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Statins and the Kidney: Good at All Stages

Statin use clearly demonstrated to reduce risk for cardiovascular events and mortality in chronic kidney disease patients (video)

Bret Stetka, MD, Enyu Imai

Hello. I'm Bret Stetka, Editorial Director at Medscape. Welcome to the F1000 Practice-Changing Minute, where we report commentaries from the Faculty of 1000 on highly rated studies that may change clinical practice.

Our commentary today covers the study "Effect of Statin Therapy on Cardiovascular and Renal Outcomes in Patients With Chronic Kidney Disease " from Drs. Hou and colleagues and published in the European Heart Journal.[1] The F1000 commentator has given this study a ranking of "Changes Clinical Practice," with the conclusion that statins should be used in all stages of chronic kidney disease (CKD), including stage 5.

The following F1000 commentary on this study was written by Enyu Imai, Associate Professor, Department of Nephrology, Nagoya University Graduate School of Medicine, Aichi, Japan.

"Hou and colleagues clearly demonstrate here that statin use reduces the risk for cardiovascular events and mortality in chronic kidney disease (CKD) patients. It had not been clear whether statins reduce risk for cardiovascular disease and mortality in stage 5 CKD including dialysis. In the present study, the absolute risk reduction and number needed to treat (NNT) were slightly lower in patients with advanced CKD compared with those with CKD stage 3 and 4. They also showed that the adverse effects of statins were not increased in the CKD population. Overall, statins confer important benefits in CKD patients. However, the beneficial effects of statins on renal outcome were not clearly demonstrated. Further study will be required on this issue.

It has been controversial to use statins in patients with stage 5 CKD. However, this meta-analysis clearly shows that statins provide beneficial effects on patients with all stages of CKD, including dialysis patients, without apparent adverse events."

This concludes today's commentary from Enyu Imai for the F1000 Practice-Changing Minute. I am Bret Stetka. Thank you for listening.

Hou W, Lv J, Perkovic V, Yang L, et al. Effect of statin therapy on cardiovascular and renal outcomes in patients with chronic kidney disease: a systematic review and meta-analysis. Eur Heart J. 2013;34:1807-1817.

http://www.eurekalert.org/pub_releases/2013-08/du-tbg073113.php

The brain's GPS: Researchers discover human neurons linked to navigation in open environments

New type of cell identified in the brain that helps people to keep track of their relative location while navigating an unfamiliar environment

Using direct human brain recordings, a research team from Drexel University, the University of Pennsylvania, UCLA and Thomas Jefferson University has identified a new type of cell in the brain that helps people to keep track of their relative location while navigating an unfamiliar environment.

The "grid cell," which derives its name from the triangular grid pattern in which the cell activates during navigation, is distinct among brain cells because its activation represents multiple spatial locations. This behavior is how grid cells allow the brain to keep track of navigational cues such as how far you are from a starting point or your last turn. This type of navigation is called path integration.

"It is critical that this grid pattern is so consistent because it shows how people can keep track of their location even in new environments with inconsistent layouts," said Dr. Joshua Jacobs, an assistant professor in Drexel's School of Biomedical Engineering, Science and Health Systems, who is the team's primary investigator.

The researchers, Jacobs, Dr. Michael Kahana, from Penn, and UCLA's Dr. Itzhak Fried were able to discern these cells because they had the rare opportunity to study brain recordings of epilepsy patients with electrodes implanted deep inside their brains as part of their treatment. Their work is being published in the latest edition of Nature Neuroscience.

During brain recording, the 14 study participants played a video game that challenged them to navigate from one point to another to retrieve objects and then recall how to get back to the places where each object was located. The participants used a joystick to ride a virtual bicycle across a wide-open terrain displayed on a laptop by their hospital beds. After participants made trial runs where each of the objects was visible in the distance, they were put back at the center of the map and the objects were made invisible until the bicycle was right in front of them. The researchers then asked the participants to travel to particular objects in different sequences.

The team studied the relation between how the participants navigated in the video game and the activity of individual neurons.

"Each grid cell responds at multiple spatial locations that are arranged in the shape of a grid," Jacobs said. "This triangular grid pattern thus appears to be a brain pattern that plays a fundamental role in navigation. Without grid cells, it is likely that humans would frequently get lost or have to navigate based only on landmarks. Grid cells are thus critical for maintaining a sense of location in an environment."

While these cells are not unique among animals — they have been discovered previously in rats — and a prior study in 2010, that used noninvasive brain imaging, suggested the existence of the cells in humans, this is the first positive identification of the human version of these cells.

"The present finding of grid cells in the human brain, together with the earlier discovery of human hippocampal 'place cells,' which fire at single locations, provide compelling evidence for a common mapping and navigational system preserved across humans and lower animals," said Kahana, a neuroscientist who is a senior author and professor of psychology at the University of Pennsylvania.

The team's findings also suggest that these grid patterns may in fact be more prevalent in humans than rats, because the study found grid cells not only in the entorhinal cortex — where they are observed in rats — but also, in a very different brain area — the cingulate cortex.

"Grid cells are found in a critical location in the human memory system called the entorhinal cortex," said Fried, who is a professor of neurosurgery at the David Geffen School of Medicine at UCLA. "This discovery sheds new light on a region of the brain that is the first to be affected in Alzheimer's Disease with devastating effects on memory"

The entorhinal cortex is part of the brain that has been studied in Alzheimer's disease research and according to Jacobs, understanding grid cells could help researchers understand why people with the disease often become disoriented. It could also help them show how to improve brain function in people suffering from Alzheimer's.

http://bit.ly/16lTTrj

More girls born in Japan after quake skews sex ratio

HERE come the girls. Fewer boys than girls were born in the months after the huge earthquake struck Japan in March 2011.

04 August 2013 by Sara Reardon

Ralph Catalano of the University of California in Berkeley and colleagues examined hospital records of births in Japan between 2006 and the end of 2011. After the quake, births in areas closest to the epicentre were more likely to be girls, but provinces farther out showed no gender bias. About 2.2 per cent fewer boys were born in the most damaged areas than expected (American Journal of Human Biology, doi.org/nbj).

It is not the first time such a skew has been noted: fewer boys were born after the US stock market crash of 2008, for instance. The reason may be evolutionary, says Catalano. Boys are more likely to be premature and suffer problems associated with low birth weight than girls. In times of stress, it may therefore be beneficial for the mother to give birth to a girl.

However, it is unclear whether stress causes mothers to miscarry more males or whether fewer males are conceived. Earthquakes provide a natural test for this, Catalano says. If births immediately after show a gender skew, it would suggest that stress is triggering miscarriages. A bias nine months later indicates fewer male conceptions.

The team found evidence for both. Several mechanisms may be at work, says Catalano. Fetuses produce a hormone called human chorionic gonadotropin, which camouflages them from the mother's immune system. Weak male fetuses make less of this hormone, meaning they may be at greater risk of attack.

William James of University College London believes the father's testosterone level plays a role. During periods of high stress, he says, men produce less testosterone, which can reduce the number and quality of the "male" sperm, which carry a Y sex chromosome.

http://www.eurekalert.org/pub_releases/2013-08/sjcr-mop080213.php

Mechanism offers promising new approach for harnessing the immune system to fight cancer

St. Jude Children's Research Hospital researchers discover how to unleash the immune system against cancer in mice without triggering autoimmune reactions

St. Jude Children's Research Hospital scientists have discovered a way to target the immune system to shrink or eliminate tumors in mice without causing autoimmune problems. Researchers also found evidence that the same mechanism may operate in humans. The study was published today in the advance online edition of Nature.

The findings provide a new target for ongoing efforts to develop immunotherapies to harness the immune system to fight cancer and other diseases.

The work focused on white blood cells called regulatory T cells. These specialized cells serve as the immune system's police force, working to control inflammation and guard against autoimmune and inflammatory disease. Regulatory T cells can, however, interfere with the immune system's ability to fight cancer.
In this study, investigators identified a mechanism that boosts the ability of regulatory T cells to cause problems by blocking an effective anti-tumor immune response. The same process, however, plays no role in maintaining immune balance or preventing the misguided immune attack on healthy tissue that leads to autoimmune problems, researchers reported. Blocking this mechanism led to the elimination or dramatic reduction of melanoma by the immune system in mice, without causing the autoimmune and inflammatory problems often associated with current cancer-treatment efforts that target immune regulators, scientists said.
"Regulatory T cells are a major barrier to effective anti-tumor immunity," said the study's corresponding author, Dario Vignali, Ph.D., vice chair of the St. Jude Department of Immunology. "We have identified a mechanism that enhances the ability of regulatory T cells to put the brakes on the immune response in tumors but plays no role in immune system maintenance. For the first time, we may now have an opportunity to selectively target the activity of regulatory T cells for treatment of cancer without inducing autoimmune or inflammatory complications."
The mechanism is built around two proteins. One, semaphorin-4a (Sema4a), is carried on the surface of various immune cells that can spark inflammation. The other, neuropilin-1 (Nrp1), is carried on the surface of regulatory T cells.
Vignali and his colleagues used a variety of molecular and cellular techniques to show that Sema4a binding to Nrp1 turns on a biochemical pathway in mouse regulatory T cells that enhances their function, stability and survival. When scientists eliminated Nrp1 on just regulatory T cells, those cells were unable to respond to signals that normally bolstered their anti-inflammatory activity.
When investigators analyzed human regulatory T cells, they found evidence that the pathway may also serve the same role.
In addition, more than 16 months after losing Nrp1 activity in their regulatory T cells, the mice showed no signs of autoimmune or inflammatory complications. "That is significant because mice and humans that lack or have substantial defects in regulatory T cells develop lethal autoimmune disease," Vignali said.
Knocking out or blocking the activity of Nrp1 on regulatory T cells in mouse models of several human cancers, including the deadly skin cancer melanoma, led to reduced, delayed or complete elimination of the tumors. Blocking Sema4a had a similar anti-tumor effect, researchers reported. "The impact was particularly dramatic in a mouse model of human melanoma," Vignali said. "Mice lacking Nrp1 on regulatory T cells were almost completely resistant to developing melanoma, but did not develop any autoimmune or inflammatory complications."
Although investigators have not yet identified which cells carry Sema4a in tumors and boost regulatory T cell function, the scientists did report that immune cells called plasmacytoid dendritic cells (pDCs) provided more than half of the Sema4a in tumors in this study. That was surprising because pDCs make up a very small percentage of immune cells, and there is a long history of suppressive interactions between regulatory T cells and pDCs in tumors, Vignali said. Both cell types are recognized as inducing the immune system to tolerate, rather than attack, tumors.
Researchers also provided new details of how the Nrp1 pathway functions, including evidence that along with bolstering the ability of regulatory T cells to suppress the immune response, the pathway also helps maintain a stable population of regulatory T cells. "This pathway does not just boost regulatory function. It may define how regulatory T cells maintain their identity," said Greg Delgoffe, Ph.D., a postdoctoral fellow in Vignali's laboratory. Delgoffe and Seng-Ryong Woo, Ph.D., a former postdoctoral fellow in Vignali's laboratory, are co-first authors.
The other authors are Meghan Turnis, Cliff Guy, Abigail Overacre, Matthew Bettini, Peter Vogel, David Finkelstein and Creg Workman, all of St. Jude; David Gravano, formerly of St. Jude; and Jody Bonnevier, R&D Systems, Inc., Minneapolis.
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http://www.eurekalert.org/pub_releases/2013-08/nlmc-srp080213.php

Study reveals potential role of 'love hormone' oxytocin in brain function

Findings of NYU Langone researchers may have relevance in autism-spectrum disorder

In a loud, crowded restaurant, having the ability to focus on the people and conversation at your own table is critical. Nerve cells in the brain face similar challenges in separating wanted messages from background chatter. A key element in this process appears to be oxytocin, typically known as the “love hormone” for its role in promoting social and parental bonding.

In a study appearing online August 4 in Nature, NYU Langone Medical Center researchers decipher how oxytocin, acting as a neurohormone in the brain, not only reduces background noise, but more importantly, increases the strength of desired signals. These findings may be relevant to autism, which affects one in 88 children in the United States.

"Oxytocin has a remarkable effect on the passage of information through the brain," says Richard W. Tsien, DPhil, the Druckenmiller Professor of Neuroscience and director of the Neuroscience Institute at NYU Langone Medical Center. "It not only quiets background activity, but also increases the accuracy of stimulated impulse firing. Our experiments show how the activity of brain circuits can be sharpened, and hint at how this re-tuning of brain circuits might go awry in conditions like autism."

Children and adults with autism-spectrum disorder (ASD) struggle with recognizing the emotions of others and are easily distracted by extraneous features of their environment. Previous studies have shown that children with autism have lower levels of oxytocin, and mutations in the oxytocin receptor gene predispose people to autism. Recent brain recordings from people with ASD show impairments in the transmission of even simple sensory signals.

The current study built upon 30-year old results from researchers in Geneva, who showed that oxytocin acted in the hippocampus, a region of the brain involved in memory and cognition. The hormone stimulated nerve cells – called inhibitory interneurons – to release a chemical called GABA. This substance dampens the activity of the adjoining excitatory nerve cells, known as pyramidal cells.

"From the previous findings, we predicted that oxytocin would dampen brain circuits in all ways, quieting both background noise and wanted signals," Dr. Tsien explains. "Instead, we found that oxytocin increased the reliability of stimulated impulses – good for brain function, but quite unexpected."

To resolve this paradox, Dr. Tsien and his Stanford graduate student Scott Owen collaborated with Gord Fishell, PhD, the Julius Raynes Professor of Neuroscience and Physiology at NYU Langone Medical Center, and NYU graduate student Sebnem Tuncdemir. They identified the particular type of inhibitory interneurons responsible for the effects of oxytocin: "fast-spiking" inhibitory interneurons.

The mystery of how oxytocin drives these fast-spiking inhibitory cells to fire, yet also increases signaling to pyramidal neurons, was solved through studies with rodent models. The researchers found that continually activating the fast-spiking inhibitory neurons – good for lowering background noise – also causes their GABA-releasing synapses to fatigue. Accordingly, when a stimulus arrives, the tired synapses release less GABA and excitation of the pyramidal neuron is not dampened as much, so that excitation drives the pyramidal neuron's firing more reliably.

"The stronger signal and muffled background noise arise from the same fundamental action of oxytocin and give two benefits for the price of one," Dr. Fishell explains. "It's too early to say how the lack of oxytocin signaling is involved in the wide diversity of autism-spectrum disorders, and the jury is still out about its possible therapeutic effects. But it is encouraging to find that a naturally occurring neurohormone can enhance brain circuits by dialing up wanted signals while quieting background noise."

Funding for this work was provided by the Burnett Family Fund, the Mosbacher Fund, the Mathers Foundation, the National Institute of Mental Health (MH071739), the National Institute of Neurological Disorders and Stroke (NS024067), and the Simons Foundation.

http://globalnews.ca/news/764548/study-finds-that-colour-of-light-may-affect-your-mood/

Study finds that colour of light may affect your mood

People who work night shifts, like doctors and nurses, may benefit from red light rather than white or blue light.

By Nicole Mortillaro Global News

People who work night shifts, like doctors and nurses, may benefit from red light rather than white or blue light.

Feeling blue has taken on a whole new meaning.

A recent study on hamsters found that blue light has the worst effect on mood, followed by white light. However, red light was the least disruptive; the hamsters exhibited less depressive-like symptoms.

The findings are significant to humans, particularly for those working night shifts, as they are most exposed to white light at night.

"Our findings suggest that if we could use red light when appropriate for night-shift workers, it may not have some of the negative effects on their health that white light does," co-author of the study, Randy Nelson said.

The research studied the instrinsically photosensitive retinal ganglian cells (ipRGCs), cells in the eyes, which don't play a part in vision, but instead detect light and send messages to the part of the brain that regulates our circadian rhythm. Our circadian rhythm is what helps tell our body when to stay awake and when to sleep.

