http://www.eurekalert.org/pub_releases/2013-12/du-njt122013.php

Not just the Koch brothers: New Drexel study reveals funders behind the climate change denial effort

Study exposes the organizational underpinnings and funding behind the powerful climate change countermovement

A new study conducted by Drexel University's environmental sociologist Robert J. Brulle, PhD, exposes the organizational underpinnings and funding behind the powerful climate change countermovement. This study marks the first peer-reviewed, comprehensive analysis ever conducted of the sources of funding that maintain the denial effort.

Through an analysis of the financial structure of the organizations that constitute the core of the countermovement and their sources of monetary support, Brulle found that, while the largest and most consistent funders behind the countermovement are a number of well-known conservative foundations, the majority of donations are "dark money," or concealed funding.

The data also indicates that Koch Industries and ExxonMobil, two of the largest supporters of climate science denial, have recently pulled back from publicly funding countermovement organizations. Coinciding with the decline in traceable funding, the amount of funding given to countermovement organizations through third party pass-through foundations like Donors Trust and Donors Capital, whose funders cannot be traced, has risen dramatically.

Brulle, a professor of sociology and environmental science in Drexel's College of Arts and Sciences, conducted the study during a year-long fellowship at Stanford University's Center for Advanced Study in the Behavioral Sciences. The study was published today in Climatic Change, one of the top 10 climate science journals in the world.

The climate change countermovement is a well-funded and organized effort to undermine public faith in climate science and block action by the U.S. government to regulate emissions. This countermovement involves a large number of organizations, including conservative think tanks, advocacy groups, trade associations and conservative foundations, with strong links to sympathetic media outlets and conservative politicians.

"The climate change countermovement has had a real political and ecological impact on the failure of the world to act on the issue of global warming," said Brulle. "Like a play on Broadway, the countermovement has stars in the spotlight – often prominent contrarian scientists or conservative politicians – but behind the stars is an organizational structure of directors, script writers and producers, in the form of conservative foundations. If you want to understand what's driving this movement, you have to look at what's going on behind the scenes."

To uncover how the countermovement was built and maintained, Brulle developed a listing of 118 important climate denial organizations in the U.S. He then coded data on philanthropic funding for each organization, combining information from the Foundation Center with financial data submitted by organizations to the Internal Revenue Service. The final sample for analysis consisted of 140 foundations making 5,299 grants totaling $558 million to 91 organizations from 2003 to 2010.

Key findings include:

Conservative foundations have bank-rolled denial. The largest and most consistent funders of organizations orchestrating climate change denial are a number of well-known conservative foundations, such as the Searle Freedom Trust, the John William Pope Foundation, the Howard Charitable Foundation and the Sarah Scaife Foundation. These foundations promote ultra-free-market ideas in many realms.

Koch and ExxonMobil have recently pulled back from publicly visible funding. From 2003 to 2007, the Koch Affiliated Foundations and the ExxonMobil Foundation were heavily involved in funding climate-change denial organizations. But since 2008, they are no longer making publicly traceable contributions.

Funding has shifted to pass through untraceable sources. Coinciding with the decline in traceable funding, the amount of funding given to denial organizations by the Donors Trust has risen dramatically. Donors Trust is a donor-directed foundation whose funders cannot be traced. This one foundation now provides about 25% of all traceable foundation funding used by organizations engaged in promoting systematic denial of climate change.

Most funding for denial efforts is untraceable. Despite extensive data compilation and analyses, only a fraction of the hundreds of millions in contributions to climate change denying organizations can be specifically accounted for from public records. Approximately 75% of the income of these organizations comes from unidentifiable sources.

"The real issue here is one of democracy. Without a free flow of accurate information, democratic politics and government accountability become impossible," said Brulle. "Money amplifies certain voices above others and, in effect, gives them a megaphone in the public square. Powerful funders are supporting the campaign to deny scientific findings about global warming and raise public doubts about the roots and remedies of this massive global threat. At the very least, American voters deserve to know who is behind these efforts."

This study is part one of a three-part project by Brulle to examine the climate movement in the U.S. at the national level. The next step in the project is to examine the environmental movement or the climate change movement. Brulle will then compare the whole funding flow to the entire range of organizations on both sides of the debate.

http://www.medscape.com/viewarticle/818226?src=rss

Vitamin D Deficiency Linked to Fatal CVD

Vitamin D deficiency is much more strongly linked to fatal than nonfatal CV events, results of a large prospective study suggest [1].

Miriam E. Tucker

Heidelberg, Germany -"Although our results were able to confirm an approximately 27% increased total CV risk in subjects with vitamin D deficiency, they indicate that the risk is much stronger for (and possibly even confined to) fatal CVD events," write the researchers, led by cofirst authors **Drs Laura Perna** and **Ben Schottker**, German Cancer Research Center (Heidelberg).

The findings were published in the December issue of the *Journal of Clinical Endocrinology and Metabolism*.

Previous observational and randomized trials linking serum 25-hydroxyvitamin D (25(OH)D) concentrations with increased CVD risk have typically used only a single vitamin D measurement and did not separately examine fatal and nonfatal outcomes, they note.

The current population-based cohort study enrolled 9949 adults aged 50 to 74 years recruited during regular health check-ups at primary-care practices in 2000 to 2002. There were more women than men (59% vs 41%); most participants (59%) had inadequate vitamin D levels (<50 nmol/L). Blood samples were collected at baseline, five, and eight years.

Mean follow-up was 9.2 years for mortality and 6.5 years for the end points of CVD, CHD, and stroke. A total of 854 patients had a nonfatal CVD event, 176 had a fatal CVD event, 460 had a nonfatal CHD event, 79 had a fatal CHD event, 313 had a nonfatal stroke, and 41 had a fatal stroke.

Overall, the proportion of individuals who had no events was significantly lower among those with vitamin D deficiency. The association continued after adjustment for age, sex, and season of blood drawn, with hazard ratios ranging from 1.46 for total CVD to 1.58 for total stroke.

Even after adjustment for other potential confounders, including smoking and physical activity, vitamin D deficiency still conferred a significant 27% increased risk for total CVD, and a 62% increased risk for fatal CVD. However, there was no association between vitamin D deficiency and nonfatal CVD events.

Individuals with low vitamin D levels also had a significant 36% increased risk of total CHD and a nonsignificant 33% increased risk of total stroke.

A possible explanation for the stronger association between 25(OH)D and CVD mortality than nonfatal CVD end points is that low vitamin D levels could lead to more severe events and perhaps also reduce capacity to cope with the events. Alternatively, the association of 25(OH)D with mortality may be more strongly affected by confounders linking to both low vitamin D and poor health status, such as diabetes or chronic kidney disease, the authors suggest.

This study was funded by the State Ministry of Science, Research, and Arts of Baden–Württemberg; German Cancer Aid (project 108250), and CHANCES project, funded in the FP7 framework program of DG-RESEARCH, European Commission (grant 242244). The authors have disclosed no relevant financial relationships.

References

1. Perna L, Schöttker B, Holleczek B, Brenner H. Serum 25-hydroxyvitamin D and incidence of fatal and nonfatal cardiovascular events: a prospective study with repeated measurements. J Clin Endocrinol Metab 2013; 98:4908-4915. Available at: http://press.endocrine.org/journal/jcem

http://www.medscape.com/viewarticle/818233?src=rss

Walking Lowers CVD Risk in Patients with Impaired Glucose Tolerance

Walking just 2000 steps per day lowers the risk of CVD by 10% in those with impaired glucose tolerance (IGT), according to the results of a new study.

Michael O'Riordan

Leicester, UK -In addition, the study also showed that, regardless of baseline walking habits, each 2000-step increase per day resulted in an 8% reduction in cardiovascular risk [1].

Two-thousand steps per day is the equivalent of approximately 20 minutes of walking at a moderate pace.

"These findings support both the promotion of increased ambulatory activity, and the avoidance of decreased ambulatory activity irrespective of the starting level, as important targets in the prevention of chronic disease," report Dr Thomas Yates (Leicester General Hospital, UK) and colleagues in the December 20, 2013 issue of the Lancet.

The findings are from the Nateglinide and Valsartan in Impaired Glucose Tolerance Outcomes Research (NAVIGATOR) study, which included 9306 randomized patients from 40 countries. All patients had IGT, defined as plasma glucose >140 mg/dL but <200 mg/dL after an oral glucose load, and at least one cardiovascular risk factor if aged 55 years or older (or known CVD if 50 years or older).
Results from the NAVIGATOR trial, previously reported by heartwire , showed that neither valsartan (Diovan, Novartis) nor nateglinide (Starlix, Novartis) had any significant effect on CV risk compared with placebo in separate randomizations. The trial also required patients to participate in a lifestyle-modification program designed to reduce the risk of diabetes through weight loss, dietary fat reduction, and increased exercise.
After a mean follow-up of five years, baseline walking habits and a change in walking activity were both inversely associated with the risk of a CV event. From baseline, each additional 2000 steps of daily walking at 12 months was associated with a 10% lower risk of a CV event. Similarly, each 2000-step increase or decrease in daily walking from baseline to 12 months was associated with an 8% decrease or increase in CV risk, respectively.
The results were unaffected when adjusted for potential confounding factors, including body-mass index, as well as a previous history of CVD, sex, age, or geographic region.
"In the absence of randomized controlled studies assessing the effect of physical activity on morbidity or mortality outcomes, our findings strengthen the evidence underpinning the importance of physical activity in the promotion of CV health and have important implications for public-health recommendations and the prevention of chronic disease," conclude Yates et al.
Novartis Pharmaceuticals provided funding for the study. Author disclosures are included in the article.
Yates T, Haffner SM, Schulte PJ. Association between change in daily ambulatory activity and cardiovascular events in people with impaired glucose tolerance (NAVIGATOR trial): a cohort analysis. Lancet 2013; DOI:10.1016/S0140-6736(13)62061-9. Available at: http://www.thelancet.com/.

http://phys.org/news/2013-12-scientists-highlight-resurrection-extinct-animals.html

Scientists highlight the resurrection of extinct animals as both a strong possibility and a major potential conservation

Scientists from across the world have "scanned the horizon" in order to identify potentially significant medium and long-term threats to conservation efforts.

