http://news.discovery.com/animals/dog-domestication-prehistoric-ice-age-110728.htm

Prehistoric Dog Domestication Derailed by Ice Age

Some dogs were domesticated by 33,000 or more years ago, but the Ice Age disrupted the process.

By Jennifer Viegas | Thu Jul 28, 2011 05:00 PM ET

Some dogs were domesticated by at least 33,000 years ago, but these canines did not generate descendants that survived past the Ice Age, suggests a new PLoS ONE study.

The theory, based on analysis of a 33,000-year-old animal that may have been a partly domesticated dog, explains why the remains of possible prehistoric dogs date to such early periods, and yet all modern dogs appear to be descended from ancestors that lived at the end of the Ice Age 17,000-14,000 years ago.

The ancient animal identified as being a partly domesticated dog was found in Razboinichya Cave in the Altai Mountains of southern Siberia.

"The Razboinichya dog find demonstrates that the right wolf/human conditions suitable for getting domestication started were present at least 33,000 years ago," co-author Susan Crockford told Discovery News. "However, such conditions would have had to be present continuously - stable - for many wolf generations, perhaps 20 over about 40 years for the domestication process to generate a true dog."

"It appears that such stable conditions were not present until after the Ice Age, sometime after 19,000 years ago," added Crockford, a researcher at Pacific Identifications Inc. and author of the book "Rhythms of Life. Even after the Ice Age, domestication of wolves could have got started at several different times and places, and still failed because the conditions were not continuous enough for the changes to become permanent."

The Siberian animal was unearthed some years ago, but was only recently dated to 33,000 years ago by three independent radiocarbon dating facilities. Crockford and her colleagues conclude that it was a partly domesticated dog because of its mixture of dog and wolf features.

Based on its skull and other remains, the scientists believe it was about the size and shape of a large male Samoyed dog. Its teeth were still wolf-sized, however, and "it probably behaved more like a wolf than a dog."

Its remains were excavated from a cave area containing wild animal bones. Usually fully domesticated dogs, even very early ones, received more careful burials, often being placed in graves with, or next to, their owners.

Since no other dog-like animals were found at the site, the researchers think this animal was an "incipient" dog in the early stages of domestication. The scientists hold that domestication can happen naturally, without direct human intervention, when wolves are attracted to settlements and gradually adjust to a human-dependent lifestyle. The Ice Age, however, changed the abundance and migration patterns of the animals that the people in the Altai Mountains of Siberia hunted for food. "As a result, the people probably had to move more often than they did before," she explained.

Without the conditions that fuel domestication, the dog or dog-like animals gradually died off, the researchers suspect. Dogs reemerged after the Ice Age, reproducing and becoming the ancestors to today's modern dogs. It is unclear when the first pre-Ice Age dogs emerged, but a dog-like skull dating to 36,500 years ago was found at Goyet Cave in Belgium. It's possible then that the first dogs appeared in parts of Europe and Asia much earlier than commonly thought.

Other experts contacted by Discovery were interested in the new study, but would like to see more specimens to strengthen the theory. For example, Keith Dobney, chair of human palaeoecology at the University of Aberdeen's Department of Archaeology, said, "This is a very interesting and potentially important find - potentially pushing the domestication of the dog much further back."

Without more specimens, however, he said it cannot be ruled out that the Siberian dog, and possibly some of the other pre-Ice Age animals, were different representatives of now-extinct wolves.

Richard Meadow, director of the Zooarchaeology Laboratory at Harvard University's Peabody Museum, echoed Dobney's reservations about the study's conclusions.

Crockford admits that the paper presents "a new way of thinking about domestication, but it fits the evidence better than the idea that people deliberately created dogs for some specific purpose."

http://www.physorg.com/news/2011-07-bacterial-resistance-antibiotics-resist.html

Bacterial resistance to antibiotics: The more they resist, the more they divide

Bacteria acquire resistance to antibiotics through mutations and by incorporating new genes. When both mechanisms of resistance are playing out in Escherichia coli (E. coli), its ability to survive and reproduce is increased.