"Light at night may result in parts of the brain regulating mood-receiving signals during times of the day when they shouldn't," said co-author Tracy Bedrosian, a former graduate student at Ohio State who is now a postdoctoral researcher at the Salk Institute. "This may be why light at night seems to be linked to depression in some people."
The researchers were able to determine depressive behaviour in the hamsters by monitoring how often they drank sugar water – something they enjoy – as well as examining the hippocampus region of their brains. Hamsters that spent a night in dim blue or white light had a reduced density of dendritic spines – hairlike growths on brain cells that are used to send chemical messages to each other. A lowered density has been linked to depression, Nelson said.
The study appears in the Aug. 7, 2013 issue of The Journal of Neuroscience.

http://bit.ly/13Xvm94

Alzheimer Disease and Parkinson Disease Do Not Appear To Share Common Genetic Risk

Examining the genetic overlap between Parkinsons disease and Alzheimers

Newswise — A study by Valentina Moskvina, Ph.D., of the Cardiff University School of Medicine, Wales, United Kingdom, and colleagues, examined the genetic overlap between Parkinson disease (PD) and Alzheimer disease (AD).
Data sets from the United Kingdom, Germany, France and the United States were used to perform a combined genome-wide association analysis (GWA). The GWA study of AD included 3,177 patients with AD and 7,277 control patients, and the GWA analysis for PD included 5,333 patients with PD and 12,298 control patients.
The gene-based analyses resulted in no significant evidence that supported the presence of loci (location of gene) that were associated with increased risk for both PD and AD, according to the study results.
"Our findings therefore imply that loci that increase the risk of both PD and AD are not widespread and that the pathological overlap could instead be 'downstream' of the primary susceptibility genes that increase the risk of each disease," the study concludes.
(JAMA Neurol. Published online August 5, 2013. doi:10.1001/.jamaneurol.2013.448. Available pre-embargo to the media at http://media.jamanetwork.com.)
This study was supported by Parkinson's United Kingdom, the Medical Research Council, and numerous other funding sources

http://www.bbc.co.uk/news/health-23534101

Tiny adrenal tumours 'cause high blood pressure'

Treating tiny benign tumours in the adrenal glands may prevent huge numbers of cases of high blood pressure, say researchers.

By James Gallagher Health and science reporter, BBC News

The team at the University of Cambridge and Addenbrooke's Hospital think that up to 10% of cases may be caused by the growths. Their study, published in Nature Genetics, said young patients could be freed from a lifetime of medication. The British Heart Foundation said it was an "exciting development".
High blood pressure can have fatal consequences as it increases the risk of heart attacks and stroke.
Most cases are caused by lifestyle choices such as smoking and diet.
Researchers had already found that large growths in the adrenal glands, which sit on the kidneys and produce hormones, could raise blood pressure. An operation to remove any tumours can reduce blood pressure.
Now the researchers in Cambridge have found that much smaller growths, in a different part of the glands, are producing the same effect.

Hormonal

Both increase the amount of aldosterone made in an adrenal gland. Release of this hormone regulates the kidneys to retain more salt in the body, so increasing blood pressure.
Around 5% of high blood pressure cases are down to the large growths, but the researchers argue that the discovery of the small tumours means far more people have preventable blood pressure.
Prof Morris Brown told the BBC: "We think it could be twice that amount. My guess is around 10%, so it could be as many as one million people [in the UK]."
Living with high blood pressure for too long causes changes in the heart and arteries that mean that operations later in life may not reverse the condition. "We can't go looking for the 50 and 60-years-olds, we've missed the boat. We should go looking for the 30-year-olds," said Prof Brown.
Prof Jeremy Pearson, of the British Heart Foundation, said: "It is an exciting development, as this group of patients can be completely cured of high blood pressure once they have been identified, so the quicker they are diagnosed the better."

http://www.eurekalert.org/pub_releases/2013-08/afot-eab080513.php

Eating a big breakfast fights obesity and disease

A high-calorie breakfast protects against diabetes, hypertension, and cardiovascular problems, says Tel Aviv University researcher

Whether you hope to lose weight or just stay healthy, what you eat is a crucial factor. The right nutrients can not only trim your waistline, but also provide energy, improve your mood, and stave off disease. Now a Tel Aviv University researcher has found that it's not just what you eat — but when.

Metabolism is impacted by the body's circadian rhythm – the biological process that the body follows over a 24 hour cycle. So the time of day we eat can have a big impact on the way our bodies process food, says Prof. Daniela Jakubowicz of TAU's Sackler Faculty of Medicine and the Diabetes Unit at Wolfson Medical Center. In a recent study, she discovered that those who eat their largest daily meal at breakfast are far more likely to lose weight and waist line circumference than those who eat a large dinner.

And the benefits went far beyond pounds and inches. Participants who ate a larger breakfast – which included a dessert item such as a piece of chocolate cake or a cookie – also had significantly lower levels of insulin, glucose, and triglycerides throughout the day, translating into a lower risk of cardiovascular disease, diabetes, hypertension, and high cholesterol. These results, published recently in the journal Obesity, indicate that proper meal timing can make an important contribution towards managing obesity and promoting an overall healthy lifestyle. The study was done in collaboration with Dr. Julio Wainstein of TAU and the Wolfson Medical Center and Dr. Maayan Barnea and Prof. Oren Froy at the Hebrew University of Jerusalem.

A dramatic difference

To determine the impact of meal timing on weight loss and health, Prof. Jakubowicz and her fellow researchers conducted a study in which 93 obese women were randomly assigned to one of two isocaloric groups. Each consumed a moderate-carbohydrate, moderate-fat diet totalling 1,400 calories daily for a period of 12 weeks. The first group consumed 700 calories at breakfast, 500 at lunch, and 200 at dinner. The second group ate a 200 calorie breakfast, 500 calorie lunch, and 700 calorie dinner. The 700 calorie breakfast and dinner included the same foods.

By the end of the study, participants in the "big breakfast" group had lost an average of 17.8 pounds each and three inches off their waist line, compared to a 7.3 pound and 1.4 inch loss for participants in the "big dinner" group. According to Prof. Jakubowicz, those in the big breakfast group were found to have significantly lower levels of the hunger-regulating hormone ghrelin, an indication that they were more satiated and had less desire for snacking later in the day than their counterparts in the big dinner group.

The big breakfast group also showed a more significant decrease in insulin, glucose, and triglyceride levels than those in the big dinner group. More important, they did not experience the high spikes in blood glucose levels that typically occur after a meal. Peaks in blood sugar levels are considered even more harmful than sustained high blood glucose levels, leading to high blood pressure and greater strain on the heart.

Eliminating late night snacking

These findings suggest that people should adopt a well thought-out meal schedule, in addition to proper nutrition and exercise, to optimize weight loss and general health. Eating the right foods at the wrong times can not only slow down weight loss, it can also be harmful. In their study, the researchers found that those in the big dinner group actually increased their levels of triglycerides - a type of fat found in the body - despite their weight loss, reports Prof. Jakubowicz.

Prof. Jakubowicz suggests an end to late night snacking. Mindless eating in front of the computer or television, especially in the late evening hours, is a huge contributor to the obesity epidemic, she believes. It increases not only poundage, but the risk of cardiovascular disease - making that midnight sugar rush more costly than it appears.

http://www.eurekalert.org/pub_releases/2013-08/uosc-sc080513.php

Study: Centers throughout the brain work together to make reading possible

A combination of brain scans and reading tests has revealed that several regions in the brain are responsible for allowing humans to read.

The findings open up the possibility that individuals who have difficulty reading may only need additional training for specific parts of the brain – targeted therapies that could more directly address their individual weaknesses.

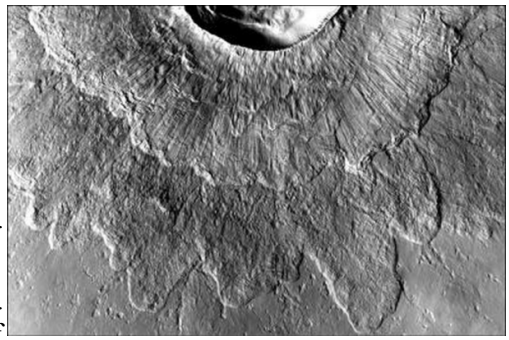
"Reading is a complex task. No single part of the brain can do all the work," said Qinghua He, postdoctoral research associate at the USC Brain and Creativity Institute and the first author of a study on this research that was published in the Journal of Neuroscience on July 31.

The study looked at the correlation between reading ability and brain structure revealed by high-resolution magnetic resonance imaging (MRI) scans of more than 200 participants.
To control for external factors, all of the participants were about the same age and education level (college students); right-handed (lefties use the opposite hemisphere of their brain for reading); and all had about the same language skills (Chinese-speaking, with English as a second language for more than nine years). Their IQ, response speed, and memory were also tested.
The study first collected data for seven different reading tests of a sample over 400 participants. These tests were aimed to explore three aspects of their reading ability:
phonological decoding ability (the ability to sound out printed words);
form-sound association (how well participants could make connections between a new word and sound);
and naming speed (how quickly participants were able to read out loud).
Each of these aspects, it turned out, was related to the gray matter volume – the amount of neurons – in different parts of the brain. The MRI analysis showed that phonological decoding ability was strongly connected with gray matter volume in the left superior parietal lobe (around the top/rear of the brain); form-sound association was strongly connected with the hippocampus and cerebellum; and naming speed lit up a variety of locations around the brain.
"Our results strongly suggest that reading consists of unique capacities and is supported by distinct neural systems that are relatively independent of general cognitive abilities," said Gui Xue, corresponding author of the study. Xue was formerly a research assistant professor of USC, and now is a professor and director of the Center for Brain and Learning Sciences at Beijing Normal University.
"Although there is no doubt that reading has to build up existing neural systems due to the short history of written language in human evolution, years of reading experiences might have finely tuned the system to accommodate the specific requirement of a given written system," Xue said.
He and Xue collaborated with Chunhui Chen, and Qi Dong of Beijing Normal University; Chuansheng Chen, of the University of California, Irvine; and Zhong-Lin Lu of Ohio State University.
One of the outstanding features of this study is its unusually wide sample size. Typically, MRI studies test a relatively small sample of individuals – perhaps around 20 to 30 – because of the high cost of using the MRI machine. Testing a single individual can cost about $500, depending on the nature of the research.
The team had the good fortune of receiving access to Beijing Normal University's new MRI center – BNU Imaging Center for Brain Research – just before it opened to the public. With support from several grants, they were able to conduct MRI tests on 233 individuals.
Next, the group will explore how to combine data from other factors, such as white matter, resting and task functional MRI and more powerful machine learning techniques to improve the accuracy of individuals' reading abilities. "Research along this line will enable the early diagnosis of reading difficulties and the development of more targeted therapies." said Xue.
This research was funded by the 111 Project of China (B07008), the National Science Foundation of China (31130025), the Foundation for the Authors of National Excellent Doctoral Dissertations of China (201108), and the National Institute of Health (HD057884-01A2).

http://www.eurekalert.org/pub_releases/2013-08/bu-omc080513.php

Odd Martian crater type made by impacts into ancient ice

Geologists from Brown University have developed a promising new explanation for a mysterious type of crater on the surface on Mars.



PROVIDENCE, R.I. [Brown University] — Double-layered ejecta craters or DLEs, like other craters, are surrounded by debris excavated by an impactor. What makes DLEs different is that the debris forms two distinct layers — a large outer layer with a smaller inner layer sitting on top.
These distinctive craters were first documented in data returned from the Viking missions to Mars in the 1970s, and scientists have been trying ever since to figure out how the double-layer pattern forms.
A new study by Brown graduate student David Kutai Weiss and James W. Head, professor of geological science, suggests that DLEs are the result of impacts onto a surface that was covered by a layer of glacial ice tens of meters thick.

Double-layer ejecta craters could form when ejected material slides down steep crater walls and across ice, forming a top layer. Striations, common in landslides on Earth, radiate out from the crater rim. **NASA**

"Recent discoveries by planetary geoscientists at Brown and elsewhere have shown that the climate of Mars has varied in the past," Head said. "During these times, ice from the polar caps is redistributed into the mid-

latitudes of Mars as a layer about 50 meters thick, in the same place that we see that the DLEs have formed. This made us think that this ice layer could be part of the explanation for the formation of the unusual DLE second layer," Head said.

In the scenario Weiss and Head lay out, the impact blasts through the ice layer, spitting rock and other ejecta out onto the surrounding ice. But because that ejected material sits on slippery ice, it doesn't all stay put. Weiss and Head believe the layering occurs when material near the top of an upraised crater rim slides down the slippery ice and overtops material on the lower slopes. That landslide, enabled by steep slopes and a slick ice layer, creates the DLEs' telltale two-layered appearance.

"I think for the first time since DLEs were discovered in the 1970s we have a model for their formation that appears to be consistent with a very wide range of known data," Weiss said. An understanding of how these and other crater types formed could help researchers to reconstruct the environmental conditions at the time of the impacts. The research will be published in the journal Geophysical Research Letters. An early version of the paper went online on July 25.

A good fit to the data

The landslide scenario explains several of the distinct features of DLEs. Most directly, it explains radial striations — grooves radiating out from the crater rim — that are common on the inner ejecta layer of DLEs. Striations are common in landslides on Earth, Weiss said, "especially landslides on glaciers."

That got Weiss and Head thinking that ice could be a key ingredient for making a DLE. Ice would reduce the coefficient of friction on the slopes of crater rims, increasing the likelihood of a slide. "When I did a quick calculation, I realized that the landslide wouldn't be expected to happen [on crater rims] unless the ejecta was landsliding on an ice layer," Weiss said.

The scenario also requires a steep slope on the outside of a crater rim. A raised rim is partly a function of crater size, with larger craters generally having less uplift. Weiss calculated that craters larger than about 25 kilometers probably wouldn't have steep enough rims to cause an icy landslide. With those results in hand, he surveyed about 600 known DLEs and found that nearly all of them are between one and 25 kilometers in diameter.

The ice model also accounts for other distinctive features of DLEs. For example, unlike other crater types, DLEs tend not to have secondary craters surrounding them. Secondary craters are the result of big chunks of ejecta blasted out of the main crater, leaving gouges in the surrounding surface when they land. But if that surrounding surface were covered by ice, evidence of shallow secondary craters would disappear when the ice disappeared.

The model also appears to explain the locations of DLEs at middle or high latitudes — areas where scientists believe there may once have been glacial ice on Mars millions of years ago. Ultimately, understanding how DLEs and other crater types are formed could lead to a better understanding of Mars' past.

"There are over 600 DLEs on the Martian surface, so reconciling how they formed with our knowledge of the climate of Mars is pretty important," Weiss said. "It could tell us a lot about the history of the martian climate on a global scale." *The work was supported by a NASA grant to Head.*

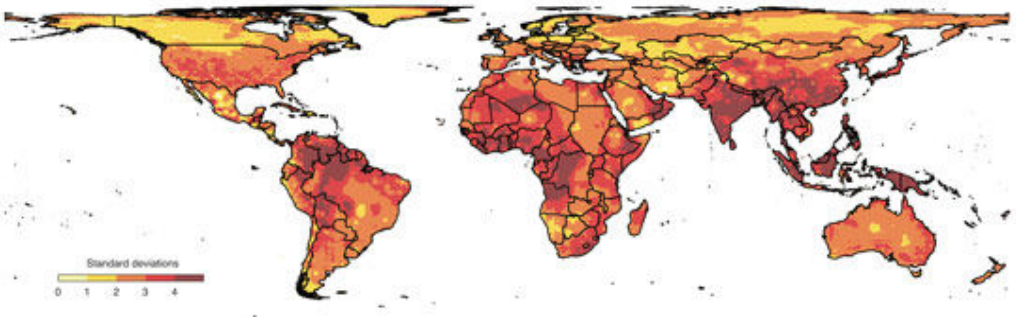
http://scitechdaily.com/warmer-weather-and-precipitation-increase-the-risk-of-violence/

Warmer Weather and Precipitation Increase the Risk of Violence

A newly published study from Princeton University and UC Berkeley reveals that slight increases in temperature and precipitation result in increased human conflict.

August 5, 2013 by Staff

Should climate change trigger the upsurge in heat and rainfall that scientists predict, people may face a threat just as perilous and volatile as extreme weather — each other.