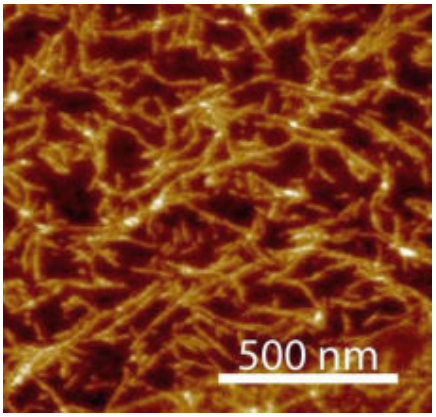
Phys.org - Resurrection of several extinct species, the increasingly accelerated loss of wild rhinoceroses and a disastrous financial response to unburnable carbon are just some future global conservation issues flagged up in this year's Horizon Scan, recently published in Trends in Ecology and Evolution.
Professor William Sutherland and Dr Mark Spalding are amongst the 18 scientists who took part in this year's Horizon Scan, seeking to identify potential future conservation issues in order to reduce the "probability of sudden confrontation with major social or environmental changes".
One such plausible issue is the resurrection or re-construction of extinct species, such as the woolly mammoth, passenger pigeon or the thylacine (a carnivorous marsupial). However, though there may be many benefits to the restoration of these animals, such a high-profile project could lead to attention and resources being diverted from attempts to thwart current threats to non-extinct species' survival.
Professor Sutherland said 'There has been discussion of this idea for some time but it is now looking more practical and the idea is being taken seriously. A key issues is whether this is really a conservation priority'.
Though the last woolly mammoth died around 4000 years ago, methods such as back-breeding, cloning and genetic engineering may lead to their resurrection. Not only could these extinct animals, and others such as the thylacine and the passenger pigeon, be re-constructed and returned to their native environments, they could potentially be used to "provide tools for outreach and education".
However, though this would be a conservational triumph, it could also hamper efforts to protect animals that are currently facing extinction, as both attention and resources would be diverted from preserving existing species and their habitats. Furthermore, there has not been any investigation into the "viability, ethics and safety of releasing resurrected species", nor the effect their presence may have on indigenous flora and fauna.
Another potential conservational issue identified by the Horizon Scan further highlights the problems facing species today. The loss of wild rhinoceroses and elephants is set to reaccelerate within the next few years, partially stimulated by a growing desire for ivory and horn.
In 2013, it is estimated that over 600 rhinoceroses were poached for their horn in South Africa alone, out of a total global population of less than 26,000. Though an increased human population and proximity to growing infrastructure is partially responsible, organised crime syndicates and intensive hunting carry the weight of the blame. In the Asian countries that use it, rhinoceros horn is more expensive than gold. Demand for the precious

horn is ever increasing, resulting in elevated levels of poaching. If attention and resources are diverted from the protection of these majestic animals, we may have yet more candidates for resurrection in the future.
Altogether, this group of scientists identified the top 15 potential conservation issues (out of an initial group of 81 issues). In addition to the above topics, extensive land loss in southeast Asia from subsidence of peatlands, carbon solar cells as an alternative source of renewable energy, and an emerging fungal disease amongst snakes, have also been voted as plausible threats that need to be stopped before they can be realised.
More information: William J. Sutherland, Rosalind Aveling, Thomas M. Brooks, Mick Clout, Lynn V. Dicks, Liz Fellman, Erica Fleishman, David W. Gibbons, Brandon Keim, Fiona Lickorish, Kathryn A. Monk, Diana Mortimer, Lloyd S. Peck, Jules Pretty, Johan Rockström, Jon Paul Rodríguez, Rebecca K. Smith, Mark D. Spalding, Femke H. Tonneijck, Andrew R. Watkinson, "A horizon scan of global conservation issues for 2014," Trends in Ecology & Evolution, Volume 29, Issue 1, January 2014, Pages 15-22, ISSN 0169-5347, dx.doi.org/10.1016/j.tree.2013.11.004.

http://phys.org/news/2013-12-scientists-closer-rna.html

New Study Brings Scientists Closer to the Origin of RNA

Chemists have shown how molecules that may have been present on early Earth can self-assemble into structures that could represent a starting point of RNA



Atomic force microscopy image of structures formed by the the self-assembly of TAP-ribose nucleoside with cyanuric acid. **Nicholas Hud.**

Phys.org - One of the biggest questions in science is how life arose from the chemical soup that existed on early Earth. One theory is that RNA, a close relative of DNA, was the first genetic molecule to arise around 4 billion years ago, but in a primitive form that later evolved into the RNA and DNA molecules that we have in life today. New research shows one way this chain of events might have started.
Today, genetic information is stored in DNA. RNA is created from DNA to put that information into action. RNA can direct the creation of proteins and perform other essential functions of life that DNA can't do. RNA's versatility is one reason that scientists think this polymer came first, with DNA evolving later as a better way to store genetic information for the long haul. But like DNA, RNA also could be a product of evolution, scientists theorize.
Chemists at the Georgia Institute of Technology have shown how molecules that may have been present on early Earth can self-assemble into structures that could represent a starting point of RNA. The spontaneous formation of RNA building blocks is seen as a crucial step in the origin of life, but one that scientists have struggled with for decades.
"In our study, we demonstrate a reaction that we see as important for the formation of the earliest RNA-like molecules," said Nicholas Hud, professor of Chemistry and Biochemistry at Georgia Tech, where he's also the director of the Center for Chemical Evolution. The study was published Dec. 14 online in the Journal of the American Chemical Society. The research was funded by the National Science Foundation and NASA.
RNA is perfect for the roles it plays in life today, Hud said, but chemically it's extraordinarily difficult to make. This suggests that RNA evolved from simpler chemical couplings. As life became more chemically complex and enzymes were born, evolutionary pressures would have driven pre-RNA into the more refined modern RNA.
RNA is made of three chemical components: the sugar ribose, the bases and phosphate. A ribose-base-phosphate unit links together with other ribose-base-phosphate units to form an RNA polymer. Figuring out how the bond between the bases and ribose first formed has been a difficult problem to address in the origins of life field, Hud said.
In the study, Hud's team investigated bases that are chemically related to the bases of modern RNA, but that might be able to spontaneously bond with ribose and assemble with other bases through the same interactions that enable DNA and RNA to store information. They homed in on a molecule called triaminopyrimidine (TAP).
The researchers mixed TAP with ribose under conditions meant to mimic a drying pond on early Earth. TAP and ribose reacted together in high yield, with up to 80 percent of TAP being converted into nucleosides, which is the name for the ribose-base unit of RNA. Previous attempts to form a ribose-base bond with the current RNA bases in similar reactions had either failed or produced nucleosides in very low yields.

"This study is important in showing a feasible step for how we get the start of an RNA-like molecule, but also how the building blocks of the first RNA-like polymers could have found each other and self-assembled in what would have been a very complex mixture of chemicals," Hud said.
The researchers demonstrated this property of the TAP nucleosides by adding another molecule to their reaction mixture, called cyanuric acid, which is known to interact with TAP. Even in the unpurified reaction mixture, noncovalent polymers formed with thousands of paired nucleosides.
"It is amazing that these nucleosides and bases actually assemble on their own, as life today requires complex enzymes to bring together RNA building blocks and to spatially order them prior to polymerization,"said Brian Cafferty, a graduate student at Georgia Tech and co-author of the study
The study demonstrated one possible way that the building blocks for an ancestor of RNA could have come together on early Earth. TAP is an intriguing candidate for one of the first bases that eventually led to modern RNA molecules, but there are certainly others, Hud said.
Future work, in Hud's lab and by other laboratories in the Center for Chemical Evolution, will investigate the origins of RNA's phosphate backbone, as well as other pathways toward modern RNA. "We're looking for a simple, robust chemistry that can explain the earliest origin of RNA or its ancestor," Hud said.

http://nyti.ms/1c6rud9

Wyoming May Act to Plug Abandoned Wells as Natural Gas Boom Ends

Hundreds of abandoned drilling wells dot eastern Wyoming like sagebrush, vestiges of a natural gas boom that has been drying up in recent years as prices have plummeted.

By DAN FROSCH

DENVER - The companies that once operated the wells have all but vanished into the prairie, many seeking bankruptcy protection and unable to pay the cost of reclaiming the land they leased. Recent estimates have put the number of abandoned drilling operations in Wyoming at more than 1,200, and state officials said several thousand more might soon be orphaned by their operators. Wyoming officials are now trying to address the problem amid concerns from landowners that the wells could contaminate groundwater and are a blight on the land.
This month, Gov. Matt Mead proposed allocating $3 million to pay for plugging the wells and reclaiming the land around them. And the issue is expected to be debated during next year's legislative session as lawmakers seek to hold drilling companies more accountable.
"The downturn in natural gas prices has forced small operators out of business, and the problem has really accelerated over the last couple of years," said the governor's policy director, Shawn Reese. "Landowners would like their land to be brought back to a productive status and have orphaned wells cleaned up."
Drilling companies in Wyoming typically lease land from the state, private owners or the federal Bureau of Land Management, depending on who owns the mineral rights.
The state's Oil and Gas Conservation Commission already budgets $1 million a year to plug abandoned wells. And under the governor's proposal, the commission would appropriate another $3 million over the next four years in an effort to restore property value and reduce the risk of contamination. The money would come from a conservation tax that oil and gas companies pay. Still, given the number of wells already abandoned and the concern that more will soon be deserted, the money is not expected to go far. The state estimated that closing the 1,200 wells already abandoned would cost about $8 million.
Compounding the problem, state officials estimate that Wyoming may also have to plug 2,300 wells that are sitting idle but have not been entirely abandoned by operators.
There are also 400 idle wells scattered across land owned by the Bureau of Land Management, which has its own criteria for determining when a well on its land is considered abandoned or idle. State officials said they would need to work with the bureau to help deal with those wells, too.
Governor Mead also wants the commission, which he sits on, to review the conservation tax and bonding requirements for drilling companies to determine whether they are sufficient.
Currently, companies must pay a $75,000 blanket bond to cover all of the wells they operate — often numbering in the hundreds — on state and private land in Wyoming. Once a well stops producing and is deemed idle, the operator must pay up to $10 a linear foot in bonding to offset the cost of reclamation.
But it is at that point that some companies drift into financial trouble and cannot pay the additional fees, leaving the state to scramble to make up the cost.
The governor's proposal has drawn support from landowner groups like the Powder River Basin Coalition, which has been pushing the state to take a tougher tack toward financially marginal drilling companies.
"There has been a lot of hand-holding and coddling over the years when it comes to oil and gas operators and their ability to pay the bonding," said Jill Morrison, an organizer with the group.