The number of multiresistant strains of bacteria in hospitals is increasing. Bacteria acquire resistance to antibiotics through mutations in their chromosomes and by incorporating new genes, either from the surrounding environment or from other bacteria. Now, a research team at the Portuguese CBA research

(University of Lisbon) and the Instituto Gulbenkian de Ciência has shown that, surprisingly, when both mechanisms of resistance are playing out in the bacterium Escherichia coli (E. coli), its ability to survive and reproduce is increased. These results are now published in the open-access journal PLoS Genetics.

Usually, the acquisition of new genes, either through the insertion of pieces of DNA – called plasmids – or through mutations, comes at a cost to the bacteria, reflected in a reduction in its rate of cell division, for example. Francisco Dionísio, senior author of the paper, describes the process using the following analogy: "if you disassembled your computer and randomly changed connections and pieces, you wouldn't expect it to work better than before."

However, Francisco and his colleagues show that, when a mutation occurs in the chromosome of a bacterium that has already incorporated a resistance-carrying plasmid, the bacteria divide faster in 10% of the mutation-plasmid combinations tested. Similarly, bacteria that first acquire resistance to antibiotics through mutation of their chromosome and then gain further resistance by insertion of plasmids into their DNA show reproduction rate increases in 32% of combinations.

In 2009, the same research groups showed, for the first time, the importance of interactions between random genes in determining antibiotic resistance in bacteria. This latest study takes their initial findings a step further, by demonstrating that this is a general phenomenon, and thus may help to predict how a bacterial population will evolve after receiving a plasmid that confers resistance to a certain antibiotic.

Francisco Dionísio adds "These results are, at least, unexpected in light of what we previously knew about genetic interactions, and may underlie the mechanism whereby rapid resistance to antibiotics appeared.

Provided by Instituto Gulbenkian de Ciencia

http://news.discovery.com/human/population-boom-110729.html

Population to Bulge, But Will Hit Ceiling

Speculation on population numbers raise questions about whether the planet can sustain us all.

By Emily Sohn | Fri Jul 29, 2011 08:00 AM ET

The world's population is on track to hit seven billion this year, which is double the number of people that lived on Earth in the 1960s, but far from what the future holds. By 2100, according to recent projections by the United Nations, we'll hit the 10 billion mark.

Those numbers, which are drastically larger than anything the Earth has experienced before, have sparked concerns about how all of those people will impact the world. They have also raised questions about whether the planet can sustain us all in the first place.

There may, however, be at least some end in sight to the relentless swelling of population pressure. Around the end of the century, many demographers believe, the global population will gradually level off.

Researchers can't predict with certainty exactly when that will happen and at what level. Also up for debate is how the current level of population growth will impact the environment, the economy and quality of life.

Overall, though, the level of rapid population growth we are experiencing today cannot be considered a good thing, said John Bongaarts, a demographer at the Population Council, a research organization in New York City.

In some parts of Africa, for example, population is doubling every 20 years, making it impossible for communities there to keep up with the growing demand for housing, roads, schools and health clinics. To many experts, those kinds of issues highlight the need for a global-wide investment in family planning programs that provide women education and access to contraception.

"Every billion people we add to the planet makes life more difficult for everyone and will do more damage to the environment," Bongaarts said. "Can we support 10 billion people? Probably. But we would all be better off with a smaller population."

The multiplication of people on the planet wasn't always so explosive, according to a series of papers in this week's Science. Growth started to accelerate with industrialization around 1750, said Ron Lee, a demographer and economist at the University of California, Berkeley.

By 1800, global population reached one billion for the first time. It took another 125 years to reach two billion. After that, though, numbers rose from three to seven billion in just the last 50 years. The population growth rate reached a peak of two percent per year in the mid 1960s, before declining to today's annual growth rate of 1.1 percent.

No one can predict the future, but the U.N. has done a good job in the past of estimating population sizes for several decades forward. By 2050, its predictions range from 8.1 billion to 10.6 billion. For 2100, projections range from 6.2 billion to 15.8 billion.

There's nothing magic about the 10 billion number," Lee said. "On the other hand, there's pretty good agreement to expect something like this leveling off."

Longer life spans and lower death rates help explain why population size is growing at its current pace. But the variable that will make the biggest difference in how many people will live on Earth 100 years from now is fertility rate, or the number of babies that women give birth to.