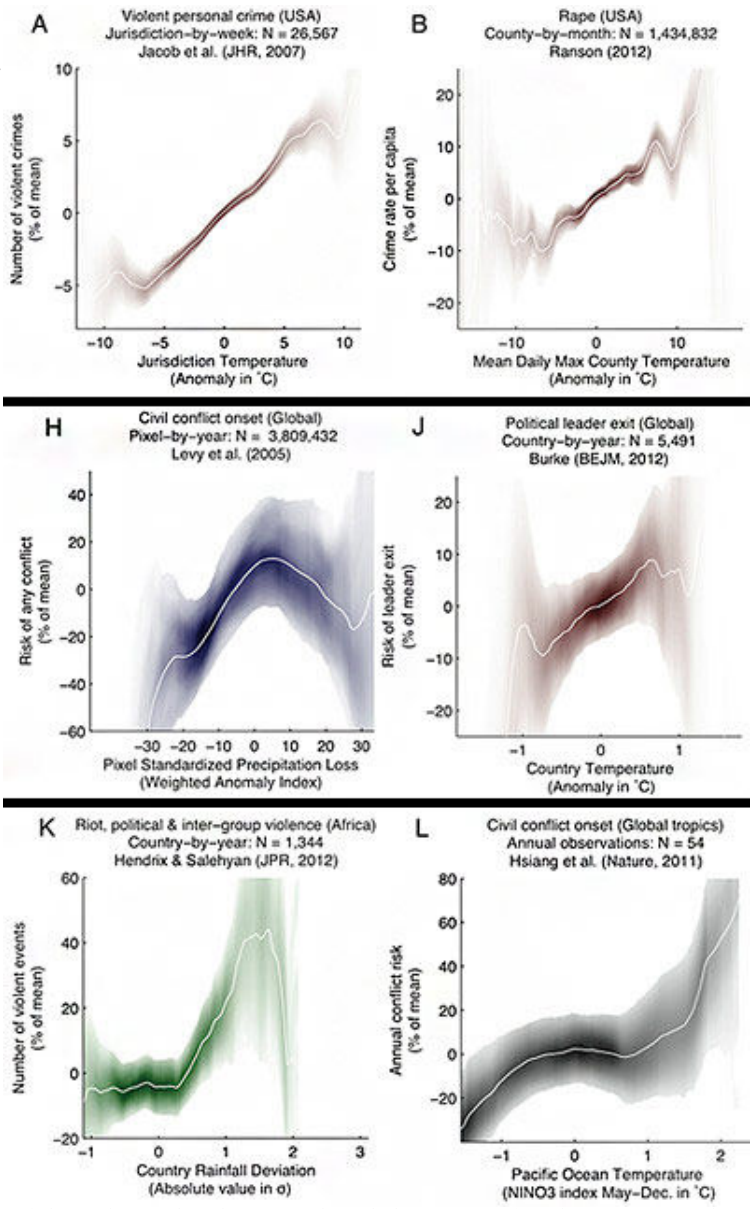


Researchers from Princeton University and the University of California-Berkeley suggest that more human conflict is a likely outcome of climate change. The researchers found that 1 standard-deviation shift — the amount of change from the local norm — in temperature and precipitation greatly increase the risk of personal violence and social upheaval. Climate-change models predict an average of 2 to 4 standard-deviation shifts in global climate conditions by 2050 (above), with 4 representing the greatest change in normal conditions. **(Image by Science/AAAS)**

Researchers from Princeton University and the University of California-Berkeley report in the journal Science that even slight spikes in temperature and precipitation have greatly increased the risk of personal violence and social upheaval throughout human history. Projected onto an Earth that is expected to warm by 2 degrees Celsius by 2050, the authors suggest that more human conflict is a likely outcome of climate change.

The researchers analyzed 60 studies from a number of disciplines — including archaeology, criminology, economics and psychology — that have explored the connection between weather and violence in various parts of the world from about 10,000 BCE to the present day. During an 18-month period, the Princeton-Berkeley researchers reviewed those studies' data — and often re-crunched raw numbers — to calculate the risk that violence would rise under hotter and wetter conditions.

They found that while climate is not the sole or primary cause of violence, it undeniably exacerbates existing social and interpersonal tension in all societies, regardless of wealth or stability. They found that 1 standard-deviation shift — the amount of change from the local norm — in heat or rainfall boosts the risk of a riot, civil war or ethnic conflict by an average of 14 percent. There is a 4 percent chance of a similarly sized upward creep in heat or rain sparking person-on-person violence such as rape, murder and assault. The researchers report that climate-change models predict an average of 2 to 4 standard-deviation shifts in global climate conditions by 2050.



Establishing a correlation between violence and climate change now allows policymakers and researchers to examine what causes it and how to intervene, said lead author Solomon Hsiang, who conducted the work as a postdoctoral research associate in the Program in Science, Technology and Environmental Policy in Princeton's Woodrow Wilson School of Public and International Affairs.

"We think that by collecting all the research together now, we're pretty clearly establishing that there is a causal relationship between the climate and human conflict," Hsiang said. "People have been skeptical up to now of an individual study here or there. But considering the body of work together, we can now show that these patterns are extremely general. It's more of the rule than the exception.

"Whether there is a relationship between climate and conflict is not the question anymore. We now want to understand what's causing it," Hsiang said. "Once we understand what causes this correlation we can think about designing effective policies or institutions to manage or interrupt the link between climate and conflict."

The researchers analyzed 60 studies from a number of disciplines that have explored the connection between weather and violence in various parts of the world, and throughout human history. A sampling of existing results (graphed above) show a correlation between temperature on violent personal crime and rape in the United States (A, B); drought and global civil conflict (H); temperature and the ouster of leaders worldwide (J); deviation from normal rainfall and large-scale violence in Africa (K); and global civil conflict and the intensity of El Niño (L). The darker areas indicate a stronger connection between climate and violence. Panel titles indicate the type of violence studied, the location, the unit of analysis and sample size, and the study citation. **(Image by Science/AAAS)**

The existing research had essentially shown an overall link between climate conditions and these conflicts, but that link needed to be extracted from reams of figures from various disciplines in order for the research to reach general conclusions, Hsiang said. Hsiang, who is now an assistant professor at Berkeley's Goldman School of

Public Policy, worked with co-first author Marshall Burke, a doctoral candidate in Berkeley's Department of Agricultural and Resource Economics, and Edward Miguel, the Oxfam Professor of Environmental and Resource Economics at Berkeley.

"We attained a huge amount of the data that was available and we used the same method on all of the data so that we could directly compare studies," Hsiang said. "Once we did that, we saw that all of the results were actually highly consistent — previously they just weren't being analyzed in a consistent way."

The researchers examined three categories of conflict: "personal violence and crime," which includes murder, assault, rape and domestic violence; "intergroup violence and political instability," such as civil wars, riots, ethnic violence and land invasions; and "institutional breakdowns," which are abrupt and major changes in governing institutions or, in extreme cases, the collapse of entire civilizations.

Extreme climatic conditions amplified violence in all three categories, regardless of geography, societal wealth or the time in history. An aberrant climate coincided with incidents including spikes in domestic violence in India and Australia; increased assaults and murders in the United States and Tanzania; ethnic violence in Europe and South Asia; land invasions in Brazil; police using force in the Netherlands; civil conflicts throughout the tropics; the collapse of ancient empires; and wars and displacement in Middle-Ages Europe.

"We find the same pattern over and over again, regardless of whether we look at data from Brazil, Somalia, China or the United States," Miguel said. "We often think of modern society as largely independent of the environment, due to technological advances, but our findings challenge that notion. The climate appears to be a critical factor sustaining peace and wellbeing across human societies."

And the climate does not have to deviate much to upset that peace and wellbeing, Burke said. The 1 standard-deviation shift he and his co-authors uncovered equates to a seemingly paltry change in weather: it's roughly equal to warming an African country by 0.35°C, or by 0.63°F, for an entire year, or warming a county in the United States by 2.9°C, or by 5.2°F, for a given month.

"These are pretty moderate changes, but they have a sizable impact on those societies," Burke said. Many global climate models project global temperature increases of at least 2 degrees Celsius over the next several decades, which, when combined with the Princeton-Berkeley findings, suggest that warming at that level could increase the risk of civil war in many countries by more than 50 percent, the researchers said.

The factors that interact with climate to produce chaos and discord are varied. A popular theory is that drought and flooding cripple an economy, especially one based on agriculture or that is already weak. When people look for someone to blame, governmental leaders have a target on their backs, as do any people with whom there is existing tension, such as an ethnic minority or a migrant group from stricken hinterlands.

But sometimes heat just makes people more aggressive. The researchers found that personal violence was far more influenced by a leap in temperature. Hsiang and his colleagues cite studies that equate excessive heat with spikes of violence in the United States and other stable, wealthy countries. For example, a 1994 study found that two groups of police officers undergoing the exact same simulation training were more likely to draw their weapons if the room was uncomfortably warm. "There's a large amount of evidence that environmental conditions actually change a person's perception of their own condition, or they also can change the likelihood of people using violence or aggressive action to accomplish some goal," Hsiang said.

"Our study is not saying that climate is the only cause of conflict, and there's no conflict that we think should be wholly attributed to some specific climatic event," he said. "Every conflict has roots in interpersonal and intergroup relations. What we're trying to point out is that climate is one of the critical factors the affect how things escalate, and if they escalate to the point of violence."

The paper, "Quantifying the influence of climate on human conflict," was published in Science Aug. 1. The study was funded by a Princeton University postdoctoral fellowship in science, technology and environmental policy, a Graduate Research Fellowship from the National Science Foundation, and the Oxfam Faculty Chair in Environmental and Resource Economics at Berkeley.

Publication: Solomon M. Hsiang, et al., "Quantifying the Influence of Climate on Human Conflict," Science, 2013; DOI: 10.1126/science.1235367 Source: Morgan Kelly, Princeton University Images: Science/AAAS

http://www.eurekalert.org/pub_releases/2013-08/nsfc-ail080513.php

Astronomers image lowest-mass exoplanet around a sun-like star

Using infrared data from the Subaru Telescope in Hawaii, an international team of astronomers has imaged a giant planet around the bright star GJ 504.

Several times the mass of Jupiter and similar in size, the new world, dubbed GJ 504b, is the lowest-mass planet ever detected around a star like the sun using direct imaging techniques. "If we could travel to this giant planet, we would see a world still glowing from the heat of its formation with a color reminiscent of a dark cherry blossom, a dull magenta," said Michael McElwain, a member of the discovery team at NASA's Goddard Space

Flight Center in Greenbelt, Md. "Our near-infrared camera reveals that its color is much more blue than other imaged planets, which may indicate that its atmosphere has fewer clouds."
GJ 504b orbits its star at nearly nine times the distance Jupiter orbits the sun, which poses a challenge to theoretical ideas of how giant planets form.
According to the most widely accepted picture, called the core-accretion model, Jupiter-like planets get their start in the gas-rich debris disk that surrounds a young star. A core produced by collisions among asteroids and comets provides a seed, and when this core reaches sufficient mass, its gravitational pull rapidly attracts gas from the disk to form the planet.
While this model works fine for planets out to where Neptune orbits, about 30 times Earth's average distance from the sun (30 astronomical units, or AU), it's more problematic for worlds located farther from their stars. GJ 504b lies at a projected distance of 43.5 AU from its star; the actual distance depends on how the system tips to our line of sight, which is not precisely known.
"This is among the hardest planets to explain in a traditional planet-formation framework," explained team member Markus Janson, a Hubble postdoctoral fellow at Princeton University in New Jersey. "Its discovery implies that we need to seriously consider alternative formation theories, or perhaps to reassess some of the basic assumptions in the core-accretion theory."
The research is part of the Strategic Explorations of Exoplanets and Disks with Subaru (SEEDS), a project to directly image extrasolar planets and protoplanetary disks around several hundred nearby stars using the Subaru Telescope on Mauna Kea, Hawaii. The five-year project began in 2009 and is led by Motohide Tamura at the National Astronomical Observatory of Japan (NAOJ).
While direct imaging is arguably the most important technique for observing planets around other stars, it is also the most challenging.
"Imaging provides information about the planet's luminosity, temperature, atmosphere and orbit, but because planets are so faint and so close to their host stars, it's like trying to take a picture of a firefly near a searchlight," explained Masayuki Kuzuhara at the Tokyo Institute of Technology, who led the discovery team.
The SEEDS project images at near-infrared wavelengths with the help of the telescope's novel adaptive optics system, which compensates for the smearing effects of Earth's atmosphere, and two instruments: the High Contrast Instrument for the Subaru Next Generation Adaptive Optics and the InfraRed Camera and Spectrograph. The combination allows the team to push the boundary of direct imaging toward fainter planets.
A paper describing the results has been accepted for publication in The Astrophysical Journal and will appear in a future issue. The researchers find that GJ 504b is about four times more massive than Jupiter and has an effective temperature of about 460 degrees Fahrenheit (237 Celsius).
It orbits the G0-type star GJ 504, which is slightly hotter than the sun and is faintly visible to the unaided eye in the constellation Virgo. The star lies 57 light-years away and the team estimates the systems is about 160 million years, based on methods that link the star's color and rotation period to it age.
Young star systems are the most attractive targets for direct exoplanet imaging because their planets have not existed long enough to lose much of the heat from their formation, which enhances their infrared brightness.
"Our sun is about halfway through its energy-producing life, but GJ504 is only one-thirtieth its age," added McElwain. "Studying these systems is a little like seeing our own planetary system in its youth."

http://www.eurekalert.org/pub_releases/2013-08/bmj-fpp080513.php

First probable person to person transmission of new bird flu virus in China

But researchers stress H7N9 is not able to spread efficiently between humans

The first report of probable person to person transmission of the new avian influenza A (H7N9) virus in Eastern China is published on bmj.com today. The findings provide the strongest evidence yet of H7N9 transmission between humans, but the authors stress that its ability to transmit itself is "limited and non-sustainable."
Avian influenza A (H7N9) virus was recently identified in Eastern China. As of 30 June 2013, 133 cases have been reported, resulting in 43 deaths. Most cases appear to have visited live poultry markets or had close contact with live poultry 7-10 days before illness onset. Currently no definite evidence indicates sustained human-to-human transmission of the H7N9 virus.
The study reports a family cluster of two patients (father and daughter) with H7N9 virus infection in Eastern China in March 2013. The first (index) patient – a 60 year old man – regularly visited a live poultry market and became ill five to six days after his last exposure to poultry. He was admitted to hospital on 11 March.
When his symptoms became worse, he was transferred to the hospital's intensive care unit (ICU) on 15 March. He was transferred to another ICU on March 18 and died of multi-organ failure on 4 May.

The second patient, his healthy 32 year old daughter, had no known exposure to live poultry before becoming sick. However, she provided direct and unprotected bedside care for her father in the hospital before his admission to intensive care.
She developed symptoms six days after her last contact with her father and was admitted to hospital on 24 March. She was transferred to the ICU on 28 March and died of multi-organ failure on 24 April.
Two almost genetically identical virus strains were isolated from each patient, suggesting transmission from father to daughter.
Forty-three close contacts of both cases were interviewed by public health officials and tested for influenza virus. Of these, one (a son in law who helped care for the father) had mild illness, but all contacts tested negative for H7N9 infection.
Environmental samples from poultry cages, water at two local poultry markets, and swans from the residential area, were also tested. One strain was isolated but was genetically different to the two strains isolated from the patients.
The researchers acknowledge some study limitations, but say that the most likely explanation for this family cluster of two cases with H7N9 infection is that the virus "transmitted directly from the index patient to his daughter." But they stress that "the virus has not gained the ability to transmit itself sustained from person to person efficiently."
They believe that the most likely source of infection for the index case was the live poultry market, and conclude: "To our best knowledge, this is the first report of probable transmissibility of the novel virus person to person with detailed epidemiological, clinical, and virological data. Our findings reinforce that the novel virus possesses the potential for pandemic spread."
So does this imply that H7N9 has come one step closer towards adapting fully to humans, ask James Rudge and Richard Coker from the London School of Hygiene and Tropical Medicine, based in Bangkok, in an accompanying editorial? Probably not, they say. Limited transmission between humans "is not surprising, and does not necessarily indicate that the virus is on course to develop sustained transmission among humans."
Nevertheless, they point to several traits of H7N9 are of particular concern, and conclude that, while this study might not suggest that H7N9 is any closer to delivering the next pandemic, "it does provide a timely reminder of the need to remain extremely vigilant: the threat posed by H7N9 has by no means passed."
The authors also summarise their findings in a video abstract. Dr Zhou says that the reason for carrying out this study was because there was "no definite evidence to show that the novel virus can transmit person-to-person", plus she and her co-authors wanted to find out whether the novel avian influenza virus possesses the capability to transmit person-to-person. She concludes that "the infection of the daughter is likely to have resulted from her father during unprotected exposure" and suggest that the virus possesses the ability to transmit person-to-person in this cluster. She does add however that the infection was "limited and non-sustainable as there is no outbreak following the two cases".

http://www.eurekalert.org/pub_releases/2013-08/esoh-loh080513.php

Length of human pregnancies can vary naturally by as much as 5 weeks

The length of a human pregnancy can vary naturally by as much as five weeks, according to research published online today (Wednesday) in Europe's leading reproductive medicine journal Human Reproduction [1].