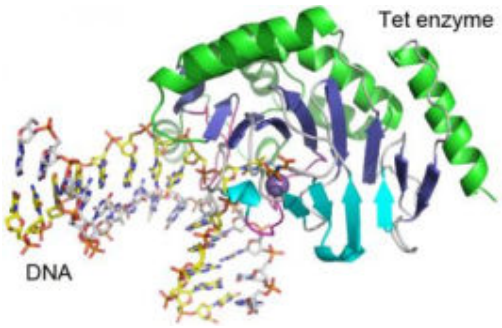
Ms. Morrison said that the issue had largely been ignored during Wyoming's peak boom years — from 1995 to 2004. "We are really pleased there is an actual plan to move forward with an aggressive plugging and reclamation strategy," she said.
The proposal is also backed by the Petroleum Association of Wyoming, which favors raising the conservation tax to help pay for plugging fees. The group also supports higher bond fees for operators with tenuous finances. "It's how you weed out companies that are too risky to go into business with," said the group's president, Bruce Hinchey.
But getting drilling companies who claim to be on the verge of collapse to take responsibility for wells they still technically own has proved difficult. One such company, Patriot Energy Resources, which owns about 900 idle wells on state and private land, said in an October letter to Governor Mead that it was $1.9 million short of full bonding on those wells after the bankruptcy filing of Luca Technologies, its parent company.
Patriot has proposed allowing another drilling company to take on a part of its debt, saying it will have to abandon its wells otherwise. "Without this deal or something similar, Patriot will be forced to file for bankruptcy and turn these wells and reservoirs over to the state of Wyoming," a company official wrote in the letter. Renny MacKay, a spokesman for Mr. Mead, said the state was weighing the offer.
State Senator John J. Hines, a Republican who represents mineral-rich Campbell and Converse Counties, said it was vital for lawmakers to take up the issue swiftly, because natural gas was so important to Wyoming's economy. "All of this just came to a head at once," said Mr. Hines, who heads the Senate's minerals committee. Last spring, Mr. Hines was told by Patriot that the hum of gas drilling activity on his own sprawling cattle ranch would soon grow quiet. Soon after, the company, which leased parcels of Mr. Hines's land, disappeared completely — leaving behind more than 40 coal-bed methane wells and a jumble of pipes and pumps.
"They informed me that they were shutting down because they were short of funds," Mr. Hines said. "All of it, in my opinion, needs to be cleaned up."

http://www.eurekalert.org/pub_releases/2013-12/ehs-eer122313.php

Epigenetics enigma resolved

First structure of enzyme that removes methylation

Scientists have obtained the first detailed molecular structure of a member of the Tet family of enzymes. The finding is important for the field of epigenetics because Tet enzymes chemically modify DNA, changing signposts that tell the cell's machinery "this gene is shut off" into other signs that say "ready for a change."



Tet enzymes' roles have come to light only in the last five years; they are needed for stem cells to maintain their multipotent state, and are involved in early embryonic and brain development and in cancer. The results, which could help scientists understand how Tet enzymes are regulated and look for drugs that manipulate them, are scheduled for publication in Nature.

This is the structure of the Tet enzyme with DNA. Note the purple ball at the active site, close to which one DNA base is flipped out of the double helix. Also note the degree to which the double helix is bent. **Xiaodong Cheng, Emory University**

Researchers led by Xiaodong Cheng, PhD, determined the structure of a Tet family member from Naegleria gruberi by X-ray crystallography. The structure shows how the enzyme interacts with its target DNA, bending the double helix and flipping out the base that is to be modified.
"This base flipping mechanism is also used by other enzymes that modify and repair DNA, but we can see from the structure that the Tet family enzymes interact with the DNA in a distinct way," Cheng says.
Cheng is professor of biochemistry at Emory University School of Medicine and a Georgia Research Alliance Eminent Scholar. The first author of the paper is research associate Hideharu Hashimoto, PhD. A team led by Yu Zheng, PhD, a senior research scientist at New England Biolabs, contributed to the paper by analyzing the enzymatic activity of Tet using liquid chromatography–mass spectrometry.
Using oxygen, Tet enzymes change 5-methylcytosine into 5-hydroxymethylcytosine and other oxidized forms of methylcytosine. 5-methylcytosine (5-mC) and 5-hydroxymethylcytosine (5-hmC) are both epigenetic modifications of DNA, which change how DNA is regulated without altering the letters of the genetic code itself.
5-mC is generally found on genes that are turned off or on repetitive regions of the genome. 5-mC helps shut off genes that aren't supposed to be turned on (depending on the cell type) and changes in 5-mC's distribution underpin a healthy cell's transformation into a cancer cell.

In contrast to 5-mC, 5-hmC appears to be enriched on active genes, especially in brain cells. Having a Tet enzyme form 5-hmC seems to be a way for cells to erase or at least modify the "off" signal provided by 5-mC, although the functions of 5-hmC are an active topic of investigation, Cheng says.
Alterations of the Tet enzymes have been found in forms of leukemia, so having information on the enzymes' molecular structure could help scientists design drugs that interfere with them.
N. gruberi is a single-celled organism found in soil or fresh water that can take the form of an amoeba or a flagellate; its close relative N. fowleri can cause deadly brain infections. Cheng says his team chose to study the enzyme from Naegleria because it was smaller and simpler and thus easier to crystallize than mammalian forms of the enzyme, yet still resembles mammalian forms in protein sequence.
Mammalian Tet enzymes appear to have an additional regulatory domain that the Naegleria forms do not; understanding how that domain works will be a new puzzle opened up by having the Naegleria structure, Cheng says.
The research was supported by the National Institute of General Medical Sciences (GM049245, GM095209, GM105132) and the Georgia Research Alliance.

http://phys.org/news/2013-12-epa-probe-texas-natural-gas.html

Report raises new concerns about EPA probe of Texas natural gas drilling

The Environmental Protection Agency was justified in intervening to examine possible risks of gas drilling to Texas drinking water, the agency's internal watchdog reported Tuesday.

But environmentalists say the report raises fresh concerns about the EPA's 2012 decision to halt its investigation into possible well water contamination in Parker County, Texas.
The EPA Inspector General's report is the latest analysis to spotlight the regulator's handling of high-profile cases of alleged drinking-water contamination near natural gas drilling sites.
Over the past three years, the EPA has sampled water in Dimock, Pa.; Pavillion, Wyo.; and Parker County, Texas, after residents complained that their water had turned foul once natural gas drilling began nearby. In each case, the EPA found evidence of contamination, but declined to pursue further water sampling or disciplinary action against the energy companies.
Critics have accused the Obama administration of backing away from the inquiries amid industry and political pressure, and because it views natural gas drilling as crucial to the economy and a cleaner environment. In July, an internal EPA report indicated that workers in its Philadelphia office wanted to keep monitoring Dimock's drinking water but EPA headquarters closed the investigation.
The Inspector General's inquiry was launched at the behest of Sen. James Inhofe, R-Okla., and other lawmakers who contend that EPA's regional office in Texas, EPA Region 6, had exceeded its authority during the Parker County investigation. Many Republicans viewed the EPA's probe as a politically motivated attack against the oil and gas industry.
On Tuesday, Inhofe's office dismissed the Inspector General's report, saying it had "failed to examine (a) closed-door conspiracy" to ruin the reputation of the energy company involved, Range Resources.
But environmentalists welcomed it as vindication of efforts by the EPA and independent scientists to explore the potential pollution risks of hydraulic fracturing, or fracking. The process involves injecting millions of gallons of water and sand laced with chemicals deep underground to crack shale formations and unlock oil and gas.
The EPA got involved in 2010 because Range Resources and Texas state regulators failed to act immediately on homeowners' complaints of possible drinking water contamination, the report said.
When EPA conducted its own tests, it found such high levels of methane in the water supply of two homes that it posed a risk of explosion, the report said. EPA tests also showed that the water contained benzene, a known carcinogen, above the EPA's maximum contamination levels.
Methane is the main component of natural gas, and an analysis performed for the EPA by an independent scientist found it to be nearly identical to the natural gas from the nearby Range Resources gas well.
Range Resources and the state of Texas denied that the company's gas development had contaminated the Parker County residents' water.
EPA issued an emergency order against Range Resources to provide drinking water to the affected residents and to better monitor the gas well. When Range Resources did not fully comply, the Justice Department filed a complaint on behalf of EPA in January 2011, but withdrew it by March 2012.
The Inspector General's report said that the EPA and Justice Department halted their action because the EPA worried about the costs and legal risks of the case. Although most officials were confident of their evidence, "there was always a risk that the judge could rule against the EPA. If that happened, it would risk establishing case law that could weaken the EPA's ability to enforce" parts of the Safe Drinking Water Act, the report said.