If every woman had two babies, the world's population would remain stable. Today, there is a global average of 2.5 births per woman - down from five in 1950. That comes with huge geographical variation.

In Japan, China and Europe, women are having fewer than two babies, while women in many developing countries are still having five or more. Ninety-seven percent of the projected population increase over the next century is expected to happen in developing nations, according to a review article in Science by David Bloom, of the Harvard School of Public Health in Boston. Nearly half of the growth will be in Africa.

Women who matter more to society and are given access to education, according to previous research, end up having fewer children. Informing people about contraception and making it available also make a big difference, and not just in places like Africa, where a disproportionately large population of young people is exacerbating problems like school overcrowding and unemployment. In the United States, Bongaarts said, about 15 percent of births are unwanted.

Even as the global population begins to level off in the coming decades, experts are already expressing concern about the environmental and economic consequences of stuffing so many people onto the planet. Parts of the world are running out of water. Prices of food and energy continue to rise.

"It's not clear how this is going to sort out," Lee said. "That's what I'm worried about."

http://www.newscientist.com/article/mg21128225.500-stegobot-steals-passwords-from-your-facebook-photos.html

Stegobot steals passwords from your Facebook photos

29 July 2011 by Jacob Aron

THINK twice before uploading your holiday pictures to Facebook - you could be helping someone to steal information from your computer.

A botnet called Stegobot was created to show how easy it would be for a crook to hijack Facebook photos to create a secret communication channel that is very difficult to detect.

Like most botnets, Stegobot gains control of computers by tricking users into opening infected email attachments or visiting suspect websites. But rather than contacting the botmasters directly, it piggybacks on the infected user's normal social network activity. "If one of your friends is a friend of a friend of the botmaster, the information transfers hop by hop within the social network, finally reaching the botmasters," says Amir Houmansadr, a computer scientist at the University of Illinois at Urbana-Champaign who worked on the botnet.

Stegobot takes advantage of a technique called steganography to hide information in picture files without changing their appearance. It is possible to store around 50 kilobytes of data in a 720 by 720 pixel image - enough to transmit any passwords or credit card numbers that Stegobot might find on your hard drive.

The botnet inserts this information into any photo you upload to Facebook, and then waits for one of your friends to look at your profile. They don't even have to click on the photo, as Facebook helpfully downloads files in the background. If your friend is also infected with the botnet - quite likely, since any email you send them will pass it on - any photo they upload will also pass on the stolen data.

From there, the data will eventually make its way to the account of someone who is also friends with the botmaster, allowing them to extract details on your identity. The botmasters can also send commands to the botnet through the reverse process - uploading a photo with hidden instructions that make their way to infected computers.

"It's scary because it's virtually undetectable," says Shishir Nagaraja of the Indraprastha Institute of Information Technology, New Delhi, India, who led the project.

Marco Cova, a computer scientist at the University of Birmingham, UK, says that criminals could employ a system like Stegobot, as it is hard to detect, but other methods allow them to steal much larger quantities of data. "It's not the most efficient or convenient way," he says.

http://medicalxpress.com/news/2011-07-drug-benefits-breast-cancer-deaths.html

Drug's lasting benefits sees breast cancer deaths down by third

The benefits of using tamoxifen to prevent recurrence of breast cancer after surgery continue to accrue long after women stop taking the drug, a study led by Oxford University has found.

The findings suggest that for women with the most common type of breast cancer, full compliance with daily tamoxifen therapy for five years would reduce the long-term chances of dying by at least a third.

"Breast cancer is a nasty disease because it can come back years later," says Dr. Christina Davies of the Clinical Trial Service Unit at Oxford University, and one of the lead investigators. "This study now shows that tamoxifen produces really long-term protection.

"For ER-positive disease, tamoxifen reduces 15-year breast cancer mortality by at least a third, whether or not chemotherapy has been given."

Most breast cancers are oestrogen receptor (ER)-positive – in the US or UK, it's about 4 out of 5 breast cancers that are ER-positive. Since tamoxifen acts on the ER protein in breast cancer cells, it can have an effect only if those cells contain some ER protein. But a simple test on surgically removed breast cancers can determine whether the cancer is ER-positive or not.