Normally, women are given a date for the likely delivery of their baby that is calculated as 280 days after the onset of their last menstrual period. Yet only four percent of women deliver at 280 days and only 70% deliver within 10 days of their estimated due date, even when the date is calculated with the help of ultrasound.
Now, for the first time, researchers in the USA have been able to pinpoint the precise point at which a woman ovulates and a fertilised embryo implants in the womb during a naturally conceived pregnancy, and follow the pregnancy through to delivery. Using this information, they have been able to calculate the length of 125 pregnancies.
"We found that the average time from ovulation to birth was 268 days – 38 weeks and two days," said Dr Anne Marie Jukic, a postdoctoral fellow in the Epidemiology Branch at the National Institute of Environmental Health Sciences (Durham, USA), part of the National Institutes for Health. "However, even after we had excluded six pre-term births, we found that the length of the pregnancies varied by as much as 37 days.
"We were a bit surprised by this finding. We know that length of gestation varies among women, but some part of that variation has always been attributed to errors in the assignment of gestational age. Our measure of length of gestation does not include these sources of error, and yet there is still five weeks of variability. It's fascinating."

The possibility that the length of pregnancies can vary naturally has been little researched, as it is impossible to tell the difference between errors in calculations and natural variability without being able to measure correctly the gestational age of a developing foetus. Previous studies conducted as long ago as the 1970s and 1980s had used the slight rise in a woman's body temperature at waking as a way of detecting when ovulation occurred. This is an inexact measurement and cannot be used to detect when the embryo actually implants in the womb.
In the current study, the researchers took information from daily urine samples collected by women taking part in an earlier study, the North Carolina Early Pregnancy Study, which took place between 1982-1985 and followed 130 singleton pregnancies from unassisted conception through to birth. The women had discontinued contraception in order to become pregnant; they were healthy, with no known fertility problems and they were also less likely to smoke or be obese. The women completed daily diaries and collected daily first-morning urine samples for six months or until the end of the eighth week if they became pregnant.
The urine samples were analysed for three hormones connected with the onset of pregnancy: hCG (human chorionic gonadotropin), estrone-3-glucoronide and pregnanediol-3-glucoronide. The day of ovulation was identified by the drop in the ratio between the hormones oestrogen and progesterone. Embryo implantation was identified as the first day of a sustained rise in levels of hCG. "Since the embryo secretes hCG, and mothers generally have little to no hCG in their urine when they are not pregnant, we used the earliest increase in hCG to indicate implantation," explained Dr Jukic.
In 2010, the researchers contacted the women for the current study to obtain information about their labour and whether induction or Caesarean section had been required. Full information was available on 125 pregnancies after excluding those that had been affected by exposure to diethylstilbestrol – an endocrine disrupter that is known to shorten pregnancies.
In addition to the variation in the length of gestation, the study found that embryos that took longer to implant, also took longer from implantation to delivery, and that pregnancies that showed a late progesterone rise were significantly shorter by an average of 12 days than pregnancies with an early rise.
Dr Jukic said: "I am intrigued by the observation that events that occur very early in pregnancy, weeks before a woman even knows she is pregnant, are related to the timing of birth, which occurs months later. I think this suggests that events in early pregnancy may provide a novel pathway for investigating birth outcomes."
Other factors that appeared to influence pregnancy duration included: older women delivered later on average, with each year of age adding roughly one day to their pregnancy; women who had themselves been heavier at birth had longer gestations, with each 100g in the mother's own birthweight corresponding roughly to a one-day longer pregnancy; and if a woman had longer pregnancies previously or subsequently to the pregnancy being investigated in the study, then the study pregnancy was likely to be longer, with a one-week increase in the average length corresponding to about a 2.5-day longer pregnancy in the study. "This last finding suggests that individual women tend to be consistent about when they deliver," said Dr Jukic.
In their paper, the authors conclude: "The length of human gestation varies considerably among healthy pregnancies, even when ovulation is accurately measured. This variability is greater than suggested by the clinical assignment of a single 'due date'. The duration of previous pregnancies may provide a useful measure of a woman's 'natural' length of pregnancy and may help in predicting an individual woman's due date. We also found that events in the first two weeks after conception were strongly predictive of the total length of pregnancy, suggesting that the trajectory for the timing of delivery may be set in early pregnancy."
They warn that it is too early to make clinical recommendations based on their study and that further research needs to be carried out.
Dr Jukic concluded: "I think the best that can be said is that natural variability may be greater than we have previously thought, and if that is true, clinicians may want to keep that in mind when trying to decide whether to intervene on a pregnancy."

[1] *"Length of human pregnancy and contributors to its natural variation", by A.M. Jukic, D.D. Baird, C.R. Weinberg, D.R. McConnaughey, A.J. Wilcox. Human Reproduction journal. doi:10.1093/humrep/det297*

http://nyti.ms/13gl7gT

Can Immunity to the Common Cold Come With Age?

Q. My mother, who lived to be 92, never caught my sniffles in her later years, even though I was her sole caretaker. And now that I'm in my 60s, I notice that my colds are less severe. Is it possible to develop immunity to the common cold?

By C. CLAIBORNE RAY

A. Possibly, to specific kinds of cold, said Dr. Jonathan L. Jacobs, professor of clinical medicine at NewYork-Presbyterian Hospital/Weill Cornell Medical Center.

"The syndrome that we call the common cold can be caused by many different viruses," he said. "After most viral infections, people develop immunity to that specific virus, which can last from a few years to a lifetime."
As immunity to different viruses builds up over time, it decreases the number of viral types that can make one sick, Dr. Jacobs said. But he added, "There are so many viruses that cause colds that complete immunity is very unlikely."
As for the strength of symptoms of colds later in life, "our genes, and the strength of the immune mechanisms that produce many of the symptoms that we associate with the common cold, are also important factors determining how sick we get when exposed to a cold virus," Dr. Jacobs said.

http://phys.org/news/2013-08-psychologists-group-level-narcissism-linked-negative.html

Psychologists say 'group-level narcissism' linked to negative attitudes toward immigrants

Feelings of entitlement and superiority that go beyond patriotism and love of country may be a key predictor for Americans who will feel or behave negatively toward undocumented Latino immigrants, according to a study from The University of Texas at Arlington.

Researchers looked at those enhanced feelings of superiority - referred to as group-level narcissism – along with a factor called national in-group identification in a new work to be published in the August issue of the Hispanic Journal of Behavioral Science.
National in-group identification happens when a person's individual identity is strongly tied to and dependent on their membership in a group, like being an American.
Previous research has found that strong in-group identity is not necessarily a predictor of negative attitudes toward other groups.
The UT Arlington team found, however, that attitudes changed when a strong in-group identity was paired with an average or above average group narcissism. Then, negative attitudes toward undocumented Latino immigrants were more likely.
"When you look at the rhetoric surrounding undocumented, Latino immigrants in the United States, the perspectives vary widely – from those who characterize undocumented immigrants as criminals to those who support expanding full citizenship rights," said Patricia Lyons, a graduate of the psychology doctoral program in the UT Arlington College of Science and a member of the research team. "We were interested in understanding how and why attitudes varied so widely from a psychological perspective. The group narcissism measure gave us a way to understand these attitudes. "
Lyons co-authored the study with Jared Kenworthy, a UT Arlington associate psychology professor, and Ph.D. candidate Lauren E. Coursey. Lyons is currently on the psychology faculty at Mountain View College in Dallas.
The team surveyed 223 university students with tools designed to measure their national in-group identity and propensity for group-level narcissism, which is defined as "an inflated image of one's group based on feelings of superiority, entitlement and the need for constant attention and praise at the collective level."
For example, the test assessing group narcissism asked participants to rank how strongly they agreed with statements such as "If America ruled the world it would be a better place" and "America is the best country in the world."
The newly published paper builds on earlier research by Kenworthy and Lyons about the relationship between in-group identification, group-level narcissism and negative attitudes toward Arab-Americans. That study also found that group-level narcissism was linked to negative attitudes.
The researchers believe that increases in group-level narcissism may be prompted by perceived threats to someone's group from an outside group. Those perceived threats could center on a loss of valuable resources or job opportunities, or threats to one's personal beliefs. The team hopes to examine the threat component in future research.
At a time when conversations about immigration reform can often turn ugly, the research team hopes their latest work adds to understanding about what can cause divisive attitudes and how those relationships might be improved.
Still, there are no easy answers, Kenworthy said.
"One thing we do know from previous research is that mere knowledge about, or even contact with, another group is not adequate to reduce negative attitudes," Kenworthy said. "First, members of different groups must be given the opportunity to come together in a setting of mutual goals, equal status and cooperation."
More information: The study is titled: "National Identity and Group Narcissism as Predictors of Intergroup Attitudes Toward Undocumented Latino Immigrants in the United States" and can be found online here: hjb.sagepub.com/content/35/3/323.abstract

http://www.sciencedaily.com/releases/2013/08/130805223112.htm

New Way to Dramatically Raise RNA Treatment Potency: Proof-Of-Principle Drug Candidate Powerfully Neutralizes Myotonic Dystrophy Defect

Scientists from the Jupiter campus of The Scripps Research Institute (TSRI) have shown a novel way to dramatically raise the potency of drug candidates targeting RNA, resulting in a 2,500-fold improvement in potency and significantly increasing their potential as therapeutic agents.

The new study, published recently online ahead of print by the journal Angewandte Chemie, confirms for the first time that a small molecule actually binds to a disease-causing RNA target -- a breakthrough that should help scientists identify precise RNA targets within living cells, profile their interactions, and predict drug candidates' side effects.

"We're trying to make tools that can target any RNA motif," said Matthew Disney, a TSRI associate professor who authored the research with a research associate in his lab, Lirui Guan. "This study completely validates our design -- it validates that our compound targets the desired RNA sequence in a complex cellular environment that contains many hundreds of thousands of RNAs."

While targeting DNA has been used as a therapeutic strategy against cancer, few similar approaches have been attempted for disease-associated RNAs.

In the new study, the scientists created a small molecule that binds to the genetic defect in RNA that causes myotonic dystrophy type 1 and improves associated defects in cell culture.

Myotonic dystrophy type 1 involves a type of RNA defect known as a "triplet repeat," a series of three nucleotides repeated more times than normal in an individual's genetic code. In this case, the repetition of the cytosine-uracil-guanine (CUG) in the RNA sequence leads to disease by binding to a particular protein, MBNL1, rendering it inactive and resulting in a number of protein-splicing abnormalities.

To achieve the increase in the drug candidate's potency, Disney and his colleagues attached a reactive molecule (a derivative of chlorambucil, a chemotherapy drug that has been used to treatment a form of leukemia) to the small molecule they had identified. As a result, the new compound not only binds to the target, it becomes a permanent part of the target -- as if it were super glued to it, Disney said. Once attached, it switches off the CUG defect and prevents the cell from turning it back on.

Disney was surprised at the approximately 2,500-fold improvement in potency with the new approach.

"I was shocked by the increase," he said. "This takes the potency into the realm where one would like to see if the compound were to have real therapeutic potential."

As a result, the new compound, known as 2H-4-CA, is the most potent compound known to date that improves DM1-associated splicing defects. Importantly, 2H-4-CA does not affect the alternative splicing of a transcript not regulated by MBNL1, demonstrating selectivity for the CUG repeat and suggesting that it might have minimal side effects. "We can now use this approach to attach reactive molecules to other RNA targeted small molecules," Disney said.

The reactive molecule model also provides a potentially general method to identify cellular targets of RNA-directed small molecules. Such probes could also identify unintended targets, information that could be used to design and identify compounds with improved selectivity in an approach similar to activity-based profiling, Disney said.

Lirui Guan, Matthew D. Disney. Covalent Small-Molecule-RNA Complex Formation Enables Cellular Profiling of Small-Molecule-RNA Interactions. Angewandte Chemie International Edition, 2013; DOI: 10.1002/anie.201301639

http://www.eurekalert.org/pub_releases/2013-08/miot-wtb080613.php

Why tumors become drug-resistant

New findings could lead to drugs that fight back when tumors don't respond to treatment

Anne Trafton, MIT News Office

CAMBRIDGE, Mass-- Cancer drugs known as ErbB inhibitors have shown great success in treating many patients with lung, breast, colon and other types of cancer. However, ErbB drug resistance means that many other patients do not respond, and even among those who do, tumors commonly come back.

A new study from MIT reveals that much of this resistance develops because a protein called AXL helps cancer cells to circumvent the effects of ErbB inhibitors, allowing them to grow unchecked. The findings suggest that combining drugs that target AXL and ErbB receptors could offer a better way to fight tumors, says Doug Lauffenburger, the Ford Professor of Bioengineering, head of MIT's Department of Biological Engineering and an affiliate member of MIT's Koch Institute for Integrative Cancer Research.

"Drug resistance is the major challenge in cancer these days. People are coming up with a lot of targeted therapies for particular genes and identifying drugs that work against them, but resistance is just invariably the

issue," says Lauffenburger, the senior author of a paper describing the findings in the Aug. 6 issue of Science Signaling.
ErbBs, a family of epithelial growth factor receptors (EGFRs), are proteins that are often overactive in cancer cells, causing them to grow and divide uncontrollably. The drug Iressa is used to treat lung cancer patients whose tumors overexpress one type of ErbB mutant, and Herceptin targets another ErbB family member that is found in certain types of breast cancer.
"There are a lot of excellent drugs that target EGFR itself or other members of that family, yet they have these limitations," Lauffenburger says.

Systems analysis

In the new study, Lauffenburger and colleagues set out to identify factors that help tumor cells become resistant to EGFR and other ErbB inhibitors. To do this, they developed a new computer model and applied it to a large dataset called the Cancer Cell Line Encyclopedia, which includes information on about 1,000 human cancer lines and their responses to different drugs.
Led by biological engineering graduate student Aaron Meyer, lead author of the paper, the researchers created a machine learning program that can sift through the data and look for pairs of overexpressed proteins that make tumor cells resistant to EGFR inhibitors. In this case, they searched for the EGFR protein in combination with every other possible protein in the database, one pair at a time.
Through this analysis, the researchers found that EGFR paired with the AXL receptor appears to be the strongest marker for EGFR inhibitor resistance. They found this pattern across many types of cancer, including lung, breast and pancreatic.
A few previous studies have shown that overexpression of AXL is associated with resistance to EGFR inhibitors in a particular tumor, but this is the first systematic study to demonstrate the correlation, Lauffenburger says. This "systems biology" approach, which focuses on complex interactions within biological systems, is critical for finding new drugs that work together to knock out cancer's defense mechanisms, he says.
"It's now well known that when you look for a single pathway, you won't get to an effective therapeutic. You will end up with resistance," Lauffenburger says. "You've got to look at pathways in combination, you've got to look at whole interacting networks. That's the only way."

Clues to a mystery

Then, in experiments on cancer cells grown in the lab, the researchers found that the AXL protein tends to cluster with EGFR on cell surfaces, so when EGFR is activated, AXL also becomes active. AXL then not only stimulates further much of the same cellular machinery targeted by EGFR, but also additional pathways provoking cell growth and division. AXL also helps cells become more motile, allowing cancer to spread through the body.
The researchers also showed that other members of the ErbB family beyond EGFR similarly cluster with AXL. This suggests that AXL inhibition may also be effective for treating breast cancers dependent on ErbB2 or ovarian cancers that overexpress ErbB3, Lauffenburger says.
The study sheds light on the complicated interactions between EGFR and other proteins that help tumors re-emerge after initial treatment with EGFR inhibitors, and could help researchers develop improved treatments, says Trever Bivona, an assistant professor of medicine at the University of California at San Francisco.
"The implication that emerges from the findings is that the way receptor kinases interact to undermine sensitivity to treatment is quite complex," says Bivona, who was not part of the research team.
High levels of AXL have previously been found in triple-negative breast cancer cells, which lack the three most common breast cancer markers — estrogen receptors, progesterone receptors and HER2 receptors. The new finding may explain why EGFR inhibitors fail to work on these tumors even though they have high EGFR levels, Lauffenburger says.
"Triple-negative breast cancer cells were a special interest of ours mainly because it's always been such a mystery why they have not responded to EGFR inhibitors," he says.
The new study suggests that AXL inhibitors, either alone or in combination with EGFR inhibitors, might be an effective treatment for triple-negative breast cancer, which is now treated with chemotherapy drugs that have severe side effects. A handful of clinical trials are currently testing AXL inhibitors against different types of cancer, and Lauffenburger is now planning studies in mice to investigate the effects of combining AXL and EGFR pathway inhibitors.
Biological engineering graduate student Miles Miller and Frank Gertler, a professor of biology and member of the Koch Institute, are also co-authors of the paper. The research was funded by the National Cancer Institute Integrative Cancer Biology Program, the Department of Defense Breast Cancer Research Program, and the Koch Institute Frontier Research Program.

http://www.sciencedaily.com/releases/2013/08/130806091817.htm

Tidy Desk or Messy Desk? Each Has Its Benefits

Working at a clean and prim desk may promote healthy eating, generosity, and conventionality, according to new research.