Range Resources threatened to refuse to cooperate with a study the EPA had launched into possible effects of fracking on drinking water if the agency disciplined it, the report said. The EPA shelved its complaint after getting a non-binding agreement for access to the company's sites, the report said, but so far, has declined to participate in the study.
A policy analyst for the Natural Resources Defense Council, Amy Mall, noted that EPA had enough evidence to intervene but "chose to step away from enforcing the law when drinking water was unsafe. ... Drinking water quality should never be traded for a hollow promise that may never be fulfilled."
In a statement, the EPA said the report determined its action were "supported by law and fact."
Range Resources spokesman Matt Pitzarella denied that its natural gas development had contaminated drinking water and said it is reviewing the report. The company is pursuing a $3 million defamation lawsuit against one of the homeowners who complained about his water, Steve Lipsky.

http://www.bbc.co.uk/news/science-environment-25506198

Diabetes risk gene 'from Neanderthals'

A gene variant that seems to increase the risk of diabetes in Latin Americans appears to have been inherited from Neanderthals, a study suggests.

By Paul Rincon Science editor, BBC News website

We now know that modern humans interbred with a population of Neanderthals shortly after leaving Africa 60,000-70,000 years ago.
This means that Neanderthal genes are now scattered across the genomes of all non-Africans living today.
Details of the study appear in the journal Nature.
The gene variant was detected in a large genome-wide association study (GWAS) of more than 8,000 Mexicans and other Latin Americans. The GWAS approach looks at many genes in different individuals, to see whether they are linked with a particular trait.
People who carry the higher risk version of the gene are 25% more likely to have diabetes than those who do not, and people who inherited copies from both parents are 50% more likely to have diabetes.
The higher risk form of the gene - named SLC16A11 - has been found in up to half of people with recent Native American ancestry, including Latin Americans.

Drug hope

The variant is found in about 20% of East Asians and is rare in populations from Europe and Africa.
The elevated frequency of this variant in Latin Americans could account for as much as 20% of these populations' increased prevalence of type 2 diabetes - the origins of which are complex and poorly understood.
"To date, genetic studies have largely used samples from people of European or Asian ancestry, which makes it possible to miss culprit genes that are altered at different frequencies in other populations," said co-author Jose Florez, associate professor of medicine at Harvard Medical School in Massachusetts.
"By expanding our search to include samples from Mexico and Latin America, we've found one of the strongest genetic risk factors discovered to date, which could illuminate new pathways to target with drugs and a deeper understanding of the disease."
The team that discovered the variant carried out additional analyses, in collaboration with Svante Paabo of the Max Planck Institute for Evolutionary Anthropology.
They discovered that the SLC16A11 sequence associated with risk of type 2 diabetes is found in a newly sequenced Neanderthal genome from Denisova Cave in Siberia.
Analyses indicate that the higher risk version of SLC16A11 was introduced into modern humans through interbreeding between early modern humans and Neanderthals.
It is not unusual to find Neanderthal genes. About 2% of the genomes of present-day non-Africans were inherited from this distinctive human group, which lived across Europe and western Asia from about 400,000-300,000 years ago until 30,000 years ago.
But scientists are only just beginning to understand the functional implications of this Neanderthal inheritance.
"One of the most exciting aspects of this work is that we've uncovered a new clue about the biology of diabetes," said co-author David Altshuler, who is based at the Broad Institute in Massachusetts.
SLC16A11 is part of a family of genes that code for proteins that transport metabolites - molecules involved in the body's various chemical reactions.
Altering the levels of the SLC16A11 protein can change the amount of a type of fat that has been implicated in the risk of diabetes. These findings suggest that SLC16A11 could be involved in the transport of an unknown metabolite that affects fat levels in cells and thereby increases risk of type 2 diabetes.

http://www.bbc.co.uk/news/health-25415587

'Without the web, I'd still be searching for a diagnosis'

Amy Garton-Hughes is 22, but the size of an eight-year-old. She has Cockayne Syndrome - a rare genetic disorder which degenerates the body and shortens young lives.

By Jane Dreaper Health correspondent, BBC News

This cruel illness has taken away Amy's balance, her speech is deteriorating and she has signs of dementia. But she still enjoys a busy life - listing swimming, bowling and seeing friends as her hobbies.

Jayne Hughes, from Merseyside, spent years trying to identify her daughter's illness. Doctors were mystified and library books gave no answers. But online searches eventually did - yielding pictures of other children with the same distinctive sunken eyes and pixie-like faces.

Jayne said: "When I found Cockayne Syndrome on the internet, there were a couple of photos of different children that all looked like her. "So I printed them off and when my dad came round, I said 'have a look at this'. He asked when I'd taken that picture of Amy. I said 'actually it's not Amy'. "It was at that point when I thought - this is it, that's definitely what she's got."

Jayne Hughes jokes that she only recently learnt to cut and paste. But she has found the internet invaluable. Her family's website, Amy and Friends, supports 1,500 other young people around the world who are affected by Cockayne Syndrome. Jayne added: "If it wasn't for the internet, I'd still be searching. Trying to find out what was wrong with Amy was like an obsession. "I couldn't settle or sleep and I couldn't look after my other children properly. Without the internet I'd be lost."

'Cyberchondria'

But for others, the world of web forums and online symptom checkers can be a minefield. At a mental health centre, run by Imperial College Healthcare in London, community psychiatrists treat people who have "cyberchondria" - health anxiety fuelled by the internet.

Professor Peter Tyrer said: "Cyberchondria is just being recognised as an extremely important part of this. We find that approximately four out of five of our patients with health anxiety spend literally hours on the internet." He says cyberchondria, which gives sufferers a deep fear of diseases, is on the rise. His research on this has been published in The Lancet.

There is good news - it can be treated effectively with simple therapy. Prof Tyrer said: "One of the first things we do in treatment is we tell them to stop browsing the internet. "And we ask them to keep diaries, which demonstrate very clearly that when they look at the internet, their anxiety increases. "The trouble is the internet contains all the knowledge you need to know - but it doesn't have any judgement associated with it."

Backlash from doctors

Technology does of course present new opportunities for solving old problems. Dr Christian Jessen - a GP, TV presenter and voracious user of Twitter - estimates about two-thirds of his 30,000 tweets have been answers to people's health questions. One recent example was someone who was struggling to get an appointment to have their ears syringed. Dr Jessen said: "I advised them to use olive oil as drops. Olive oil is anti-bacterial, antiseptic and softening - they may not need their ears syringing after using that."

He has some ground rules: he always retweets the original question, and he refuses to offer medical opinion on photos that people send him. He sees this as a modern and succinct way of helping people.

Dr Jessen added: "When I started doing this, there was a backlash. Doctors hated me doing it. They said I shouldn't interact with people I'd never seen before and answer medical questions. "But it's no different to being at a party, when you announce you're a doctor. The first thing people do is launch into a long medical story and ask for advice. It's exactly the same thing." He recommends patient.co.uk and NHS Choices as balanced sources of information - but warns nothing can replace a doctor for actual diagnosis.

'Horrified' cancer patient

Another respected website, HealthTalkOnline, focuses on patients' stories. It is carefully researched and curated by academics. In its seventieth collection of stories, people talk frankly about being on anti-depressants.

Stuart Jessup, an engineer turned teacher, was recruited by Twitter to take part in the work. He has walked the British coastline to raise awareness about depression. At the launch, he jokes about how online forums for depressed people are best avoided. He said: "Depression is a cycle of negative thinking. So put lots of people who are stuck in negative thinking together and moderate it - it's dangerous!"

Professor Sue Ziebland, from Oxford University, has spent 15 years examining how patients use the internet - including people with cancer. She said: "One of the men we interviewed went to his local library to go online and look for information about local support groups. "Almost the first thing he found, on one of the voluntary society websites for that particular cancer, was the very distressing five-year survival rate. "He was horrified.

He shut down the computer and ran out of the library. "The information he found was entirely accurate - but perhaps it shouldn't be on the front page. It's about signposting. "
After sometimes seeing the internet as a threat in its early days, Prof Ziebland says doctors now routinely discuss it as a resource with patients during consultations.
It has become an everyday part of medical conversation.

http://www.eurekalert.org/pub_releases/2013-12/uomh-sbc122313.php

Surgery beats chemotherapy for tongue cancer, U-M study finds

Patients with tongue cancer who started their treatment with a course of chemotherapy fared significantly worse than patients who received surgery first, according to a new study from researchers at the University of Michigan Comprehensive Cancer Center.

ANN ARBOR, Mich. - This is contrary to protocols for larynx cancer, in which a single dose of chemotherapy helps determine which patients fare better with chemotherapy and radiation and which patients should elect for surgery. In larynx cancer, this approach, which was pioneered and extensively researched at U-M, has led to better patient survival and functional outcomes.
But this new study, which appears in JAMA Otolaryngology Head and Neck Surgery, describes a clear failure.
"To a young person with tongue cancer, chemotherapy may sound like a better option than surgery with extensive reconstruction. But patients with oral cavity cancer can't tolerate induction chemotherapy as well as they can handle surgery with follow-up radiation. Our techniques of reconstruction are advanced and offer patients better survival and functional outcomes," says study author Douglas Chepeha, M.D., MSPH, professor of otolaryngology – head and neck surgery at the University of Michigan Medical School.
The study enrolled 19 people with advanced oral cavity cancer. Patients received an initial dose of chemotherapy, called induction chemotherapy. Those whose cancer shrunk by half went on to receive additional chemotherapy combined with radiation treatment. Those whose cancer did not respond well had surgery followed by radiation. Enrollment in the trial was stopped early because results were so poor.
Ten of the patients had a response to the chemotherapy, and of that group, only three had a complete response from the treatment and were cancer-free five years later. Of the nine patients who had surgery after the induction chemotherapy, only two were alive and cancer-free after five years. The researchers then looked at a comparable group of patients who had surgery and sophisticated reconstruction followed by radiation therapy and found significantly better survival rates and functional outcomes.
"The mouth is a very sensitive area," Chepeha says. "We know the immune system is critical in oral cavity cancer, and chemotherapy suppresses the immune system. If a person is already debilitated, they don't do well with chemotherapy." "Despite the proven success of this strategy in laryngeal cancer, induction chemotherapy should not be an option for oral cavity cancer, and in fact it results in worse treatment-related complications compared to surgery," Chepeha adds.