Various treatments can be given after apparently successful breast cancer surgery to prevent any tiny residual fragments eventually causing the cancer to come back as an incurable disease.

Many randomized trials have been conducted to try to determine the best treatment options, and every 5 years for the past 25 years the Early Breast Cancer Trialists' Collaborative Group (EBCTCG) has brought together all the evidence from all of these trials.

In the current study, funded by Cancer Research UK, the Medical Research Council and the British Heart Foundation, the researchers brought together individual patient data for over 20,000 women with early-stage breast cancer from 20 randomised trials. The trials compared treatment with tamoxifen for 5 years against no tamoxifen, with participants showing 80% compliance in taking the daily pill.

Most of the trials of tamoxifen began in the 1980s, meaning there is now lots of data available on the long-term effects of treatment after women stopped taking the drug. That long-term analysis, published in the Lancet medical journal, now reveals large additional benefits of tamoxifen in reducing breast cancer deaths, not only during the first decade but also during the second decade after treatment began.

The researchers found that in women with ER-positive disease, 5 years of daily tamoxifen safely reduced the long-term (15-year) risks of breast cancer recurrence and death. It was effective whether or not chemotherapy had been given.

Remarkably, the researchers found a highly significant reduction in breast cancer mortality, not only during the five years of treatment and the five years following, but also during years 10–14.

Even in weakly ER positive disease, tamoxifen substantially reduced the likelihood of the cancer recurring.

There is a newer class of drugs called aromatase inhibitors (AIs) that offer an alternative to tamoxifen for some patients, but AIs are effective only in post-menopausal women.

Dr. Davies explains that tamoxifen was developed 50 years ago and is long out of patent, so is relatively cheap. But even if costs are ignored it remains a major first-line treatment option for women with ER-positive breast cancer, she says – especially for women pre-menopause.

Moreover, the rare life-threatening side-effects of tamoxifen (uterine cancer and blood clots) are mainly experienced by women over 55 years of age, so there is little risk from giving tamoxifen to younger women.

Worldwide, half of all new patients diagnosed with breast cancer are younger than 55 years – that's 0.7 million women.

More information: doi:10.1016/S0140-6736(11)60993-8 Provided by Oxford University

http://www.bbc.co.uk/news/science-environment-14316028

Dyslexia makes voices hard to discern, study finds

By Jennifer Carpenter Science reporter, BBC News

People with dyslexia struggle to recognise familiar voices, scientists suggest.

The finding is the first tentative evidence that small sounds in the human voice that vary between people are difficult for dyslexics to hear. Writing in the journal Science, the scientists say that many people could have some degree of "voice blindness". And by studying it, scientists hope to better understand how the human brain has evolved to recognise speech.

Humans rely on small sounds called phonemes to tell one person from another. As we first try to form the word dog, for example, phonemes are the "duh"-"og"-"guh" sounds that our parents prompt us to make. But as we master the ability to read, we become less reliant on recognising these sounds to read, and eventually stop noticing them. Despite ignoring them, however, phonemes remain important for voice recognition.

The tiny inflections in the way people pronounce phonemes gives a listener cues to tell one voice from another. Because people who suffer from dyslexia are known to struggle with phonemes when reading, a US-based team of scientists wondered whether they might also struggle hearing them in people's voices.

Listen well

To investigate, the team grouped 30 people of similar age, education and IQ into two camps: those with and without a history of dyslexia. The subjects then went through a training period to learn to associate 10 different voices - half speaking English and half speaking Chinese - with 10 computer-generated avatars. The subjects were then later quizzed on how many of those voices they could match to the avatars.Non-dyslexics outperformed people with a history of dyslexia by 40% when listening to English.

However, this advantage disappeared when the groups were listening to Chinese.

Dorothy Bishop from the University of Oxford thinks that this is because "when [they] are listening to Chinese, it is a level playing field, because no one has learned to hear [Chinese] phonemes".