But, the research also shows that a messy desk may confer its own benefits, promoting creative thinking and stimulating new ideas. The new studies, conducted by psychological scientist Kathleen Vohs and her fellow researchers at the University of Minnesota are published in Psychological Science, a journal of the Association for Psychological Science.

"Prior work has found that a clean setting leads people to do good things: Not engage in crime, not litter, and show more generosity," Vohs explains. "We found, however, that you can get really valuable outcomes from being in a messy setting."

In the first of several experiments, participants were asked to fill out some questionnaires in an office. Some completed the task in a clean and orderly office, while others did so in an unkempt one -- papers were strewn about, and office supplies were cluttered here and there.

Afterward, the participants were presented with the opportunity to donate to a charity, and they were allowed to take a snack of chocolate or an apple on their way out. Being in a clean room seemed to encourage people to do what was expected of them, Vohs explains. Compared with participants in the messy room, they donated more of their own money to charity and were more likely to choose the apple over the candy bar.

But the researchers hypothesized that messiness might have its virtues as well. In another experiment, participants were asked to come up with new uses for ping pong balls.

Overall, participants in the messy room generated the same number of ideas for new uses as their clean-room counterparts. But their ideas were rated as more interesting and creative when evaluated by impartial judges.

"Being in a messy room led to something that firms, industries, and societies want more of: Creativity," says Vohs.

The researchers also found that when participants were given a choice between a new product and an established one, those in the messy room were more likely to prefer the novel one -- a signal that being in a disorderly environment stimulates a release from conventionality. Whereas participants in a tidy room preferred the established product over the new one.

"Disorderly environments seem to inspire breaking free of tradition, which can produce fresh insights," Vohs concludes. "Orderly environments, in contrast, encourage convention and playing it safe."

Surprisingly, the specific physical location didn't seem to matter: "We used 6 different locations in our paper -- the specifics of the rooms were not important. Just making that environment tidy or unkempt made a whopping difference in people's behavior," says Vohs.

The researchers are continuing to investigate whether these effects might even transfer to a virtual environment: the Internet. Preliminary findings suggest that the tidiness of a webpage predicts the same kind of behaviors. These preliminary data, coupled with the findings just published, are especially intriguing because of their broad relevance:

"We are all exposed to various kinds of settings, such as in our office space, our homes, our cars, even on the Internet," Vohs observes. "Whether you have control over the tidiness of the environment or not, you are exposed to it and our research shows it can affect you."

Co-authors on this research include Joseph Redden and Ryan Rahinel of the University of Minnesota. Redden discusses the new research in this video from the Carlson School of Management of the University of Minnesota.

K. D. Vohs, J. P. Redden, R. Rahinel. Physical Order Produces Healthy Choices, Generosity, and Conventionality, Whereas Disorder Produces Creativity. Psychological Science, 2013; DOI: 10.1177/0956797613480186

http://www.eurekalert.org/pub_releases/2013-08/nsfc-tsm080613.php

The sun's magnetic field is about to flip

Something big is about to happen on the sun. According to measurements from NASA-supported observatories, the sun's vast magnetic field is about to flip.

"It looks like we're no more than three to four months away from a complete field reversal," said solar physicist Todd Hoeksema of Stanford University. "This change will have ripple effects throughout the solar system."

The sun's magnetic field changes polarity approximately every 11 years. It happens at the peak of each solar cycle as the sun's inner magnetic dynamo re-organizes itself. The coming reversal will mark the midpoint of Solar Cycle 24. Half of "solar max" will be behind us, with half yet to come.

Hoeksema is the director of Stanford's Wilcox Solar Observatory, one of the few observatories in the world that monitors the sun's polar magnetic fields. The poles are a herald of change. Just as Earth scientists watch our

planet's polar regions for signs of climate change, solar physicists do the same thing for the sun. Magnetograms at Wilcox have been tracking the sun's polar magnetism since 1976, and they have recorded three grand reversals—with a fourth in the offing.
Solar physicist Phil Scherrer, also at Stanford, describes what happens: "The sun's polar magnetic fields weaken, go to zero and then emerge again with the opposite polarity. This is a regular part of the solar cycle."
A reversal of the sun's magnetic field is, literally, a big event. The domain of the sun's magnetic influence (also known as the "heliosphere") extends billions of kilometers beyond Pluto. Changes to the field's polarity ripple all the way out to the Voyager probes, on the doorstep of interstellar space.
When solar physicists talk about solar field reversals, their conversation often centers on the "current sheet." The current sheet is a sprawling surface jutting outward from the sun's equator where the sun's slowly rotating magnetic field induces an electrical current. The current itself is small, only one ten-billionth of an amp per square meter (0.0000000001 amps/m2), but there's a lot of it: the amperage flows through a region 10,000 km thick and billions of kilometers wide. Electrically speaking, the entire heliosphere is organized around this enormous sheet.
During field reversals, the current sheet becomes very wavy. Scherrer likens the undulations to the seams on a baseball. As Earth orbits the sun, we dip in and out of the current sheet. Transitions from one side to another can stir up stormy space weather around our planet.
Cosmic rays are also affected. These are high-energy particles accelerated to nearly light speed by supernova explosions and other violent events in the galaxy. Cosmic rays are a danger to astronauts and space probes, and some researchers say they might affect the cloudiness and climate of Earth. The current sheet acts as a barrier to cosmic rays, deflecting them as they attempt to penetrate the inner solar system. A wavy, crinkly sheet acts as a better shield against these energetic particles from deep space.
As the field reversal approaches, data from Wilcox show that the sun's two hemispheres are out of synch. "The sun's north pole has already changed sign, while the south pole is racing to catch up," Scherrer said. "Soon, however, both poles will be reversed, and the second half of solar max will be underway."
When that happens, Hoeksema and Scherrer will share the news with their colleagues and the public.

http://www.eurekalert.org/pub_releases/2013-08/aaon-cmh073113.php

Chocolate may help keep brain healthy

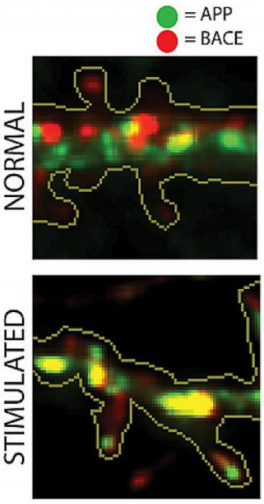
Drinking two cups of hot chocolate a day may help older people keep their brains healthy and their thinking skills sharp, according to a study published in the August 7, 2013, online issue of Neurology®, the medical journal of the American Academy of Neurology.

MINNEAPOLIS – The study involved 60 people with an average age of 73 who did not have dementia. The participants drank two cups of hot cocoa per day for 30 days and did not consume any other chocolate during the study. They were given tests of memory and thinking skills. They also had ultrasounds tests to measure the amount of blood flow to the brain during the tests.
"We're learning more about blood flow in the brain and its effect on thinking skills," said study author Farzaneh A. Sorond, MD, PhD, of Harvard Medical School in Boston and a member of the American Academy of Neurology. "As different areas of the brain need more energy to complete their tasks, they also need greater blood flow. This relationship, called neurovascular coupling, may play an important role in diseases such as Alzheimer's."
Of the 60 participants, 18 had impaired blood flow at the start of the study. Those people had an 8.3-percent improvement in the blood flow to the working areas of the brain by the end of the study, while there was no improvement for those who started out with regular blood flow.
The people with impaired blood flow also improved their times on a test of working memory, with scores dropping from 167 seconds at the beginning of the study to 116 seconds at the end. There was no change in times for people with regular blood flow.
A total of 24 of the participants also had MRI scans of the brain to look for tiny areas of brain damage. The scans found that people with impaired blood flow were also more likely to have these areas of brain damage.
Half of the study participants received hot cocoa that was rich in the antioxidant flavanol, while the other half received flavanol-poor hot cocoa. There were no differences between the two groups in the results.
"More work is needed to prove a link between cocoa, blood flow problems and cognitive decline," said Paul B. Rosenberg, MD, of Johns Hopkins School of Medicine in Baltimore, who wrote an editorial accompanying the study. "But this is an important first step that could guide future studies."
The study was supported by the National Institute on Aging and the National Heart, Lung, and Blood Institute. The cocoa was provided by Mars Inc.

http://www.eurekalert.org/pub_releases/2013-08/uoc--wdw080213.php

Why don't we all get Alzheimer's disease?

Though one might think the brains of people who develop Alzheimer's disease (AD) possess building blocks of the disease absent in healthy brains, for most sufferers, this is not true.



Every human brain contains the ingredients necessary to spark AD, but while an estimated 5 million Americans have AD – a number projected to triple by 2050 – the vast majority of people do not and will not develop the devastating neurological condition.

For researchers like Subhojit Roy, MD, PhD, associate professor in the Departments of Pathology and Neurosciences at the University of California, San Diego School of Medicine, these facts produce a singular question: Why don't we all get Alzheimer's disease?

In a paper published in the August 7 issue of the journal Neuron, Roy and colleagues offer an explanation – a trick of nature that, in most people, maintains critical separation between a protein and an enzyme that, when combined, trigger the progressive cell degeneration and death characteristic of AD.

"It's like physically separating gunpowder and match so that the inevitable explosion is avoided," said principal investigator Roy, a cell biologist and neuropathologist in the Shiley-Marcos Alzheimer's Disease Research Center at UC San Diego. "Knowing how the gunpowder and match are separated may give us new insights into possibly stopping the disease."

Top: Vesicles containing APP (green) and BACE (red) are normally segregated in neurons. Bottom: After neuronal stimulation, known to produce more beta-amyloid, APP and BACE converge in common vesicles, depicted in yellow.

UC San Diego School of Medicine

The severity of AD is measured in the loss of functioning neurons. In pathological terms, there are two tell-tale signs of AD: clumps of a protein called beta-amyloid "plaques" that accumulate outside neurons and threads or "tangles" of another protein, called tau, found inside neurons.

Most neuroscientists believe AD is caused by the accumulating assemblies of beta-amyloid protein triggering a sequence of events that leads to impaired cell function and death. This so-called "amyloid cascade hypothesis" puts beta-amyloid protein at the center of AD pathology.

Creating beta-amyloid requires the convergence of a protein called amyloid precursor protein (APP) and an enzyme that cleaves APP into smaller toxic fragments called beta-secretase or BACE.

"Both of these proteins are highly expressed in the brain," said Roy, "and if they were allowed to combine continuously, we would all have AD."

But that doesn't happen. Using cultured hippocampal neurons and tissue from human and mouse brains, Roy – along with first author Utpal Das, a postdoctoral fellow in Roy's lab, and colleagues – discovered that healthy brain cells largely segregate APP and BACE-1 into distinct compartments as soon as they are manufactured, ensuring the two proteins do not have much contact with each other.

“Nature seems to have come up with an interesting trick to separate co-conspirators," said Roy.

The scientists also found that the conditions promoting greater production of beta-amyloid protein boost the convergence of APP and BACE.

Specifically, an increase in neuronal electrical activity – known to increase the production of beta-amyloid – also led to an increase in APP-BACE convergence. Post-mortem examinations of AD patients revealed increased physical proximity of the proteins as well, adding support to the pathophysiological significance of this phenomenon in human disease.

Das said the findings are fundamentally important because they elucidate some of the earliest molecular events triggering AD and show how a healthy brain naturally avoids them. In clinical terms, they point to a possible new avenue for ultimately treating or even preventing the disease.

"An exciting aspect is that we can perhaps screen for molecules that can physically keep APP and BACE-1 apart," said Das. "It's a somewhat unconventional approach."

Co-authors are David Scott, Archan Ganguly and Yong Tang, UCSD Departments of Pathology and Neurosciences; and Edward H. Koo, UCSD Department of Neurosciences. Roy and Koo are also members of the Shiley-Marcos Alzheimer's Disease Research Center (ADRC) at UC San Diego.

Funding for this research came from the American Federation for Aging Research, National Institutes of Health grant P50AG005131 and a gift from Darlene Shiley to the ADRC.

http://www.sciencedaily.com/releases/2013/08/130806132750.htm

Emotional Behavior of Adults Could Be Triggered in the Womb

Adults could be at greater risk of becoming anxious and vulnerable to poor mental health if they were deprived of certain hormones while developing in the womb according to new research by scientists at Cardiff and Cambridge universities.

New research in mice has revealed the role of the placenta in long-term programming of emotional behaviour and the first time scientists have linked changes in adult behaviour to alterations in placental function.

Insulin-like growth factor-2 has been shown to play a major role in fetal and placental development in mammals, and changes in expression of this hormone in the placenta and fetus are implicated in growth restriction in the womb.

"The growth of a baby is a very complex process and there are lots of control mechanisms which make sure that the nutrients required by the baby to grow can be supplied by the mother," according to Professor Lawrence Wilkinson, a behavioural neuroscientist from Cardiff University's School of Psychology who led the research. "We were interested in how disrupting this balance could influence emotional behaviours a long time after being born, as an adult," he added.

In order to explore how a mismatch between supply and demand of certain nutrients might affect humans, Professor Wilkinson and his colleagues Dr Trevor Humby, Mikael Mikaelsson -- both also from Cardiff University -- and Dr Miguel Constancia of Cambridge University, examined the behaviour of adult mice with a malfunctioned supply of a vital hormone.

Dr Humby added: "We achieved this by damaging a hormone called Insulin-like growth factor-2, important for controlling growth in the womb. What we found when we did this was an imbalance in the supply of nutrients controlled by the placenta, and that this imbalance had major effects on how subjects were during adulthood -- namely, that subject became more anxious later in life.

"These symptoms were accompanied by specific changes in brain gene expression related to this type of behaviour. This is the first example of what we have termed 'placental-programming' of adult behaviour. We do not know exactly how these very early life events can cause long-range effects on our emotional predispositions, but we suspect that our research findings may indicate that the seeds of our behaviour, and possibly vulnerability to brain and mental health disorders, are sown much earlier than previously thought."

Although these studies were carried out in mice, the findings may have wider implications for human development. Further studies are planned to investigate the brain mechanisms linking early life events, placental dysfunction and the emotional state of adults.

Mikael Allan Mikaelsson, Miguel Constância, Claire L. Dent, Lawrence S. Wilkinson, Trevor Humby. Placental programming of anxiety in adulthood revealed by Igf2-null models. Nature Communications, 2013; 4 DOI: 10.1038/ncomms3311

http://www.eurekalert.org/pub_releases/2013-08/uocm-npf080113.php

New proto-mammal fossil sheds light on evolution of earliest mammals

A newly discovered fossil reveals the evolutionary adaptations of a 165-million-year-old proto-mammal, providing evidence that traits such as hair and fur originated well before the rise of the first true mammals.

The biological features of this ancient mammalian relative, named Megaconus mammaliaformis, are described by scientists from the University of Chicago in the Aug 8 issue of Nature.

"We finally have a glimpse of what may be the ancestral condition of all mammals, by looking at what is preserved in Megaconus. It allows us to piece together poorly understood details of the critical transition of modern mammals from pre-mammalian ancestors," said Zhe-Xi Luo, professor of organismal biology and anatomy at the University of Chicago.

Discovered in Inner Mongolia, China, Megaconus is one of the best-preserved fossils of the mammaliaform groups, which are long-extinct relatives to modern mammals. Dated to be around 165 million years old, Megaconus co-existed with feathered dinosaurs in the Jurassic era, nearly 100 million years before Tyrannosaurus Rex roamed Earth.

Megaconus was a nocturnal animal, foraging mostly in the night. It lived on the shores of a shallow freshwater lake in what is now the Inner Mongolia Region of China.

Preserved in the fossil is a clear halo of guard hairs and underfur residue, making Megaconus only the second known pre-mammalian fossil with fur. It was found with sparse hairs around its abdomen, leading the team to hypothesize that it had a naked abdomen. On its heel, Megaconus possessed a long keratinous spur, which was

possibly poisonous. Similar to spurs found on modern egg-laying mammals, such as male platypuses, the spur is evidence that this fossil was most likely a male member of its species.
"Megaconus confirms that many modern mammalian biological functions related to skin and integument had already evolved before the rise of modern mammals," said Luo, who was also part of the team that first discovered evidence of hair in pre-mammalian species in 2006 (Science, 331: 1123-1127, DOI:10.1126/science.1123026).
A terrestrial animal about the size of a large ground squirrel, Megaconus was likely an omnivore, possessing clearly mammalian dental features and jaw hinge. Its molars had elaborate rows of cusps for chewing on plants, and some of its anterior teeth possessed large cusps that allowed it to eat insects and worms, perhaps even other small vertebrates. It had teeth with high crowns and fused roots similar to more modern, but unrelated, mammalian species such as rodents. Its high-crowned teeth also appeared to be slow growing like modern placental mammals.
The skeleton of Megaconus, especially its hind-leg bones and finger claws, likely gave it a gait similar to modern armadillos, a previously unknown type of locomotion in mammaliaforms.
Luo and his team identified clearly non-mammalian characteristics as well. Its primitive middle ear, still attached to the jaw, was reptile-like. Its anklebones and vertebral column are also similar to the anatomy of previously known mammal-like reptiles.
"We cannot say that Megaconus is our direct ancestor, but it certainly looks like a great-great-grand uncle 165 million years removed. These features are evidence of what our mammalian ancestor looked like during the Triassic-Jurassic transition," Luo said.