Tongue cancer statistics: 13,590 Americans will be diagnosed with tongue cancer this year and 2,070 will die from the disease, according to the American Cancer Society

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Gene therapy for human skin disease produces long-term benefits

Transplanting small number of skin stem cells sufficient to restore normal skin function, without causing adverse side effects

Stem cell-based gene therapy holds promise for the treatment of devastating genetic skin diseases, but the long-term clinical outcomes of this approach have been unclear. In a study online December 26th in the ISSCR's journal Stem Cell Reports, published by Cell Press, researchers evaluated a patient with a genetic skin disorder known as epidermolysis bullosa (EB) nearly seven years after he had undergone a gene therapy procedure as part of a clinical trial. The study revealed that a small number of skin stem cells transplanted into the patient's legs were sufficient to restore normal skin function, without causing any adverse side effects.
"These findings pave the way for the future safe use of epidermal stem cells for combined cell and gene therapy of epidermolysis bullosa and other genetic skin diseases," says senior study author Michele De Luca of the University of Modena and Reggio Emilia.

EB is a painful condition that causes the skin to be very fragile and to blister easily, and it can also cause life-threatening infections. Because there is no cure for the disease, current treatment strategies focus on relieving symptoms. To evaluate stem cell-based gene therapy as a potential treatment, De Luca and his colleagues previously launched a phase I/II clinical trial at the University of Modena and recruited an EB patient named Claudio. The researchers took skin stem cells from Claudio's palm, corrected the genetic defect in these cells, and then transplanted them into Claudio's upper legs.

In the new study, De Luca and his team found that this treatment resulted in long-term restoration of normal skin function. Nearly seven years later, Claudio's upper legs looked normal and did not show signs of blisters, and there was no evidence of tumor development. Remarkably, a small number of transplanted stem cells was sufficient for long-lasting skin regeneration.

Even though Claudio's skin had undergone about 80 cycles of renewal during this time period, the transplanted stem cells still retained molecular features of palm skin cells and did not adopt features of leg skin cells. "This finding suggests that adult stem cells primarily regenerate the tissue in which they normally reside, with little plasticity to regenerate other tissues," De Luca says. "This calls into question the supposed plasticity of adult stem cells and highlights the need to carefully chose the right type of stem cell for therapeutic tissue regeneration."

Stem Cell Reports, De Rosa et al.: "Long-Term Stability And Safety Of Transgenic Cultured Epidermal Stem Cells In Gene Therapy Of Junctional Epidermolysis Bullosa."

http://www.eurekalert.org/pub_releases/2013-12/uorm-wdc122613.php

What does compassion sound like?

"Good to see you. I'm sorry. It sounds like you've had a tough, tough, week."

Spoken by a doctor to a cancer patient, that statement is an example of compassionate behavior observed by a University of Rochester Medical Center team in a new study published by the journal Health Expectations. Rochester researchers believe they are the first to systematically pinpoint and catalogue compassionate words and actions in doctor-patient conversations. By breaking down the dialogue and studying the context, scientists hope to create a behavioral taxonomy that will guide medical training and education.

"In health care, we believe in being compassionate but the reality is that many of us have a preference for technical and biomedical issues over establishing emotional ties," said senior investigator Ronald Epstein, M.D., professor of Family Medicine, Psychiatry, Oncology, and Nursing and director of the UR Center for Communication and Disparities Research. Epstein is a national and international keynote speaker and investigator on mindfulness and communication in medical education.

His team recruited 23 oncologists from a variety of private and hospital-based oncology clinics in the Rochester, N.Y., area. The doctors and their stage III or stage IV cancer patients volunteered to be recorded during routine visits. Researchers then analyzed the 49 audio-recorded encounters that took place between November 2011 and June 2012, and looked for key observable markers of compassion.

In contrast to empathy – another quality that Epstein and his colleagues have studied in the medical community -- compassion involves a deeper and more active imagination of the patient's condition. An important part of this study, therefore, was to identify examples of the three main elements of compassion: recognition of suffering, emotional resonance, and movement towards addressing suffering.

Emotional resonance, or a sense of sharing and connection, was illustrated by this dialogue: Patient: "I should just get a room here." Oncologist: "Oh, I hope you don't really feel like you're spending that much time here."

Another conversation included this response from a physician to a patient, who complained about a drug patch for pain: "Who wants a patch that makes you drowsy, constipated and fuzzy? I'll pass, thank you very much."

Some doctors provided good examples of how they use humor to raise a patient's spirits without deviating from the seriousness of the situation. In one case, for example, a patient was concerned that he would not be able to drink two liters of barium sulfite in preparation for a CT scan.

Doctor: "If you just get down one little cup it will tell us what's going on in the stomach. What I tell people when we're not being recorded is to take a cup and then poor the rest down the toilet and tell them you drank it all (laughter)… Just a creative interpretation of what you are supposed to take."

Patient: "I love it, I love it. Well, I thank you for that. I'm prepared to do what I've got to do to get this right."

Researchers evaluated tone of voice, animation that conveyed tenderness and understanding, and other ways in which doctors gave reassurances or psychology comfort.

Here's an instance in which an oncologist encouraged a reluctant patient to follow through with a planned trip to Arizona: "You know, if you decide to do it, break down and allow somebody to meet you at the gates and use a cart or wheelchair to get you to your next gate and things like that. And having just sent my father-in-law off to Hawaii and told him he had to do that, he said no, no, I can get there.

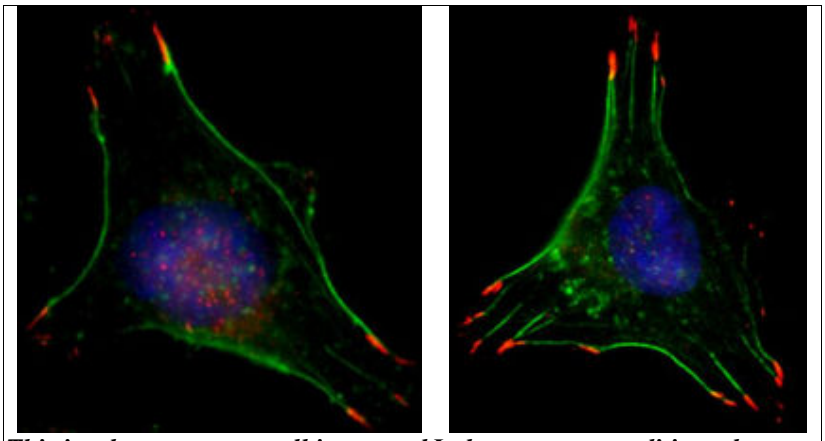
Just, it's okay. Nobody is gonna look at you and say, 'What's an able-bodied man doing in a cart?' Just, it's okay. It's part of setting limits."
Researchers also observed non-verbal communication, such as pauses or sighs at appropriate times, as well as speech features and voice quality (tone, pitch, loudness) and other metaphorical language that conveyed certain attitudes and meaning.
Compassion unfolds over time, researchers concluded. During the process, physicians must challenge themselves to stay with a difficult discussion, which opens the door for the patient to admit uncertainty and grieve the loss of normalcy in life.
"It became apparent that compassion is not a quality of a single utterance but rather is made up of presence and engagement that suffuses and entire conversation," the study said. First author, Rachel Cameron, B.A., is a student at the University of Rochester School of Medicine and Dentistry; the audio-recordings were reviewed by a diverse group of medical professionals with backgrounds in literature and linguistics, as well as palliative care specialists. *The National Cancer Institute funded the study.*

http://www.eurekalert.org/pub_releases/2013-12/jhm-rar122613.php

Rock And Rho: Proteins that help cancer cells groove

Cells' adaptations to low oxygen conditions inside tumors promote breast cancer's spread

Biologists at The Johns Hopkins University have discovered that low oxygen conditions, which often persist inside tumors, are sufficient to initiate a molecular chain of events that transforms breast cancer cells from being rigid and stationery to mobile and invasive. Their evidence, published online in Proceedings of the National Academy of Sciences on Dec. 9, underlines the importance of hypoxia-inducible factors in promoting breast cancer metastasis.



This is a breast cancer cell in normal oxygen conditions. **Daniele Gilkes**

In low oxygen conditions, breast cancer cells form structures that facilitate movement, such as filaments that allow the cell to contract (green) and cellular 'hands' that grab surfaces to pull the cell along (red). **Daniele Gilkes**

"High levels of RhoA and ROCK1 were known to worsen outcomes for breast cancer patients by endowing cancer cells with the ability to move, but the trigger for their production was a mystery," says Gregg Semenza, M.D., Ph.D., the C. Michael Armstrong Professor of Medicine at the Johns Hopkins University School of Medicine and senior author of the article. "We now know that the production of these proteins increases dramatically when breast cancer cells are exposed to low oxygen conditions."
To move, cancer cells must make many changes to their internal structures, Semenza says. Thin, parallel filaments form throughout the cells, allowing them to contract and cellular "hands" arise, allowing cells to "grab" external surfaces to pull themselves along. The proteins RhoA and ROCK1 are known to be central to the formation of these structures.
Moreover, the genes that code for RhoA and ROCK1 were known to be turned on at high levels in human cells from metastatic breast cancers. In a few cases, those increased levels could be traced back to a genetic error in a protein that controls them, but not in most. This activity, said Semenza, led him and his team to search for another cause for their high levels.
What the study showed is that low oxygen conditions, which are frequently present in breast cancers, serve as the trigger to increase the production of RhoA and ROCK1 through the action of hypoxia-inducible factors.
"As tumor cells multiply, the interior of the tumor begins to run out of oxygen because it isn't being fed by blood vessels," explains Semenza. "The lack of oxygen activates the hypoxia-inducible factors, which are master control proteins that switch on many genes that help cells adapt to the scarcity of oxygen." He explains that, while these responses are essential for life, hypoxia-inducible factors also turn on genes that help cancer cells escape from the oxygen-starved tumor by invading blood vessels, through which they spread to other parts of the body.