The researchers think that dyslexics don't have as comprehensive a phoneme sound library in their heads, and so they struggle when they hear phonemes spoken by unfamiliar voices because their "reference copy" isn't as well-defined. "It is a very interests result... the only thing that I would really like to see to convince me... is if they were to repeat the experiment using Jabberwocky." Using Jabberwocky, the nonsense poem by Lewis Carroll, would allow the researchers to determine whether the listeners identify who's who from the meaning of what they are saying, or whether listeners are purely relying on the phonemes.

Dr Bishop speculated that non-dyslexics may be worse at extracting the meaning of the words, meaning they under perform in this task.

Understanding the mechanics of voice recognition is important, said the study's lead author Tyler Perrachione from the Massachusetts Institute of Technology in Cambridge, US, because it allows a listener to pinpoint a familiar voice above the hubbub of a crowded room. Mr Perrachione explained that very little is known about voice blindness, which is formally called phonagnosia.

"In reality, phonagnosia is probably much more common," he explained, "but people who don't recognize that voices sound different may not even realize they lack the ability to tell voices apart."

http://www.scientificamerican.com/article.cfm?id=what-causes-prejudice-aga

What Causes Prejudice against Immigrants, and How Can It Be Tamed?

Hostility toward others can explode into senseless violence. Reciprocal relationships and trust are keys to preventing such tragedies

By Sophie Bushwick | Friday, July 29, 2011 | 22

In the wake of the bombing in Oslo and the shooting on Utoya Island in Norway, the spotlight has focused on confessed perpetrator Anders Behring Breivik. What drove the Norwegian citizen with extremist right-wing views to these mass killings? Although one of the terrorist's driving motives was anti-immigrant sentiment, he also killed fellow Norwegians belonging to his own ethnic group.

Why do human beings develop this kind of prejudice, and what makes it sometimes erupt into violence? Scientific American spoke with Steven Neuberg, a professor of psychology at Arizona State University in Tempe, about the psychology of anti-immigrant prejudice. [An edited transcript of the interview follows.]

How would you define prejudice in psychological terms?

Prejudice is traditionally defined in social psychology as a negative feeling towards a particular group and its members. It turns out, though, that there are different kinds of prejudices and different prejudices towards different groups - and these prejudices have very different emotional components to them. For instance, towards some groups, the prejudice is characterized by disgust, others by anger, yet others by fear.

What underlies prejudice against foreigners?

We're highly dependent on people in our own groups. In fact, one could argue that our highly ultrasocial, interdependent form of group living may be the most important human adaptation. People tend to be invested in members of their groups, to have ongoing histories of fair exchanges and reciprocal relations, to treat one another reasonably well, to create and follow a set of agreed-upon norms, and thereby build up trust. Outsiders aren't going to have that same built-up investment in us or our group. Because of this, we tend to believe that people who are foreign to us are more likely to pose certain kinds of threats: We believe they may be more interested in taking our resources, more likely to cheat us in exchanges, to violate our norms and values, to take more than their fair share, and the like. These perceptions of threats are linked to negative emotions such as anger and moral disgust that contribute to anti-immigrant prejudices.

My colleague Mark Schaller at the University of British Columbia has explored an additional threat that people are likely to see in foreigners: People who come from faraway places, who live in somewhat different ecologies, carry different pathogens within their bodies - pathogens that their immune systems have had an opportunity to adapt to but that ours have not. Schaller's work shows that people perceived as being foreign - perhaps because they look different than us, speak different languages, eat different foods - automatically activate perceptions of disease threat. And groups who are perceived to pose disease threats activate prejudices characterized by physical disgust.

The alleged attacker in Norway, Anders Behring Breivik, had strong anti-immigrant prejudices. What was he feeling?

I can't tell you exactly what he was thinking, but as I mentioned, foreign groups coming into one's own society - immigrants - activate perceptions of a wide range of threats and elicit accompanying negative emotions such as anger, disgust and fear, which increases the likelihood of discrimination. If the perceived threats and emotions

are strong enough, an individual may believe that he needs to rid his country of those who pose them. Moreover, anger and disgust, together, contribute to feelings of contempt, which we feel towards others we believe to be "less" than us, and can serve to motivate extreme actions.