Photographed by Zhe-Xi Luo/University of Chicago

Megaconus mammaliaformis is preserved as a slab (left) and a counter-slab (right) of shale deposited in a shallow lake. The preserved part of the skeleton, from head to rump, is about 21 cm (8 inches). By the length of long bones, Megaconus is estimated to weigh about 250 grams (almost 9 ounces). The fossil assemblage from the Daohugou Site include several other mammals, such as semi-aquatic swimmer Castorocauda, gliding mammal Volaticotherium, feathered dinosaurs, amphibians, abundant arthropods and plants. Megaconus is the first skeletal fossil of a mammaliaform group otherwise only known by their teeth, but show a long history extending back to Late Triassic, and a wide distribution in the Jurassic.

"Megaconus shows that many adaptations found in modern mammals were already tried by our distant, extinct relatives. In a sense, the three big branches of modern mammals are all accidental survivors among many other mammaliaform lineages that perished in extinction," Luo added.
The fossil, now in the collections in Paleontological Museum of Liaoning in China, was discovered and studied by an international team of paleontologists from Paleontological Museum of Liaoning, University of Bonn in Germany, and the University of Chicago.

http://bit.ly/14xYScL

The 'Eyeball Licking' Fad and other Media Scares

A weird news story circulated in June about a trend among Japanese schoolchildren licking each other's eyeballs and supposedly spreading the highly contagious disease pink eye.

Aug 7, 2013 06:12 AM ET // by Benjamin Radford

Many news outlets covered the story as a grave risk; CBS News, for example, warned that this "Japanese 'eyeball licking' trend carries blindness risk."
Not so fast, says Mark Schreiber, an American journalist living in Japan and writer for The Japan Times. In an article for the "Number 1 Shimbum" publication, Schreiber investigated the story and traced it to an "article in Japanese titled "Shogakusei ni gankyuname hentai purei ga dairyuukou" ("The perverted play of eyeball-licking is a hit among primary schoolers"), which appeared on Friday, June 7 on Bucchi News, a site for subculture enthusiasts.
The story's sole informant was "Y," an anonymous teacher at a primary school in Tokyo, who revealed how he had traced an epidemic of pink eye at his school to "hentai (perverted) play" in the form of rampant eyeball licking among students. Notably lacking in attribution and details, the story had all the trappings of an urban legend.
Who was this anonymous teacher in this unknown school and how did he verify that kids were spreading disease by licking each other's eyeballs for chaste sexual kicks? If the trend was sweeping across Japan, where were the hundreds or thousand of other schoolteachers reporting this disturbing public health threat?

It doesn't matter, because it was too good a story not to pass along, facts be damned. It's tempting to suggest that the tabloids were to blame, but as Schreiber wryly notes, "A UK-based medical bulletin board, Medical News Today, even beat out ABC News by one day, running the alarming story under the title "Eyeball Licking (Oculolinctus) Can Be Dangerous, Doctors Warn." The site's readers, including medical professionals, rated the story an average of 4.5 stars out of 5."

But what about the epidemic of tongue-induced pink eye that threatened the health of Japanese schoolchildren everywhere? If eyeball licking wasn't the cause, what was?

Schreiber found no trace of it: "I contacted three Japanese professional organizations, including two opthamological associations and an organization of school clinicians. Queries were also sent to a professor of nursing at a national university and a Yokohama-based ophthalmologist. None of them had the faintest idea of what I was talking about. None knew anything about the rampant spread of disease."

An Irresistible and Creepy Mix

It's easy to see why the story spread so far despite its dubious premise and utter lack of hard journalistic evidence. After all, the subject is eyeball licking! How weird and freaky is that?

If there was ever a subject created specifically to fit the "weird news" category, it's eyeball licking. Just typing those words is creepy.

Another factor is a 24-hour news cycle that emphasizes what's new and rarely offers follow-up reporting on such topics. Countless UFO, ghost and Bigfoot sightings and photographs make a big splash in the news when they first appear but then soon fade away.

When and if the report is finally debunked, it rarely makes the news — partly because for many people a rational explanation is less interesting than the original, mystery-mongering story, and partly because some reporters may not want to admit they were duped in the first place.

Another important reason the story got so much play is that it fits nicely into deep, universal and preexisting concerns about the crazy, dangerous things that kids are up to. It's no accident that the story is about this bizarre practice among schoolchildren specifically. These weren't prisoners or farm workers, but impressionable, wayward children experimenting with some bizarre sexual fetish that even Marquis de Sade would have reservations about.

Kids Today

Worrying about "kids today" is a time-honored pastime among parents and older generations. In the 1990s, the public was told to brace themselves for fires set by impressionable kids who saw their favorite cartoon characters, Beavis and Butthead, committing arson.

Then there were the hordes of teens who were going to injure or kill themselves mimicking pro-wrestling moves they saw on TV. When that dangerous fad didn't materialize, the same concern appeared in other forms. This case has aspects of what sociologists call a moral panic — a concern, often exaggerated, about some perceived threat to life, health or social order.

Every year or two a story circulates widely in the news about some dangerous new trend that parents and teachers need to be aware of, some hidden threat to the health and safety of children based on some strange behavior. Though verified facts and hard numbers are rarely offered in these stories, they are often framed as hidden, emerging fads that are increasing with alarming frequency.

In 2009 experts worried that kids were choking themselves — not to feel light-headed, but instead for sexual pleasure.

According to one news report, "More children and teens than pediatricians realize could be participating in a dangerous, potentially fatal sex act known as autoerotic asphyxiation."

The following year there was widespread parental concern over "sack tapping," another dangerous (and, some claim, potentially fatal) childhood game in which boys slap or punch each others' testicles.

Of course kids do sometimes choke themselves (and each other) and they do sometimes let themselves be kicked or punched in uncomfortable places — just as kids have exchanged smacks, punches and rope burns for ages.

But did any schoolkids really ever lick each other's eyeballs? There's no evidence it's true, though it's possible that some kids somewhere tried it, either intentionally or accidentally, and a teacher saw it and assumed it was a new fad.

Ironically, the story was so widely reported might have caused a few people to try it, in an act folklorists refer to as "ostension." It's the re-enacting of folklore for entertainment and fun, and is the basis for many ghost-hunting expeditions.

This does not mean, of course, that a handful of children may not have engaged in these strange behaviors and been harmed by them. But eyeball licking is, fortunately, one threat that parents can cross off their list.

http://www.eurekalert.org/pub_releases/2013-08/osu-tta080713.php

Treadmill training after spinal cord injury promotes recovery when inflammation is controlled

Researchers observe inflammation in animal models far from trauma location

COLUMBUS, Ohio – New research suggests that treadmill training soon after a spinal cord injury can have long-lasting positive effects on recovery – as long as the training is accompanied by efforts to control inflammation in the lower spinal cord.

The study, in animals, also is among the first to show that spinal cord injuries can create impairments in parts of the cord located many spine segments away from the trauma site.

Researchers observed signs of inflammation in the lumbar region of the spine, at least 10 segments below the mid-back injury, within 24 hours of the trauma. The health of the lumbar region is particularly important to recovery of lower-body movement because it contains important circuitry responsible for walking and other types of locomotion.

The study suggested that controlling inflammation is critical to the success of treadmill training.

Mice that received treadmill training just a few days after injury during a period of heightened inflammation got no lasting benefits. In contrast, animals trained on treadmills when inflammation was minimal regained the use of their hind legs to walk and retained those benefits for up to 42 days.

"We got positive and negative effects with the same intervention, and it's all influenced by inflammation," said D. Michele Basso, professor of health and rehabilitation sciences at The Ohio State University and senior author of the study. "There's so much happening so far away from the injury, and it's all in the heart of where the neural circuits are for locomotion."

The study showed that an enzyme called MMP-9 has a role in causing the lumbar inflammation. Because previous research has linked this enzyme to cancer, experimental drugs that inhibit it are already in the drug-development pipeline. Common antibiotics also could help control this type of inflammation, researchers say.

"The opportunity is there to begin to think about doing this in humans," Basso said.

The research is published in the Aug. 7, 2013, issue of The Journal of Neuroscience.

Basso's lab focuses on studying which activity-related interventions are most likely to improve recovery after a spinal cord injury. She also is a practicing physical therapist who provides treadmill training to patients, but usually months after the injury and in an outpatient rehabilitation setting.

So part of this study was geared toward understanding the window of time after injury that is best for treadmill training. With this intervention, patients' body weight is supported by a harness while therapists move their legs on a treadmill. The theory behind the technique is that sensory information coming from the legs to the lumbar spinal cord will allow nerve cells in this region to relearn how to walk – without needing input from the brain, because those signals are interrupted at the injury site.

The researchers added the inflammation component to the study based on the known properties of the MMP-9 enzyme. MMP-9 is involved in a number of central nervous system processes such as learning and plasticity, tissue remodeling and inflammation, and is regulated by exercise. But it also may create a toxic interaction after spinal cord injury.

Studying normal mice with thoracic spinal cord injuries, the researchers found five times more MMP-9 in the lumbar region seven days after the trauma as well as high levels of inflammation-related chemicals and changes in cell behavior. In contrast, mice that were genetically modified so they could not produce the MMP-9 enzyme had far fewer signs of inflammation in the lumbar region after the injury.

"We found neurovascular reactivity around locomotor networks, which suggests inflammatory processes are occurring 10 segments away from the injury. We've defined the role of MMP-9 in the lumbar cord as proinflammatory," said Christopher Hansen, a doctoral student in the Neuroscience Graduate Studies Program at Ohio State and lead author of the study. "This is significant, to be able to characterize the cellular microenvironment around networks that control locomotion.

"It's also new evidence that a thoracic injury can have effects on the motor system that is 10 or more segments away."

The researchers conducted early treadmill training on the same two types of animals: normal mice and mice that could not activate MMP-9. Control animals of both types received no exercise training.

Only mice deficient in MMP-9 showed significant improvements in walking on the treadmill with their body weight supported and also without any aid after seven days of training. These improvements lasted for four weeks. No other group of mice had the same results.

Researchers also tested the effects of late treadmill training, which started at least 35 days after the injury. The training failed to produce significant walking improvements in any of the animals.
"This was the exact same type of training, and in one group of mice the inflammation was controlled, but even in those mice the late training had no effect," Basso said. "We still don't know exactly when the window is for treadmill training, but this suggests early, and with controlled inflammation. "
The scientists are continuing this work to further clarify the best timing for treadmill training and other exercise-based interventions, as well as other factors that affect the inflammatory response after a spinal cord injury.
Grants from the National Institutes of Health supported the research.
Additional co-authors include Lesley Fisher, Rochelle Deibert and Lyn Jakeman (also in physiology and cell biology) of the Center for Brain and Spinal Cord Repair and Susan White of the School of Health and Rehabilitation Sciences, all at Ohio State; and Haoqian Zhang and Linda Noble-Haeusslein of the University of California, San Francisco. Basso and Hansen also are investigators in the Center for Brain and Spinal Cord Repair.

http://www.eurekalert.org/pub_releases/2013-08/uoc--t1d080713.php

Type 1 diabetes drug strikingly effective in clinical trial

Drug developed by UCSF researcher continues to show promise

An experimental drug designed to block the advance of type 1 diabetes in its earliest stages has proven strikingly effective over two years in about half of the patients who participated in the phase 2 clinical trial. Patients who benefited most were those who still had relatively good control of their blood sugar levels and only a moderate need for insulin injections when the trial began. With the experimental drug, teplizumab, they were able to maintain their level of insulin production for the full two years -- longer than with most other drugs tested against the disease.
Results are published online in the journal Diabetes, and will appear in the November issue of the print edition.
The treatment did not benefit all patients. Some lost half or more of their ability to produce insulin – a drop similar to many of the controls not receiving the drug. Reasons for the different responses are unclear, but likely involve differences in the metabolic condition of the patients and in the severity of their disease at the trial's start, the researchers said.
"The benefits of treatment among the patients who still had moderately healthy insulin production suggests that the sooner we can detect the pre-diabetes condition and get this kind of drug onboard, the more people we can protect from the progressive damage caused by an autoimmune attack," said Jeffrey Bluestone, PhD, co-leader of the research and A.W. and Mary Clausen Distinguished Professor at UC San Francisco, who collaborated in developing the drug.
The clinical trial was led by Kevan Herold, MD, PhD, a professor of immunobiology and deputy director for translational science at Yale University. He and Bluestone have collaborated on four previous clinical trials of the experimental drug. "We are very excited by the efficacy of the drug," Herold said. "Some of our patients and families have described a real impact on their diabetes."
The results underscore the importance of diagnosing and treating diabetes in its earliest stages, the researchers said. Current treatment studies include "pre-diabetes" patients who have abnormal blood sugar levels but do not need to take insulin.
Formerly referred to as juvenile diabetes because it disproportionately strikes children, type 1 diabetes is caused by an autoimmune condition in which the body's immune system destroys insulin-producing beta cells in the pancreas. Even with insulin treatments, the blood glucose levels fluctuate abnormally, and as the disease progresses, diabetes increases the risk of kidney failure, heart disease and other serious disorders.
According to the Juvenile Diabetes Research Foundation, as many as 3 million American have type 1 diabetes, and each year, more than 15,000 children and 15,000 adults are diagnosed with the disease in the United States. For reasons still unknown, the incidence of type 1 diabetes is increasing, and the age of onset is decreasing.
Teplizumab is one of a number drugs under active investigation to control autoimmune reactions. Teplizumab uses an antibody targeted against a molecule called CD3 to bind to the immune system¹s T-cells and restrain them from attacking beta cells.
Immunotherapies are designed to treat organ transplant rejection and autoimmune diseases, including multiple sclerosis, Crohn's disease, rheumatoid arthritis, and asthma. The use of these agents in type 1 diabetes is emerging based on work in preclinical models and clinical trials.
The journal's print edition will include a commentary by Jay S. Skyler, MD, chairman of the NIH-funded Type 1 Diabetes Trial Net, an international network of researchers that also studies teplizumab for prevention of type 1 diabetes. Skyler writes that the new results make a compelling case for U.S. Food and Drug Administration approval to launch a much larger-scale, phase 3 clinical trial of the drug's effectiveness.

Bluestone, an immunologist who is now executive vice chancellor and provost at UCSF, developed teplizumab in collaboration with Ortho Pharmaceuticals in 1987. He is a leader in research that aims to understand how and why the immune system attacks the body's own tissues and organs, and to develop drug strategies to eliminate the autoimmune response without producing severe side effects.

The study focused on 52 participants, most of whom were less than 14 years old, who had been diagnosed with "new-onset type 1 diabetes" within eight weeks of the trial's start. All 52 were treated with the experimental drug for two weeks at diagnosis and again one year later, and their capacity to produce their own insulin to control their blood sugar was compared with a non-treated group.

Because the participants received daily insulin injections before and throughout the trial, researchers instead monitored their blood levels of C-peptide, a molecule produced in the pancreas at the same rate as insulin.

This research was a project of the Immune Tolerance Network (NIH contract #NO1 AI15416), an international clinical research consortium supported by the National Institute of Allergy and Infectious Diseases and the Juvenile Diabetes Research Foundation. It also was supported by NIH grants UL1 RR024131 and UL1 RR024139.

Coauthors on the paper and collaborators in the clinical trial with Herold and Bluestone include Stephen E. Gitelman, MD, UCSF; Mario R. Ehlers, PhD, and Peter H. Sayre, MD, of the Immune Tolerance Network (ITN), San Francisco; Peter A. Gottlieb, MD, University of Colorado; Carla J. Greenbaum, MD, Benaroya Research Institute, Seattle; William Hagopian, MD, Pacific Northwest Diabetes Research Institute, Seattle; Karen D. Boyle, MS, and Lynette Keyes-Elstein, DrPh, Rho Federal Systems Division, Chapel Hill; Sudeepta Aggarawal, PhD, and Deborah Phippard, PhD, ITN, Bethesda; James McNamara, MD, National Institutes of Allergy and Infectious Diseases.