Daniele Gilkes, Ph.D., a postdoctoral fellow and lead author of the report, analyzed human metastatic breast cancer cells grown in low oxygen conditions in the laboratory. She found that the cells were much more mobile in the presence of low levels of oxygen than at physiologically normal levels. They had three times as many filaments and many more "hands" per cell. When the hypoxia-inducible factor protein levels were knocked down, though, the tumor cells hardly moved at all. The numbers of filaments and "hands" in the cells and their ability to contract were also decreased.

When Gilkes measured the levels of the RhoA and ROCK1 proteins, she saw a big increase in the levels of both proteins in cells grown in low oxygen. When the breast cancer cells were modified to knock down the amount of hypoxia-inducible factors, however, the levels of RhoA and ROCK1 were decreased, indicating a direct relationship between the two sets of proteins. Further experiments confirmed that hypoxia-inducible factors actually bind to the RhoA and ROCK1 genes to turn them on.

The team then took advantage of a database that allowed them to ask whether having RhoA and ROCK1 genes turned on in breast cancer cells affected patient survival. They found that women with high levels of RhoA or ROCK1, and especially those women with high levels of both, were much more likely to die of breast cancer than those with low levels.

"We have successfully decreased the mobility of breast cancer cells in the lab by using genetic tricks to knock the hypoxia-inducible factors down," says Gilkes. "Now that we understand the mechanism at play, we hope that clinical trials will be performed to test whether drugs that inhibit hypoxia-inducible factors will have the double effect of blocking production of RhoA and ROCK1 and preventing metastases in women with breast cancer."

Other authors of the report include Lisha Xiang, Sun Joo Lee, Pallavi Chaturvedi, Maimon Hubbi and Denis Wirtz of the Johns Hopkins University School of Medicine.

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http://nyti.ms/1kKHt50

In the Human Brain, Size Really Isn't Everything

There are many things that make humans a unique species, but a couple stand out. One is our mind, the other our brain.

By CARL ZIMMER

The human mind can carry out cognitive tasks that other animals cannot, like using language, envisioning the distant future and inferring what other people are thinking.

The human brain is exceptional, too. At three pounds, it is gigantic relative to our body size. Our closest living relatives, chimpanzees, have brains that are only a third as big.

Scientists have long suspected that our big brain and powerful mind are intimately connected. Starting about three million years ago, fossils of our ancient relatives record a huge increase in brain size. Once that cranial growth was underway, our forerunners started leaving behind signs of increasingly sophisticated minds, like stone tools and cave paintings.

But scientists have long struggled to understand how a simple increase in size could lead to the evolution of those faculties. Now, two Harvard neuroscientists, Randy L. Buckner and Fenna M. Krienen, have offered a powerful yet simple explanation.

In our smaller-brained ancestors, the researchers argue, neurons were tightly tethered in a relatively simple pattern of connections. When our ancestors' brains expanded, those tethers ripped apart, enabling our neurons to form new circuits.

Dr. Buckner and Dr. Krienen call their idea the tether hypothesis, and present it in a paper in the December issue of the journal Trends in Cognitive Sciences.

"I think it presents some pretty exciting ideas," said Chet C. Sherwood, an expert on human brain evolution at George Washington University who was not involved in the research.

Dr. Buckner and Dr. Krienen developed their hypothesis after making detailed maps of the connections in the human brain using f.M.R.I. scanners. When they compared their maps with those of other species' brains, they saw some striking differences.

The outer layers of mammal brains are divided into regions called cortices. The visual cortex, for example, occupies the rear of the brain. That is where neurons process signals from the eyes, recognizing edges, shading and other features.

There are cortices for the other senses, too. The sensory cortices relay signals to another set of regions called motor cortices. The motor cortices send out commands. This circuit is good for controlling basic mammal behavior. "You experience something in the world and you respond to it," Dr. Krienen said.

This relatively simple behavior is reflected in how the neurons are wired. The neurons in one region mostly make short connections to a neighboring region. They carry signals through the brain like a bucket brigade from the sensory cortices to the motor cortices.
The bucket brigade begins to take shape when mammals are still embryos. Different regions of the brain release chemical signals, which attract developing neurons.
"They will tell a neuron, 'You're destined to go to the back of the brain and become a visual neuron,' for example," Dr. Krienen said.
After mammals are born, their experiences continue to strengthen this wiring. As a mammal sees more of the world, for example, neurons in the visual cortex form more connections to the motor cortices, so that the bucket brigade moves faster and more efficiently.
Human brains are different. As they got bigger, their sensory and motor cortices barely expanded. Instead, it was the regions in between, known as the association cortices, that bloomed.
Our association cortices are crucial for the kinds of thought that we humans excel at. Among other tasks, association cortices are crucial for making decisions, retrieving memories and reflecting on ourselves.
Association cortices are also unusual for their wiring. They are not connected in the relatively simple, bucket-brigade pattern found in other mammal brains. Instead, they link to one another with wild abandon. A map of association cortices looks less like an assembly line and more like the Internet, with each region linked to others near and far.
Dr. Buckner and Dr. Krienen argue that this change occurred because of the way brains develop. In the human brain, some neurons still receive chemical signals that cause them to form a bucket brigade from the sensory cortices to the motor cortices. But because of the brain's size, some neurons are too far from the signals to follow their commands. "They may have broken off and formed a new circuit," Dr. Buckner said.
This new wiring may have been crucial to the evolution of the human mind. Our association cortices liberate us from the rapid responses of other mammal brains. These new brain regions can communicate without any input from the outside world, discovering new insights about our environment and ourselves.
Dr. Buckner foresees a number of ways in which the tether hypothesis could be tested. For example, many mammal brains, including chimpanzees', have yet to be fully mapped. "We're hoping that in the next 10 or 15 years, that might be possible," he said.
Dr. Sherwood, the George Washington University expert, praised the hypothesis for being "fairly frugal." The emergence of the human mind might not have been a result of a vast number of mutations that altered the fine structure of the brain. Instead, a simple increase in the growth of neurons could have untethered them from their evolutionary anchors, creating the opportunity for the human mind to emerge.

http://www.medscape.com/viewarticle/818339?src=rss

Bupropion Linked With Delayed-Onset Urticaria

Bupropion may put patients at risk for delayed-onset urticaria, a nationwide study from Taiwan has shown.

By Lorraine L. Janeczko

NEW YORK Reuters Health - Doctors should consider this possibility in patients on bupropion -- especially patients under 40 and those with a history of urticaria -- to avoid more severe allergic responses, the authors wrote in a report online November 14 in PLoS ONE.
"Clinicians may not relate this adverse effect, which is not commonly appreciated, to the drug because of its delayed onset," said Dr. James M. Wright of the University of British Columbia in Vancouver, Canada, in an email to Reuters Health. Dr. Wright, who edited the journal article manuscript but was not otherwise involved in the study, said, "Doctors should be more cautious about prescribing bupropion."
Bupropion-induced urticaria seldom develops alone and sometimes appears with other more serious allergic reactions, including arthralgia, angioedema, serum sickness-like reaction, and anaphylactic symptoms, the authors warn.
They say hives mustn't be confused with other skin diseases that appear similar and are also itchy.
To estimate the incidence of bupropion-induced delayed-onset urticaria, Dr. Tung-Ping Su of Taipei Veterans General Hospital and National Yang-Ming University and colleagues worked with 10 years of data from Taiwan's National Health Insurance Dataset.
From 65,988 patients with depression, they identified 2839 new bupropion users (4.3% of patients) and matched them 1:4 by age and sex with a non-bupropion group (n=11,356; 17.2% of patients).
The median age overall was 41. Most patients (n=8,540; 60.16%) were female. The most common comorbidities were liver disease (n=4,682; 33.3% of patients) and diabetes (n=2,833; 20.3% of patients).
Overall, 4,416 patients (31.32%) had a history of urticaria.

Within four weeks of starting the medication, the bupropion users had a higher risk of urticaria (cumulative incidence, 16.56% vs 9.16%; risk ratio 1.81; p=0.001). The risk was confined to patients under age 40 (risk ratio, 2.25; p<0.001), and it was high in both males and females. In the bupropion group, urticaria occurred more frequently on Days 15 through 28 than on Days 1 through 14 (p=0.002).
Delayed-onset urticaria was more frequent in patients in the bupropion group than for patients in the non-bupropion group (cumulative incidence, 11.98% vs 5.11%; risk ratio, 2.34; p<0.001). The only independent risk factor for bupropion-related urticaria was a history of urticaria (hazard ratio 3.03; p<0.001).
When the authors compared cumulative rates of urticaria in bupropion users and in patients receiving all other types of new antidepressant prescriptions in Taiwan, only bupropion users showed delayed onset.
"This was a good large study that established a firm connection between bupropion and urticaria. It is likely that bupropion causes urticaria," said Dr. Anton Alexandroff, a consultant dermatologist at the University Hospitals of Leicester in Leicestershire, United Kingdom, by email to Reuters Health.
"Previously no firm connection had been established. There were only published case reports, which had limited value," he said. "Now physicians should advise their patients that bupropion can cause urticaria, and that, if this happens, they should consider changing to another medication," he said. Dr. Alexandroff was not involved in the study. The authors did not respond to a request for comment. *SOURCE: http://bit.ly/1e92z8s*

http://bit.ly/K8YYyg

Darwin's Morning After Pill: How Couples Who Want Children Can Increase Their Chances

If you're desperate for a child but have been having trouble in this area, semen may be the solution to your reproductive woes.