It's useful to note a couple of things here. First, because immigrants are perceived to pose multiple kinds of threats, they are likely to be on the receiving end of especially pernicious prejudices and acts of discrimination. Second, such reactions to immigrants are nothing new - and we can look not only to current anti-immigrant sentiments throughout the world, but also to our own history in the U.S. Whether it was Italians or Irish, Poles, Jews, Germans, Chinese or whomever, each of these groups were initially perceived to pose a wide range of threats and consequently evoked powerful prejudices. It was only once people came to see these groups as nonthreatening, usually as they were seen to adopt "American" norms, that they were perceived as Americans.

Given his prejudice against immigrants, why did Breivik target ethnic Norwegians, his own people?

I haven't read his writings, but I hypothesize he was going after members of his group he saw as responsible for allowing the immigrant threat to exist. I think he saw the liberal politicians and government bureaucracy - whom he perceived as supporting Muslim immigration, cultural diversity and overall tolerance - as betraying the Norwegian people. Indeed, he attacked the liberal political class: The bomb was set off in a government center and the shootings took place at a camp for teenagers being educated in liberal politics. To Breivik, these folks may have been traitors because, to his mind, they were allowing immigrant Muslims to adulterate and contaminate his country. People seen as traitors are universally despised and stigmatized. Given how much humans, as social animals, invest in and depend upon their groups, betrayal of one's group is seen as one of the worst things one can possibly do. My guess is that Breivik saw the liberal politics of his country as a betrayal of his people, and so he attacked those politics and those engaged in them.

What makes someone like Breivik break and decide to use violence?

It's normal for people to over-perceive threats; our mind is designed to err in that direction. It's also normal for people, when confronted with the kinds of threats we've been discussing, to experience emotions like anger, disgust and fear. But just because we stereotype groups as posing certain threats, and hold certain prejudices against them, doesn't mean that we act on these stereotypes and prejudices in extreme ways. It just doesn't make sense to do so, and the normal mind typically weighs the consequences of engaging in such planned, extreme actions. I suspect that Breivik, and other extremists like him, possess a much lower threshold for perceiving others as threats and perhaps also a much more intense emotional reaction to those perceptions. Moreover, for someone like him, the ability to dive deeply into media that's like-minded, on the Web or otherwise, and to spend time with like-minded others, may significantly reinforce his sense of threat and his belief that something needs to be done about it. Like most rare, extreme behaviors, it takes a perfect storm - a psychological disposition shaped by genes and environment, in concert with current experiences, circumstances and opportunities.

What are some ways we can combat this kind of prejudice?

Prejudice against new immigrant groups is a natural aspect of our psychology. What's natural, however, isn't always good, and we can try to reduce inclinations to those prejudices we find morally problematic. Throughout history, immigrant groups that were once stigmatized very often end up accepted into society, because people come to understand that they aren't actually posing the threats they were once thought to pose. It helps when immigrant groups begin to adopt the norms and practices of their new homes, and the reduction of threat perceptions is furthered as people begin to form friendships across group lines.

How do friendships help?

Friendship entails interacting interdependently with another - sharing, taking turns, self-disclosing, and the like - and such actions reveal that many of the threats initially expected to exist may not be there after all. With friendship also comes a sense of "we," a sense that the person is like me and that we share something important and can trust them. Having a close friend that's a member of another group then provides a model that the group may not actually be as threatening as initially believed. As members of groups come to interact with one another more, the likelihood that they'll form friendships increases, and this will accelerate the reduction of prejudices.

Can we prevent prejudice from turning into violence?

I'm not very confident that we'll ever be able to eliminate the kinds of rare acts of violence we saw in Norway. I am, however, somewhat more optimistic that we'll be able to develop the behavioral and political "technologies" to reduce, or at least to manage, the more typical intergroup prejudices that characterize all of our everyday lives.