Conflict of interest statement: Jeffrey Bluestone has a patent on the teplizumab molecule. Kevan Herold has received grant support from MacroGenics, Inc., a company that owns rights to the drug. The paper can be found online at diabetes.diabetesjournals.org.

http://www.sciencedaily.com/releases/2013/08/130806203325.htm

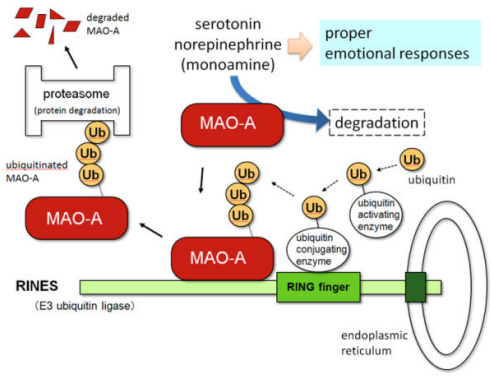
Brain Molecule Regulating Human Emotion, Mood Uncovered

A RIKEN research team has discovered an enzyme called Rines that regulates MAO-A, a major brain protein controlling emotion and mood.

The enzyme is a potentially promising drug target for treating diseases associated with emotions such as depression.

Monoamine oxidase A (MAO-A) is an enzyme that breaks down serotonin, norephinephrine and dopamine, neurotransmitters well-known for their influence on emotion and mood. Nicknamed the "warrior gene," a variant of the MAOA gene has been associated with increased risk of violent and anti-social behavior.

While evidence points to a link between MAO-A levels and various emotional patterns, however, the mechanism controlling MAO-A levels in the brain has remained unknown.



A RIKEN research team has discovered an enzyme called Rines that regulates MAO-A, a major brain protein controlling emotion and mood. The enzyme is a potentially promising drug target for treating diseases associated with emotions such as depression. **(Credit: RIKEN)**

Now, a research team headed by Jun Aruga at the RIKEN Brain Science Institute has shown for the first time that a ligase named Rines (RING finger-type E3 ubiquitin ligase) regulates these levels. Their research shows that mice without the Rines gene exhibit impaired stress responses and enhanced anxiety, controlled in part through the regulation of MAO-A levels. The study is published today in Journal of Neuroscience.

As the first study to demonstrate regulation of MAO-A protein via the ubiquitin proteasomal system, this research presents a promising new avenue for analyzing the role of MAO-A in brain function. Further research promises insights into the treatment of anxiety, stress-related disorders and impaired social functions.

Kabayama et al. Rines E3 Ubiquitin Ligase Regulates MAO-A Levels and Emotional Responses. Journal of Neuroscience, 2013 DOI: 10.1523/JNEUROSCI.5717-12.2013

http://www.eurekalert.org/pub_releases/2013-08/bu-ssw080813.php

Study shows who survives Burkitt lymphoma

Proposed score delineates risk, prognosis

A new study in the journal Cancer that tracked survival of more than 2,200 adults over the last decade with a highly aggressive form of lymphoma finds that with notable exceptions, medicine has made substantial progress in treating them successfully.

To help doctors and researchers better understand who responds well to treatment and who doesn't, the study authors used their findings to create a stratified risk score of patient prognosis.

Burkitt Lymphoma is not a common lymphoma but it is especially aggressive.

The apparent progress doctors have made over the last two decades has come, unlike with many other cancers, with little guidance about how to treat different patients or what outcomes to expect.
The same regimen of intensive chemotherapy and the monoclonal antibody rituximab are recommended for most patients.
"There was little available for Burkitt lymphoma in terms of prognostic factors, indicators or scoring," said Dr. Jorge Castillo, an Assistant Professor of Medicine at the Warren Alpert Medical School of Brown University and a hematology/oncology specialist at Rhode Island Hospital. He's the lead author of the study, which first appeared online July 30.
Castillo wanted to better understand the prognosis of patients, so he and his co-authors looked at 11 years of patient records in the Surveillance, Epidemiology and End Results (SEER) database, which keeps patient demographic and outcomes data from 18 areas around the country.
They analyzed survival rates among 2,284 patients by factors including age, race, stage of the cancer at diagnosis, and in what region of the body the cancer struck.
What they found is that while survival rates have risen substantially overall, outcomes have not improved much for patients who are older than 60, black, or whose cancer is diagnosed at a late stage. They used these risk factors to create a simple new risk score that allowed them to make meaningful distinctions about prognosis. Patients with the lowest score had a better than 7 in 10 chance of survival with treatment, while those with the highest score have a less than 3 in 10 chance of surviving.

Improved survival, for most.

Age makes a big difference in survival, Castillo and his co-authors found. Their analysis yielded the calculation that patients over 80 years old have nearly 5 times the risk of dying from the cancer as people aged 20-39. Patients aged 60-79 had twice the risk of dying as the youngest patients and those aged 40-59 had a risk 1.5 times greater than those aged 20-39.
Risk of death climbed similarly with the stage of cancer. Stage IV patients had a 2.4 times greater risk of dying than those at Stage I. Stage III patients had a 1.5 times greater risk.
Race was also a factor, although to a more mild degree. Hispanics and whites had similar risk levels but black people, who were 9.3 percent of the patients, had a 1.6 times higher risk of death.
These same risk factors are also evident in whether patients have seen improved survival over time, for instance as intensive chemotherapy and later rituximab have gained prevalence.
In 1998, the survival rate was fairly uniform for all age groups, Castillo and his colleagues found: 34.7 percent overall. As of 2007, survival had risen to 62.1 percent for the youngest adult patients, but patients over 60 have only drifted slightly upward to a survival rate of 43.5 percent.
Among patients of different ethnic backgrounds, there is a similarly widening gap: Survival among non-Hispanic whites rose from 31.7 percent to 50.9 percent and among Hispanics from 22.7 percent to 47.1 percent. Among blacks, however, survival has remained low and flat: from 28.8 percent in 1998 to only 29.9 percent in 2007.
Castillo said he does not know with certainty from the study or from the medical literature why blacks fare relatively poorly, but in his study he was able to control for socioeconomic status and the disparity was independent of it.

Prognostic score

Using the significant risk factors they discovered, Castillo and co-authors Dr. Eric Winer, also of RIH and Dr. Adam Olszewski of Memorial Hospital in Pawtucket created the risk score in which an age 40-59 or being black adds 1 point, being age 60 to 79 or stage III or IV adds two points, and age over 80 adds 4 points.
Doing so separated the 2,284 patients into roughly equal groups with a wide range of 5-year relative survival rates (relative to the likelihood of survival of a similar person without the disease).
Among the groups those with a score of 0 to 1 had a 71 percent relative survival rate. A score of 2 reduced the rate to 55 percent, a score of 3 had a 41 percent relative survival and for those at 4 or higher the rate was merely 29 percent.
Castillo said there are several applications of the score, including helping doctors, patients and their families understand what to expect, and to evaluate whether intensive regimens of difficult therapy are truly desirable, compared to possible alternatives. The score can also inform researchers about how to design clinical trials of treatments of the disease.
"It helps to identify people who don't benefit from what we're doing right now," he said.
But thankfully for many people, care appears to be working.

http://bit.ly/17BaxlL

News in Brief: Camels implicated as possible hosts of MERS virus | Body & Brain

Antibodies to mysterious pathogen found in animals in Oman, Canary Islands

By Tina Hesman Saey

Camels may be intermediate hosts of a mysterious and deadly respiratory virus related to SARS that has sickened 94 people, killing 46, in parts of the Middle East, Europe and northern Africa.

Last year, the virus — now known as the Middle East respiratory syndrome, or MERS, coronavirus — revealed itself to scientists after a few people became sick with severe pneumonia (SN: 3/23/13, p. 5). After examining the virus's DNA, researchers discovered that the pathogen is related to similar viruses that infect bats, but none of the sick people had any known contact with bats.

Now, Chantal Reusken of the National Institute for Public Health and the Environment in Bilthoven, the Netherlands, and colleagues report August 9 in the Lancet Infectious Diseases that 50 retired racing camels from Oman carry antibodies against the MERS coronavirus in their blood. The result suggests that the animals have been exposed to MERS or a closely related virus.

The team also found low levels of antibodies against the virus in the blood of dromedary camels from the Canary Islands. Neither Oman nor the Canary Islands has reported human cases of the disease. But anecdotal reports suggest that some of the sick people from other countries may have been around camels or goats before falling ill.

The results could mean that camels and camel relatives such as goats may be intermediaries in a chain of infection that sometimes ends with humans or that a virus similar to MERS has been in camels for a long time and recently gained the ability to infect people.

Editor's Note: This story was updated on August 9, 2013, to correct the journal the paper appears in.

C. B. E. M. Reusken et al. Middle East respiratory syndrome coronavirus neutralizing serum antibodies in dromedary camels: a comparative serological study. The Lancet Infectious Diseases. Published online August 9, 2013. doi: 10.1016/S1473-3099(13)70164-6. [Go to]

http://www.eurekalert.org/pub_releases/2013-08/uosd-tdb080913.php

The day before death: A new archaeological technique gives insight into the day before death

The day before the child's death was not a pleasant one, because it was not a sudden injury that killed the 10-13 year old child who was buried in the medieval town of Ribe in Denmark 800 years ago.

The day before death was full of suffering because the child had been given a large dose of mercury in an attempt to cure a severe illness.

This is now known to chemist Kaare Lund Rasmussen from University of Southern Denmark – because he and his colleagues have developed a new methodology that can reveal an unheard amount of details from very shortly before a person's death. Mercury is of particular interest for the archaeologists as many cultures in different part of the world have been in contact with this rare element.

"I cannot say which diseases the child had contracted. But I can say that it was exposed to a large dose of mercury a couple of months before its death and again a day or two prior to death. You can imagine what happened: that the family for a while tried to cure the child with mercury containing medicine which may or may not have worked, but that the child's condition suddenly worsened and that it was administered a large dose of mercury which was, however, not able to save its life", says Kaare Lund Rasmussen.

The detailed insight into the life of the child did not come from analyses of the child's bones. Instead Kaare Lund Rasmussen and his colleagues have developed a method to extract information from the soil surrounding the body of the dead child in the cemetery in Ribe, Denmark.

"When the body decays in the grave a lot of compounds are released to the surrounding soil – by far most of them organic compounds. Also most of the inorganic elements are transformed to other compounds and later removed by the percolating groundwater throughout the centuries that follows. If we can localize an element in the soil in the immediate vicinity of the skeleton which is not normally found in the soil itself, we can assume that it came from the deceased and this can tell us something about how the person lived. We are not interested in death, but in the life before death", Kaare Lund Rasmussen explains.

Mercury in particular is worth looking for, he explains. This element is very rare in normal soil, but has been used in several cultures worldwide, and it is therefore expected sometimes to be found in archaeological excavations in a variety of places like Italy, China, Central America and - as it appears - also in medieval Denmark. In medieval Europe mercury was used for centuries in the colour pigment cinnabar, which was used for illuminating manuscripts by medieval monks, and since Roman times mercury was widely used as the active ingredient in medicine administered against a variety of diseases.

"Mercury is extremely toxic and surely some died from mercury poisoning and not the ailment it was meant to cure. Treatment with mercury was practiced well into the 1900's, where for instance the Danish novelist Karen Blixen (Seven Gothic Tales) received treatment in 1914", says Kaare Lund Rasmussen.
"Concerning past archaeological excavations it is appalling to think about all the soil that archaeologists have wheel barrowed away for more than a century – if we had samples of this soil, we would have access to a lot of important information", he says.
The soil samples must be taken precisely in the position of the original tissue, e.g. inner organs or muscle tissue where there is now only soil to be seen.
"At the position of a kidney, which is now completely decayed, compounds originally sitting in the kidney tissue are now part of the soil, if it has not been transported away by the groundwater. If there was mercury present in the kidney at the time of death it would have been transformed rapidly to mercury sulphide which is very immobile and undissolvable in water. So in this way we can obtain information about the deceased even though we do not analyse the bones", Kaare Lund Rasmussen explains.
In the town of Ribe the chemists have been assisted by anthropologists to take soil samples from the places originally occupied by the mercury poisoned child's lungs, kidneys, liver and muscle tissue. As the half-life of mercury varies between the different tissue types, Kaare Lund Rasmussen can ascertain when the body was last exposed to mercury prior to death.
The mercury concentration is for instance excreted very fast from the lungs, within hours or at the most a couple of days, and it is therefore a question of hours or at the most a couple of days before most of the mercury has vanished from the lungs after inhaling mercury vapour.
"When we found high mercury concentrations in the soil that had once been the lungs of the child, we could conclude that the child probably was exposed to mercury within the last 48 hours or so before its death" says Kaare Lund Rasmussen.
It is also possible to test the bones for their content of excess mercury, and this technique has been used by archaeologists for several years now.
"But there are certain limitations to what the bones can reveal; while the soil give insight into the last months and days before death, the bones can only give information about the mercury exposure from ca. ten to three years prior to death", Kaare Lund Rasmussen explains.
Kaare Lund Rasmussen and his colleagues have used their newly developed sampling technique on soil samples from 19 medieval burials in the cemeteries Lindegaarden in Ribe and Ole Wormsgade in Horsens, Denmark.
The results are published in the journal Heritage Science, 2013,1:16.
The work is part of a new research project,"People in Ribe in a 1000 years", supported by the VELUX Foundation. Besides the chemists from University of Southern Denmark, anthropologists from University of Southern Denmark and archaeologists from Sydvestjyske Museer also participate in the research.

http://www.sciencedaily.com/releases/2013/08/130810063014.htm

Cultural Mythologies Strongly Influence Women's Expectations About Being Pregnant

Morning sickness, shiny hair, and bizarre and intense cravings for pickles and ice cream -- what expectations do pregnant women impose on their bodies, and how are those expectations influenced by cultural perspectives on pregnancy?

Danielle Bessett, an assistant professor of sociology at the University of Cincinnati, will present her research on this issue at the 108th Annual Meeting of the American Sociological Association.
Although previous studies have indicated that women primarily rely on their health care providers and pregnancy guides to find out what to expect when they're expecting, Bessett's research, titled, "Expecting Embodiment: Pregnancy Symptoms and the Cultural Mythologies of Pregnancy," found that pregnant women are also strongly influenced about their pregnancies by common hearsay in their social circles and in entertainment media. Bessett calls this phenomenon "pregnancy mythologies" -- fragmentary, contradictory, and elusive forms of knowledge.
The study relied on interviews with 64 pregnant women in the greater New York metropolitan area from 2003-2006. Bessett noted that all 64 women confronted mythologies in pregnancy.
"In contrast to survey research that asks women to identify information sources that help them make specific decisions, in-depth interviews such as mine reveal a more complex web of taken-for-granted assumptions that women bring to pregnancy -- a condition commonly represented in both fictional and reality television, films, commercials, and other entertainment media," Bessett said. "My research shows that we may underestimate the extent to which all of us hold understandings of pregnancy built incrementally through a succession of ephemeral encounters over our lifetimes and the extent to which those understandings affect us. It is important

to recognize this phenomenon because it may result in different perspectives on what we can take for granted about pregnancy which may affect communication between women and their health care providers."
According to Bessett, some women drew heavily from ethnic-religious traditions. Some had little or no personal experience with pregnancy, while others had complicated reproductive histories. "Depending on these varied biographical and structural locations, women affirmed, grieved, critiqued, and contested key aspects of pregnancy mythology," Bessett said.
Bessett found that most women tended to minimize the influence of pregnancy mythologies when asked directly about information sources they trusted most. It was only when pushed to explain how they came to hold specific expectations for what would happen during their pregnancies that women referenced entertainment media sources. Interestingly, women often found themselves without an explanation for how they learned about what "normally happened" in pregnancy.
Through her interviews, Bessett found that in some cases, women were alarmed when they weren't experiencing symptoms popularly associated with pregnancy, such as morning sickness, fearing that something might be wrong with the health of the fetus.
"Whether pleasurable, inconvenient or debilitating, pregnancy symptoms are not simply treated as pregnancy side-effects in our culture, but rather as a significant connection to the fetus and fetal subjectivity," Bessett said. For example, one mother said that her intense vomiting resulted because her baby didn't like what she ate. Another explained that her craving was due to her baby "liking fried chicken."
"Many symptoms were frequently seen as tangible manifestations of the fetus's desires, needs, or personal characteristics," Bessett said.
While issues such as nausea, cravings, and labor pain were prominent in mythology, Bessett said that other ailments such as exhaustion, insomnia, gas, headaches, and swollen ankles weren't as popularly linked or discussed.
"Whether it's because they are somewhat rare (like pregnancy-related nosebleeds) or because they concern parts of the body that are not 'polite' to talk about (such as hemorrhoids), some symptoms are not typically portrayed in entertainment media narratives on pregnancy, nor were they symptoms that friends and family frequently shared with women in advance of their first pregnancy," she said.