By Jesse Bering | December 26, 2013

That may sound like the most obvious sentence ever written in the history of the English language, but sometimes beneath the most ancient truisms lie remarkable secrets. People have known semen to be a required element in human reproduction ever since that metaphorical Adam first ejaculated into Eve, but it's only now, in 2013, that we are beginning to fully realize just how, and more importantly *why* it all really works the way it does.
Actually, it was seven years ago that the theory I'm about to describe first saw the light of day, or at least a dim, filtered light through the pages of an obscure academic volume. In that specialist book, psychologists Jennifer Davis and Gordon Gallup from SUNY-Albany articulated a groundbreaking evolutionary theory—*seminal priming theory*—that ever since has been criminally unread (and thus unshared) by scientists and fertility doctors alike. Just off the top of my head, I can think of two still fruitless couples that undoubtedly would have benefited by having this information years ago.
The key to understanding seminal priming theory, and the part that is likely to be the most difficult for many people to accept—particularly those people who suffer the illusion that they are not animals—is the observation that certain cases of spontaneous abortion are biologically adaptive *for the mother*. One helpful hint is always to remember that "adaptive" in an evolutionary sense has an entirely different meaning from "adaptive" in a contemporary mental health sense. Very bad things, such as violence, depression, perhaps even suicide, may also be biologically adaptive while being outrageously maladaptive in the more everyday sense of the word. This is because the former implies simply a mindless, net genetic fitness advantage to the organism, whereas the latter centers on a person's subjective wellbeing. So while it may sound especially bizarre, and perhaps somewhat insensitive, to say that losing a child (even if that child is still a fetus) could ever be adaptive, context is everything.
Now, the theory. I should start by pointing out that pregnancy in human beings and the African great apes appears to be different from that of every other known mammal in one crucial way. Only in our species, chimpanzees and gorillas are there two distinct stages of implantation, not just one. For all mammals, the fertilized egg migrates to the uterine wall shortly after conception and implants there. But only for human embryos and those of our closest living relatives is there another implantation at the end of the first trimester, in which the uterine spiral arteries are deeply "invaded" by the trophoblast, which digs into the wall to siphon off a greater blood flow to the placenta. The natural history of this unique second stage of implantation is thought to be related to large brain evolution; basically, our prenatal cortices required considerably more nutritional supplies than those of other animals. Unfortunately, a leading cause of prenatal infant mortality, called *preeclampsia,* may also be unique to the African great apes—and substantially most common in human beings—since it is believed to stem from problems associated with this crucial second stage.

The most prominent symptoms of preeclampsia are maternal hypertension and proteinuria (significant protein in the mother's urine), and it can arise at any point during the second or third trimesters. Because it often correlates with spontaneous abortions—the only "cure" is immediate delivery of the child—the earlier it occurs in the pregnancy the more likely it is that the infant will not survive a premature birth. This was especially true, of course, during the yawning expanse of human history in which modern medical technology could not intervene to save the preterm infant. There are a host of ancillary risk factors for developing preeclampsia, including such things as obesity, maternal age, exercise, vitamin deficiency and maternal genetics, but the most astonishing common denominator is related to the *father's* genetic profile. More often than not, preeclampsia is the result of a hostile immunological maternal response to the paternal genome in the developing conceptus. In other words, the mother's body is unwittingly terminating a pregnancy that has arisen with a man for whom she has an incompatible biochemistry.

What makes this biochemistry incompatible? Its unfamiliarity, mainly. And this is the essence of the "seminal priming" construct: the risk of preeclampsia is substantially reduced when the woman has been exposed for an extended period of time to the father's semen prior to conception—and perhaps even *after* conception. Davis and Gallup, in fact, were not the first to discover this curious priming effect. By the early 1980s, scientists had started to notice that preeclampsia was more likely to occur in pregnancies resulting from "one-night stands," artificial insemination, and rape than in pregnancies that were the product of long-term sexual cohabitation. That it was the woman's prior exposure to the male's semen that was responsible for this pattern was evident by the fact that couples who'd been using barrier contraceptives (such as condoms), or who practiced coitus interruptus (in which the man withdraws prior to ejaculation) before they began trying to conceive also had higher rates of preeclampsia than those who'd been engaging in unprotected sex for some time. And you're going to love me for this, my heterosexual male friends: several studies even found that women who regularly performed oral sex were dramatically less likely to develop preeclampsia than were those who shied away from this sex act.

Yet while these causative effects of unfamiliar semen on preeclampsia had been known for some time, Davis and Gallup were the first to interpret these peculiar semen-related facts within a theoretically meaningful evolutionary framework. "It may be useful to think about preeclampsia not simply as a medical anomaly," reason the authors, "but as an adaptation that may have evolved to terminate pregnancies where future paternal investment was questionable or unlikely." Their argument, which is admittedly speculative, is predicated on the basic parental investment theory in evolutionary biology. While males could impregnate a potentially limitless number of females and spread their genes far and wide without any cost but a euphoric 90-second time investment, ancestral women's genetic interests were compromised by having sex with a man who had no intention of helping her to raise any resulting offspring. Yet, if she did, and conceived as a consequence of that intercourse, preeclampsia was a second line of adaptive defence that would terminate this "costly" pregnancy—a sort of Darwinian morning after pill, as Gallup explained it to me.

The authors are aware that, in rare cases, preeclampsia is fatal not only to the infant, but to the mother as well. "In our view, however," they reason, "the costs associated with preeclampsia for the mother would typically by outweighed by the benefits that derive from terminating a costly pregnancy before the child has been born."

A problem for many women today is that because this hypothesized preeclampsia adaptation predates modern technologies such as condoms and in vitro fertilization, and since natural selection could not have anticipated such recent cultural innovations, female physiology remains biased toward harboring fetuses that contain genetic material that their bodies have become intimately familiar with by way of a particular partner's seminal infusions. "Frequent insemination would be a relatively good biochemical index of the existence of a committed pair bond," write the authors:

... and, therefore, semen familiarity would predict the likelihood of long-term provisioning, protection, and care of the mother and the child during pregnancy and following parturition.

Clinical data are an embarrassment of riches supporting this model. Even after controlling for maternal age and parity (whether the mother has given birth before), the incidence of preeclampsia is still higher in donor-inseminated pregnancies than normally conceived pregnancies. And when looking exclusively at artificial inseminations, it is more common in women who became pregnant from a donor's sperm than those artificially inseminated by their partner's sperm. Studies with "multiparous" women—those who have given birth to more than one child—in regions as diverse as Nigeria and the Caribbean reveal that a change in paternity, from one pregnancy to the next, is also correlated with an increased risk of preeclampsia.

Davis and Gallup surmise that there would have been three "categories of unfamiliar semen" in the ancestral past that likely exerted selective pressure on this preeclampsia mechanism. Rape is the most obvious of these, and also the most theoretically important, because very often it would have meant a single instance of exposure

to foreign semen. Prior to bottle-feeding, conception and childbirth meant that a woman foreclosed on any other reproductive opportunities for at least 2-4 years, so pregnancies arising through single acts of coercive sex, in which paternal investment was improbable, would have meant an enormous gamble for her genetic fitness. Although preeclampsia was once a strategic adaptation adjusting for these prospective losses, however, today novel scenarios such as artificial insemination of a donor's sperm invoke the exact same mechanism. From the mindless perspective of the evolved woman's proactive womb, there's simply no way to tell this apart from rape.

The second category of unfamiliar semen, according to the authors, falls under the heading of "dishonest mating strategies." "It is not uncommon for males to feign good intentions and commitment to females," they point out, "as a means of attempting to gain sexual access." But female physiology may have worked out a way to thwart this age-old love-'em-and-leave-'em tactic. Familiar semen continues to promote pregnancy retention (or to reduce the chances of preeclampsia) for a period of time after conception. This is an important point, because remember, preeclampsia traces back to problems with the second implantation occurring at the end of the first trimester. Frequent, continuing insemination of a woman during at least the initial duration of the pregnancy would have signalled a man's ongoing commitment.

This same principle of recurrent insemination in the already-pregnant woman applies also to Davis and Gallup's last category of unfamiliar semen. This involves honest mating strategies—the male genuinely feels committed upon having sex—but because conception happens to occur very early in the relationship, when the romantic bond is still tenuous, the chances of his abandoning her and the child are in fact quite high.

Now, if you'll excuse me, I'm off to share the seminal priming theory with a lovely lesbian couple I know that soon will begin trying to conceive through laboratory methods. I suspect that their physician is unlikely to instruct them to do so, but they will be delighted to hear that simply gargling with the chosen donor's semen each night, or perhaps gently dabbing some of this substance against one of their vaginal walls before bedtime, may very well work wonders for them. I jest. This isn't a ludicrous idea, in fact, and some scientists have already recognized the potential of developing sublingual sprays or vaginal gels that are liquid concatenations of various immune factors mixed with the male's protein—essentially, it's a gay and singles-friendly, modern pharmaceutical proxy of good old-fashioned seminal priming.

[Author's note: A version of this article originally appeared in the Swiss magazine* Das Magazin *and was published in the German language on July 21, 2012.]

http://phys.org/news/2013-12-nasa-jaxa-date-global-precipitation.html

NASA and JAXA announce launch date for global precipitation satellite

Environmental research and weather forecasting are about to get a significant technology boost as NASA and the Japan Aerospace Exploration Agency (JAXA) prepare to launch a new satellite in February.

Phys.org - NASA and JAXA selected 1:07 p.m. to 3:07 p.m. EST Thursday, Feb. 27 (3:07 a.m. to 5:07 a.m. JST Friday, Feb. 28) as the launch date and launch window for a Japanese H-IIA rocket carrying the Global Precipitation Measurement (GPM) Core Observatory satellite from JAXA's Tanegashima Space Center.

GPM is an international satellite mission that will provide advanced observations of rain and snowfall worldwide, several times a day to enhance our understanding of the water and energy cycles that drive Earth's climate. The data provided by the Core Observatory will be used to calibrate precipitation measurements made by an international network of partner satellites to quantify when, where, and how much it rains or snows around the world.