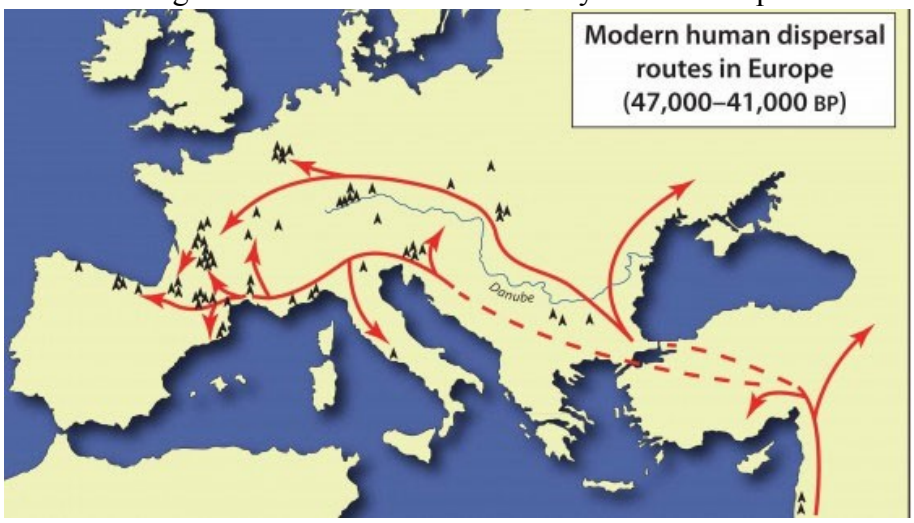
http://www.pasthorizonspr.com/index.php/archives/07/2011/strength-in-numbers-the-tide-of-homo-sapiens

Strength in numbers: the tide of Homo sapiens

New research sheds light on why, after 300,000 years of domination, European Neanderthals abruptly disappeared.

Researchers from the University of Cambridge have discovered that modern humans coming from Africa swarmed the region, arriving with over ten times the population as the Neanderthal inhabitants.

The reasons for the relatively sudden disappearance of the European Neanderthal populations across the continent around 40,000 years ago has for long remained one of the great mysteries of human evolution. After 300 millennia of living, and evidently flourishing, in the cold, sub–glacial environments of central and western Europe, they were rapidly replaced over all areas of the continent by new, anatomically and genetically 'modern' (i.e. Homo sapiens) populations who had originated and evolved in the vastly different tropical environments of Africa.



The most plausible answer to this long-debated question has now been published today, 29 July, in the journal Science by two researchers from the Department of Archaeology at Cambridge – Professor Sir Paul Mellars, Professor Emeritus of Prehistory and Human Evolution, and Jennifer French, a second-year PhD student.

By conducting a detailed statistical analysis of the archaeological evidence from the classic '' region of southwestern France, which contains the largest concentration of Neanderthal and early modern human sites in Europe, they have found clear evidence that the earliest modern human populations penetrated the region in at least ten times larger numbers than those of the local Neanderthal populations already established in the same regions. This is reflected in a sharp increase in the total number of occupied sites, much higher densities of occupation residues (i.e. stone tools and animal food remains) in the sites, and bigger areas of occupation in the sites, revealing the formation of much larger and apparently more socially integrated social groupings.

Faced with this dramatic increase in the incoming modern human population, the capacity of the local Neanderthal groups to compete for the same range of living sites, the same range of animal food supplies (principally reindeer, horse, bison and red deer), and the same scarce fuel supplies to tide the groups over the extremely harsh glacial winters, would have been massively undermined. Additionally, almost inevitably, repeated conflicts or confrontations between the two populations would arise for occupation of the most attractive locations and richest food supplies, in which the increased numbers and more highly coordinated activities of the modern human groups would ensure their success over the Neanderthal groups.

The archaeological evidence also strongly suggests that the incoming modern groups possessed superior hunting technologies and equipment (e.g. more effective and long-range hunting spears), and probably more efficient procedures for processing and storing food supplies over the prolonged and exceptionally cold glacial winters. They also appear to have had more wide-ranging social contacts with adjacent human groups to allow for trade and exchange of essential food supplies in times of food scarcity.

Whether the incoming modern human groups also possessed more highly developed brains and associated mental capacities than the Neanderthals remains at present a matter of intense debate. But the sudden appearance of a wide range of complex and sophisticated art forms (including cave paintings), the large-scale production of elaborate decorative items (such as perforated stone and ivory beads, and imported sea shells), and clearly 'symbolic' systems of markings on bone and ivory tools – all entirely lacking among the preceding Neanderthals – strongly point to more elaborate systems of social communications among the modern groups, probably accompanied by more advanced and complex forms of language.