Study Participants

The findings from the study are based on interviews with 64 pregnant women in the greater New York metropolitan area from 2003-2006. All participants were enrolled in prenatal care at the time of the interviews. Just over half of the participants were expecting their first child. Twenty-three of the participants sought care from public, hospital-based clinics, while the other participants received care from private practices. The women represented a range of socioeconomic and racial/ethnic backgrounds.
Just over half of the women interviewed identified as white; 12 as black; 14 as Hispanic/white; two as Hispanic/black; two as Asian; and one as other/mixed race. The sample was economically diverse, with a third of the women reporting household incomes of less than $40,000 and just under half reporting household incomes of $80,000 or higher.
Participants agreed to at least two interviews -- one before and one after giving birth, yet most participants took part in three interviews.
The research was supported by funding from a National Science Foundation Dissertation Improvement Grant; the Dr. Mary P. Dole Medical Fellowship from the Mount Holyoke College Alumnae Association; and the Charlotte Ellerston Social Service Postdoctoral Fellowship in Abortion and Reproductive Health.

http://www.sciencedaily.com/releases/2013/08/130810063715.htm

Combined Therapy Could Repair and Prevent Damage in Duchenne Muscular Dystrophy, Study Suggests

Results from a clinical trial of eteplirsen, a drug designed to treat Duchenne muscular dystrophy, suggest that the therapy allows participants to walk farther than people treated with placebo and dramatically increases production of a protein vital to muscle growth and health.

The study, led by a team in The Research Institute at Nationwide Children's Hospital, is the first of its kind to show these results from an exon-skipping drug -- a class of therapeutics that allows cells to skip over missing parts of the gene and produce protein naturally.
"I've been doing this for more than 40 years and this is one of the most exciting developments we've seen," says Jerry Mendell, MD, lead author of the study and director of the Center for Gene Therapy at Nationwide Children's. "It offers great hope to patients with Duchenne muscular dystrophy and their families."
The research, which appears online Aug. 1 in the journal Annals of Neurology, is the first study from a double-blind controlled randomized trial of an exon-skipping agent to provide conclusive proof based on the standard

six-minute walk test used to measure muscle function in patients with Duchenne muscular dystrophy (DMD), the most common form of muscular dystrophy in children.
About one in every 5,000 male births in the U.S. has the disorder, which usually leaves patients unable to walk on their own by age 12. Children with DMD have a mutation that cripples the body's ability to produce a protein called dystrophin, which helps absorb the shock or energy that's created when a muscle contracts. Without it, that released energy injures muscle fibers. Over time, the muscle degenerates, scar tissue builds up and fat slowly replaces the dead muscle.
The exact mutation varies from patient to patient but in 65 percent of cases, the dystrophin gene is missing large sections of DNA called exons, which carry the instructions for protein production. Accompanying this type of mutation is a spontaneously occurring reaction that enables muscle cells to skip over the deleted sections and produce smaller -- but functional -- versions of protein.
Eteplirsen, manufactured by Sarepta Therapeutics in Cambridge, Mass., mimics this naturally occurring phenomenon, allowing cells to skip over exon 51 in the dystrophin gene. About 13 percent of patients with the disorder have this mutation. Nationwide Children's began the phase II trial of eteplirsen in August 2011, enrolling 12 boys age 7 to 12 years.
Participants received the drug via weekly IVs, with one group getting a 30 mg/kg dose and another group receiving 50 mg/kg. A control group received a placebo. Participants completed a six-minute walk test at the outset and again at weeks 12, 24 and 48. Muscle biopsies were also taken when the study began and again at those intervals to measure for dystrophin-positive muscle fibers.
Although there was no dystrophin production at 12 weeks, participants showed a 23 percent increase in dystrophin-positive muscle fibers by the 24-week mark. The striking improvement and lack of side effects prompted researchers to switch participants in the placebo group to the drug. By week 48, participants had a 52 percent increase in dystrophin-positive muscle fibers and were able to walk 67.3 meters farther than the placebo group on the six-minute walk test.
Although the results are promising, Dr. Mendell is quick to note that the small study leaves many questions unanswered. For example, researchers would like to know how the drug affects dystrophin production in muscles throughout the limbs and whether some muscles may get a bigger boost than others.
"We know that if you have an area that is not expressing dystrophin, the membrane will be fragile and vulnerable to activity-related degeneration," says Dr. Mendell, who also is director of the Neuromuscular Disorders program at Nationwide Children's and a professor of pediatrics in The Ohio State University College of Medicine. "There may be factors that lead to preferential localization of the dystrophin production. That's one of many issues we'd like to investigate further."
That information would help researchers determine what dose and frequency of administration would have the best benefit.
"Another big issue is whether patients who start to produce dystrophin will plateau quickly or if the drug would continue to show benefits over the long term," Dr. Mendell says.
Sarepta Therapeutics, which funded the study, plans to submit a New Drug Application to the Food and Drug Administration early next year. If approved, eteplirsen would be the first therapy for DMD to target the underlying cause of the disease.
Jerry Mendell, Louise R Rodino-Klapac, Zarife Sahenk, Kandice Roush, Loren Bird, Linda P Lowes, Lindsay Alfano, Ann Maria Gomez, Sarah Lewis, Janaiah Kota, Vinod Malik, Kim Shontz, Christopher M Walker, Kevin M Flanigan, John R Kean, Hugh D Allen, Chris Shilling, Kathleen R Melia, Peter Sazani, Jay B Saoud, Edward M Kaye. Eteplirsen for the treatment of duchenne muscular dystrophy. Annals of Neurology, 2013; DOI: 10.1002/ana.23982

http://www.sciencedaily.com/releases/2013/08/130810063010.htm

People Have More Empathy for Battered Dogs Than Human Adult, but Not Child, Victims

People have more empathy for battered puppies and full grown dogs than they do for some humans -- adults, but not children, finds new research to be presented at the 108th Annual Meeting of the American Sociological Association.

"Contrary to popular thinking, we are not necessarily more disturbed by animal rather than human suffering," said Jack Levin, the Irving and Betty Brudnick Professor of Sociology and Criminology at Northeastern University. "Our results indicate a much more complex situation with respect to the age and species of victims, with age being the more important component. The fact that adult human crime victims receive less empathy than do child, puppy, and full grown dog victims suggests that adult dogs are regarded as dependent and vulnerable not unlike their younger canine counterparts and kids."

In their study, Levin and co-author Arnold Arluke, a sociology professor at Northeastern University, considered the opinions of 240 men and women, most of whom were white and between the ages of 18-25, at a large northeastern university. Participants randomly received one of four fictional news articles about the beating of a one-year-old child, an adult in his thirties, a puppy, or a 6-year-old dog. The stories were identical except for the victim's identify. After reading their story, respondents were asked to rate their feelings of empathy towards the victim.

"We were surprised by the interaction of age and species," Levin said. "Age seems to trump species, when it comes to eliciting empathy. In addition, it appears that adult humans are viewed as capable of protecting themselves while full grown dogs are just seen as larger puppies."

Interestingly, the researchers found that the difference in empathy for children versus puppies was statistically non-significant.

As for considering the opinions of 240 college students, Levin said it is common practice to use homogenous samples for studies such as his that center around an experiment. "Unlike survey research, experiments usually employ a homogenous sample in order to establish a cause and effect relationship rather than to generalize a large population," Levin said. "However, there is really no reason to believe that our results would differ very much nationally, particularly among college students."

While the study focused on dogs and humans, Levin thinks the findings would be similar for cats and people as well. "Dogs and cats are family pets," he said. "These are animals to which many individuals attribute human characteristics."

http://www.eurekalert.org/pub_releases/2013-08/asa-seb080913.php

Study examines beliefs about who should pay for dates

Men's and women's beliefs about who should pay for dates during courtship

NEW YORK CITY — Chapman University's David Frederick will present new research at the 108th Annual Meeting of the American Sociological Association that examines men's and women's beliefs about who should pay for dates during courtship, and how couples actually go about splitting expenses. The paper, "Who Pays for Dates? Following versus Challenging Conventional Gender Norms," contains survey data from more than 17,000 participants; a quarter of whom also provided written commentaries to explain their beliefs and actions regarding paying for dates.

"The motivation for the study was to understand why some gendered practices are more resistant to change than others; for example, the acceptance of women in the workplace versus holding onto traditional notions of chivalry," said Frederick, who co-authored the study with Janet Lever, of California State University, Los Angeles, and Rosanna Hertz, of Wellesley College.

Conventional notions of chivalry dictate that on a "date," the man pays, whereas egalitarian ideals suggest gender should not determine who pays for the entertainment expenses. This research examines the extent to which people embrace or reject these competing notions after nearly 50 years of feminism. It is known that most marriages (8 in 10) today are based on sharing the breadwinner's burden, so one question was whether that role is shared prior to marriage and, if so, how early in the dating process.

Consistent with conventional norms, most men (84 percent) and women (58 percent) reported that men pay for most expenses, even after dating for a while. Over half (57 percent) of women claim they offer to help pay, but many women (39 percent) confessed they hope men would reject their offers to pay, and 44 percent of women were bothered when men expected women to help pay. Nearly two-thirds (64 percent) of men believed that women should contribute to dating expenses, and many feel strongly about that: Nearly half of men (44 percent) said they would stop dating a woman who never pays. A large majority of men (76 percent), however, reported feeling guilty accepting women's money.

In terms of behavior, even if men are paying a larger proportion of expenses, 4 in 10 men and women agreed that dating expenses were at least partially shared within the first month, and roughly three-fourths (74 percent of men, 83 percent of women) reported some sharing of expenses by six months. These data illustrate which people are resisting or conforming to conventional gender norms in one telling aspect of dating that historically was related to the male's displaying benevolent sexism and dominance as a breadwinner. Whereas young men and women in their 20s were the most likely to endorse egalitarian practices, this is a mass culture phenomenon — the same basic patterns were seen regardless of daters' ages, income, or education. Although there is evidence of resistance to change, the data suggest that the deep-rooted courtship ritual around who pays is also changing along with the transformation of the relative material and social power of women and men.

http://nyti.ms/14A8NeU

Autism's Unexpected Link to Cancer Gene

Some with autism have mutated cancer or tumor genes that apparently caused their brain disorder

By GINA KOLATA

Researchers studying two seemingly unrelated conditions — autism and cancer — have unexpectedly converged on a surprising discovery. Some people with autism have mutated cancer or tumor genes that apparently caused their brain disorder.

Ten percent of children with mutations in a gene called PTEN, which causes cancers of the breast, colon, thyroid and other organs, have autism. So do about half of children with gene mutations that can lead to some kinds of brain and kidney cancer and large tumors in several organs, including the brain. That is many times the rate of autism in the general population.

"It's eerie," Evan Eichler, a professor of genome science at the University of Washington, said about the convergence.

He and others caution that the findings apply to only a small proportion of people with autism; in most cases, the cause remains a mystery. And as with nearly all genetic disorders, not everyone with the mutations develops autism or cancer, or other disorders associated with the genes, like epilepsy, enlarged brains and benign brain tumors.

But researchers say the findings are intriguing, given that there are no animals that naturally get autism, no way of analyzing what might cause autism in developing brains and no cure. The newly discovered link has enabled scientists to genetically engineer mice with many symptoms of the human disorder.

And it has led to the first clinical trial of a treatment for children with autism, using the drug that treats tumors that share the same genetic basis.

Richard Ewing of Nashville, a 10-year-old who has a form of autism caused by a tumor-causing gene, is among those in the new study. His parents, Alexandra and Rick Ewing, know he is at risk for tumors in the brain, heart, kidney, skin and eyes. But that bad news was tempered by his eligibility for the clinical trial, which has only just started.

"There is a big difference between us and the rest of the autism community," Mr. Ewing said. "We have an honest-to-God genetic diagnosis."

Not everyone agrees that the discovery is so promising. Steven McCarroll, a geneticist at Harvard, notes that autistic children with the cancer gene mutation have "a brain that is failing in many ways." Autism in these children could be a manifestation of a general brain malfunction, he said, adding, "The fact that autism is one of the many neurological problems that arise in these patients doesn't necessarily tell us anything penetrating about the social and language deficits that are specific to autism."

But other scientists who are not involved in the research that produced these findings say the work is changing their understanding of autism and why it develops. Like cancer, autism can involve unregulated growth of cells, in this case neurons in the brain.

Jonathan Sebat, chief of the Center for Molecular Genomics of Neuropsychiatric Diseases at the University of California, San Diego, describes the parallels between cancer and autism as "quite uncanny."

"We haven't solved it all; we have only solved a tiny bit," he added. "But the small bit we solved has been very illuminating."

It was Dr. Charis Eng, a cancer geneticist at the Cleveland Clinic, who first noticed a surprising incidence of autism in children whose parents had the PTEN mutation (pronounced p-10). Eventually, investigators discovered that the rate of autism was 10 percent, about 10 times what would normally be expected.

At the same time, researchers found that another genetic disorder was even more likely to result in autism. That disorder, tuberous sclerosis, increases the risk for kidney cancer and a type of brain cancer; half of tuberous sclerosis patients had autism.

Although PTEN and tuberous sclerosis genes are not the same, they are part of the same network of genes that put a brake on cell growth. Disabling PTEN or one of the tuberous sclerosis genes releases that brake. One result can be cancer or tumors. Another can be abnormal wiring of nerve fibers in the brain and autism.

Dr. Mustafa Sahin of Boston Children's Hospital decided to test whether drugs used to treat tumors caused by tuberous sclerosis gene mutations might also treat autism in people with the same mutated genes.

He started with mice, deleting tuberous sclerosis genes in their cerebellums. Nerve fibers in the animals' brains grew wildly, and the mice had unusual behaviors, reminiscent of autism. They had repetitive movements and groomed themselves constantly, so much that they sometimes rubbed their skin raw. And unlike normal mice, which prefer other mice to an inanimate object, these mice liked a plastic cup just as much.

But rapamycin, which targets the tuberous sclerosis gene and blocks a protein involved in cell division, changed the animals. They no longer compulsively groomed themselves, and they no longer liked the plastic cup as much as a live mouse. The animals did better on tests of learning and memory, and the growth of nerve fibers in their brains was controlled. Before treatment, for example, the mice had trouble learning that an underwater platform had been moved. Afterward, they learned its new location.

Now Dr. Sahin is giving a similar drug, everolimus, to autistic children with a tuberous sclerosis gene mutation, asking if it can improve their mental abilities. Richard is among the children. Each child takes the drug or a placebo for six weeks. The study is scheduled to be completed by December 2014.

While Dr. Eng started with cancer gene mutations and discovered a link to autism, Dr. Eichler, of the University of Washington, started with autism and found a connection to cancer genes.

He focused on what he calls "out of the blue autism," which occurs with no family history, recruiting 209 families with autistic children.

He saw a striking genetic difference. Compared with their parents and normal siblings, the autistic children had two to three times as many mutations that disabled a gene. The mutated genes were often part of a pathway that controls cells growth. At first, the researchers thought the pathway was ubiquitous, and its link to autism was murky.

"We were a bit bummed," Dr. Eichler said. "Then I said: 'Wait, some of those genes are cancer genes.' "

But he does not yet know whether these children with autism are also at risk for cancer.

"It's obviously a significant issue," Dr. Eichler said. "But we need to let the science nail it first."

The Ewings, whose son is in the autism clinical trial, have learned to live with the tumor threat. For now, their biggest problems are dealing with Richard's autism.

When Richard's parents heard about Dr. Sahin'sstudy, they immediately signed him up, though it meant traveling to Boston from Nashville nine times in six months. They had not dared to take their son on planes before, worried that he could not handle the security lines and crowded airports.

But the study was too important to pass up, Mr. Ewing said.

"Traveling with a kid who can't talk, who has food issues, who is not patient: we hadn't really done these things," Mr. Ewing said.

They hope the drug will make a difference.

"We always thought Richard has a lot going on in his brain," Mrs. Ewing said. "We feel there is a lot of untapped potential."

For Andrew and Lucy Dabinett's 9-year-old son, Tommy, whose autism is caused by a PTEN gene mutation, there are no clinical trials as of yet.

Tommy, who lives with his family in Rye, N.Y., has a limited vocabulary, flaps his arms, rocks back and forth, and needs diapers.

When he was 3, a doctor told his parents that he had a PTEN mutation and that in addition to autism, he had a high risk of cancer.

"Of course it is terrifying," Ms. Dabinett said. "But I already knew there was something terribly wrong with my child. I just needed an answer."

"Honestly," she said, "it was a relief to have an answer."