"Launching this core observatory and establishing the Global Precipitation Measurement mission is vitally important for environmental research and weather forecasting," said Michael Freilich, director of NASA's Earth Science Division in Washington. "Knowing rain and snow amounts accurately over the whole globe is critical to understanding how weather and climate impact agriculture, fresh water availability, and responses to natural disasters."

With the addition of the new Core Observatory, the satellites in the GPM constellation will include the NASA-National Oceanic and Atmospheric Administration (NOAA) Suomi National Polar-orbiting Partnership mission, launched in 2012; the NASA-JAXA Tropical Rainfall Measuring Mission (TRMM), launched in 1997; and several other satellites managed by JAXA, NOAA, the U.S. Department of Defense, the European Organisation for the Exploitation of Meteorological Satellites, the Centre National D'Etudies Spatiales of France and the Indian Space Research Organisation.

"We will use data from the GPM mission not only for Earth science research but to improve weather forecasting and respond to meteorological disasters," said Shizuo Yamamoto, executive director of JAXA. "We would also like to aid other countries in the Asian region suffering from flood disasters by providing data for

flood alert systems. Our dual-frequency precipitation radar, developed with unique Japanese technologies, plays a central role in the GPM mission."
The GPM Core Observatory builds on the sensor technology developed for the TRMM mission, with two innovative new instruments. The GPM Microwave Imager, built by Ball Aerospace and Technology Corp., Boulder, Colo., will observe rainfall and snowfall at 13 different frequencies. The Dual-frequency Precipitation Radar, developed by JAXA with the National Institute of Information and Communication Technology in Tokyo, transmits radar frequencies that will detect ice and light rain, as well as heavier rainfall. It also will be able to measure the size and distribution of raindrops, snowflakes and ice particles.

http://www.sciencedaily.com/releases/2013/12/131219195928.htm

Protein Links Liver Cancer With Obesity, Alcoholism, Hepatitis

Obesity, alcoholism, and chronic hepatitis all increase the risk of getting liver cancer, which is the third leading cause of cancer death worldwide.

Obesity in particular is driving a significant increase in liver cancer in the United States. These three health problems also increase cellular stress in the liver, but until now it has not been clear if there is a direct biological link between cellular stress and the development of liver cancer.
In a new study, University of Iowa researchers have identified an unexpected molecular link between liver cancer, cellular stress, and these health problems that increase the risk of developing this cancer.
The study, published Dec. 19 in the journal PLOS Genetics, shows that a protein called CHOP, which had previously been thought to generally protect against cancer, actually promotes liver cancer in mice and may do the same in humans.
"Obesity, alcoholism, and viral hepatitis are all known independently to cause cellular stress and to induce expression of CHOP," says Thomas Rutkowski, Ph.D., assistant professor of anatomy and cell biology in the UI Carver College of Medicine and senior study author. "So this finding suggests a biological pathway that links those 'upstream' health problems to liver cancer at the end."
CHOP is a transcription factor that is produced when cells experience certain kinds of stress. It is known to promote cell death, or apoptosis. Usually, factors that promote cell death protect against cancer by causing damaged cells to die.
The study shows that, despite its role in cell death, CHOP actually is elevated in liver tumor cells in mice. Furthermore, mice without CHOP are partially protected from liver cancer, developing fewer and smaller tumors than the normal mice in response to liver cancer-causing drugs. The mice without CHOP also had less liver scarring and inflammation than mice with the protein.
Tissue samples from human patients show that CHOP also is elevated in human liver tumors compared to surrounding non-tumor tissue from the same patients.
"We turned out to be completely wrong about CHOP. We found that it contributes to the development of liver cancer in mice and is associated with liver cancer in humans," Rutkowski says. "CHOP is indeed killing cells, just as we thought it would, but we think the consequence of this killing is not the prevention of tumors, but instead the stimulation of inflammatory signals in the liver that cause excessive proliferation of other cells," he explains. Rutkowski notes that although this proposed mechanism is not proven yet, it is consistent with what is known about the role of CHOP.
Collaboration was critical to the success of the study, Rutkowski adds. Postdoctoral researcher Diane McCabe in his lab performed most of the experiments, and McCabe and Rutkowski worked closely with cancer biology expert Adam Dupuy, Ph.D., UI associate professor of anatomy and cell biology, and his graduate student Jesse Riordan. Another collaboration with Michael Icardi, M.D., UI associate professor of pathology, gave Rutkowski access to liver tissue samples from patients that allowed the team to show the association between elevated CHOP and human liver cancer.
Having implicated CHOP as a contributing factor in liver cancers associated with obesity, alcoholism, and hepatitis, Rutkowski next wants to learn whether CHOP acts early in the process of tumor formation or if it plays a role in helping established tumors to grow. He also is interested in identifying the other proteins that partner with CHOP to promote liver cancer.
"This discovery opens up an avenue into a new pathway that promotes liver cancer," he says. "Once we know what those other genes are that interact with CHOP, then maybe we can find a 'druggable' target molecule. The hope is that down the line scientists will be able to convert that finding into something therapeutically useful for patients.
"Federal funding is the backbone of this kind of research, which has the potential to make unexpected discoveries that, in this case, could help improve cancer treatment," he adds.

Diane DeZwaan-McCabe, Jesse D. Riordan, Angela M. Arensdorf, Michael S. Icardi, Adam J. Dupuy, D. Thomas Rutkowski. The Stress-Regulated Transcription Factor CHOP Promotes Hepatic Inflammatory Gene Expression, Fibrosis, and Oncogenesis. PLoS Genetics, 2013; 9 (12): e1003937 DOI: 10.1371/journal.pgen.1003937

http://www.eurekalert.org/pub_releases/2013-12/kf-srr122713.php

Stroke researchers report improvement in spatial neglect with prism adaptation therapy

Benefits shown in subset of patients with spatial neglect following right brain stroke. Findings support behavioral classification and early intervention

West Orange, NJ. - Stroke rehabilitation researchers report improvement in spatial neglect with prism adaptation therapy. This new study supports behavioral classification of patients with spatial neglect as a valuable tool for assigning targeted, effective early rehabilitation. Results of the study, "Presence of motor-intentional aiming deficit predicts functional improvement of spatial neglect with prism adaptation" DOI: 10.1177/1545968313516872 were published ahead of print in Neurorehabilitation and Neural Repair on December 27, 2013.

The article is authored by Kelly M. Goedert, PhD, of Seton Hall University, Peii Chen, PhD, of Kessler Foundation, Raymond C. Boston, PhD, of the University of Pennsylvania, Anne L. Foundas, MD, of the University of Missouri, and A.M. Barrett, MD, director of Stroke Rehabilitation Research at Kessler Foundation, and chief of Neurorehabilitation Program Innovation at Kessler Institute for Rehabilitation. Drs. Barrett and Chen have faculty appointments at Rutgers New Jersey Medical School.

Spatial neglect, an under-recognized but disabling disorder, often complicates recovery from right brain stroke," noted Dr. Barrett. "Our study suggests we need to know what kind of neglect patients have in order to assign treatment." The research team tested the hypothesis that classifying patients by their spatial neglect profile, i.e., by Where (perceptional-intentional) versus Aiming (motor-intentional) symptoms, would predict response to prism adaptation therapy. Moreover, they hypothesized that patients with Aiming bias would have better response to prism adaptation recovery than those with isolated Where bias.

The study involved 24 patients with right brain stroke who completed 2 weeks of prism adaptation treatment. Participants also completed the Behavioral Inattention Test and Catherine Bergego Scale (CBS) tests of neglect recovery weekly for 6 weeks. Results showed that those with only Aiming deficits improved on the CBS, whereas those with only Where deficits did not improve. Participants with both types of deficits demonstrated intermediate improvement. "These findings suggest that patients with spatial neglect and Aiming deficits may benefit the most from early intervention with prism adaptataion therapy," said Dr. Barrett. "More broadly, classifying spatial deficits using modality-specific measures should be an important consideration of any stroke trial intending to obtain the most valid, applicable, and valuable results for recovery after right brain stroke."

Study supported by Kessler Foundation, the National Institutes of Health and the National Institute of Disability and Rehabilitation Research (K02 NS 047099, R01 NS 055808, K24 HD062647, H133 G120203 PI: Barrett).

http://www.sciencedaily.com/releases/2013/12/131229112055.htm

Researchers Have Breakthrough On How Persistent Bacteria Avoid Antibiotics

The mechanism by which some bacteria are able to survive antibacterial treatment has been revealed for the first time by Hebrew University of Jerusalem researchers.

Their work could pave the way for new ways to control such bacteria.

In addition to the known phenomenon by which some bacteria achieve resistance to antibiotics through mutation, there are other types of bacteria, known as "persistent bacteria," which are not resistant to the antibiotics but simply continue to exist in a dormant or inactive state while exposed to antibacterial treatment. These bacteria later "awaken" when that treatment is over, resuming their detrimental tasks, presenting a dilemma as to how to deal with them. Until now, it had been known that there is a connection between these kind of bacteria and the naturally occurring toxin HipA in the bacteria, but scientists did not know the cellular target of this toxin and how its activity triggers dormancy of the bacteria.

Now, the Hebrew University researchers, led by Prof. Gadi Glaser of the Faculty of Medicine and Prof. Nathalie Balaban of the Racah Institute of Physics, have been able to demonstrate how this comes about. Their research showed that when antibiotics attack these bacteria, the HipA toxin disrupts the chemical "messaging" process necessary for nutrients to build proteins. This is interpreted by the bacteria as a "hunger signal" and sends them into an inactive state, (dormancy) in which they are able to survive until the antibacterial treatment is over and they can resume their harmful activity.

The research on persistent bacteria has been conducted in Prof. Balaban's lab for several years, focusing on the development of a biophysical understanding of the phenomenon. It will be combined with other work being done in Prof. Glaser's laboratory focusing on combating persistent bacteria, in the hope of leading to more effective treatment for bacterial infections.