedom of speech, press and academic research, extreme capitalism that tries to commercialise everything including science and education, traditional culture, the lack of scientific spirit, the culture of saving face and so on. It's also because there is not a credible official channel to report, investigate and punish academic misconduct. The cheaters don't have to worry they will someday be caught and punished.

What have been the worst moments?

I have been sued more than 10 times. Because the Chinese legal system is very corrupt and a ruling is not always made according to the evidence, it is not surprising that I have lost some libel cases even though I did nothing wrong. In one of these, a local court at Wuhan ordered me to pay 40,000 yuan in compensation and transferred the money from my wife's account. I have also narrowly escaped from an attack with pepper spray and a hammer.

Has it been worth it?

Yes. I fully understand the risk I am facing and am willing to take it. What troubles me most is that my wife and my young daughter also have to endure vituperation and personal attacks.

Profile

Shi-min Fang has held research posts at the University of Rochester, New York, and the Salk Institute for Biological Studies in La Jolla, California. He is now a freelance science writer