All of these new and more complex behavioural patterns can be shown to have developed first among the ancestral African Homo sapiens populations, at least 20,0000 to 30,000 years before their dispersal from Africa, and progressive colonisation (and replacement of earlier populations) across all regions of Europe and Asia from around 60,000 years onwards.

If, as the latest genetic evidence strongly suggests, the African Homo sapiens and European Neanderthal populations had been evolving separately for at least half a million years, then the emergence of some

significant contrasts in the mental capacities of the two lineages would not be a particularly surprising development, in evolutionary terms.

"In any event, it was clearly this range of new technological and behavioural innovations which allowed the modern human populations to invade and survive in much larger population numbers than those of the preceding Neanderthals across the whole of the European continent. Faced with this kind of competition, the Neanderthals seem to have retreated initially into more marginal and less attractive regions of the continent and eventually – within a space of at most a few thousand years – for their populations to have declined to extinction – perhaps accelerated further by sudden climatic deterioration across the continent around 40,000 years ago.".

Professor Sir Paul Mellars, Professor Emeritus of Prehistory and Human Evolution at the Department of Archaeology

Whatever the precise cultural, behavioural and intellectual contrasts between the Neanderthals and intrusive modern human populations, this new study published in Science demonstrates for the first time the massive numerical supremacy of the earliest modern human populations in western Europe, compared with those of the preceding Neanderthals, and thereby largely resolves one of the most controversial and long-running debates over the rapid decline and extinction of the enigmatic Neanderthal populations.

http://www.eurekalert.org/pub_releases/2011-08/tju-ldr080111.php

Leukemia drug reverses tamoxifen-resistance in breast cancer cells

Researchers at the Kimmel Cancer Center at Jefferson demonstrate drug combination's 'antioxidant effect' on cancer cells and fibroblasts

PHILADELPHIA - Taking a leukemia chemotherapy drug may help breast cancer patients who don't respond to tamoxifen overcome resistance to the widely-used drug, new research from the Kimmel Cancer Center at Jefferson suggests.

Interestingly, researchers found that tamoxifen combined with dasatinib, a protein-tyrosine kinase inhibitor, reverses the chemo-resistance caused by cancer-associated fibroblasts in the surrounding tissue by normalizing glucose intake and reducing mitochondrial oxidative stress, the process that fuels the cancer cells.

Previous animal studies have confirmed that combining tyrosine kinase inhibitors with anti-estrogen therapies, like tamoxifen, can prevent drug resistance, but none have suggested that the target of the inhibitors is the cancer-associated fibroblasts. The researchers report their findings in the August 1 issue of Cell Cycle.

About 70 percent of women diagnosed with breast cancer will have estrogen receptor positive (ER(+)) disease, which indicates that the tumor may respond to tamoxifen. However, a large percentage of these tumors - up to 35 percent - have little to no response to the drug or eventually develop resistance to it.

In this study, researchers sought to better understand drug resistance by looking at the metabolic basis in an ER (+) cell line and cancer-associated fibroblasts. The researchers have previously established a relationship between the two, where cancer cells induce aerobic glycolysis by secreting hydrogen peroxide in adjacent fibroblasts via oxidative stress. In turn, these fibroblasts provide nutrients to the cancer cells to proliferate, a process that ultimately makes tumors grow.

Here, they investigated and then demonstrated that this interaction was also the basis of tamoxifen resistance.

In a sense, the drug combination had an "antioxidant effect" in these types of cancer cells, according to Michael P. Lisanti, M.D., Ph.D., Professor and Chair of Stem Cell Biology and Regenerative Medicine at Jefferson Medical College of Thomas Jefferson University and a member of the Kimmel Cancer Center.

"The fibroblasts are what make ER (+) cancer cells resistant to the tamoxifen," said Dr. Lisanti. "But the tamoxifen plus dasatinib maintained both fibroblasts and cancer cells in a 'glycolytic state,' with minimal oxidative stress and more cell death, most likely because of an absence of metabolic coupling. The supply between the two was cut. This suggests resistance to chemotherapeutic agents is a metabolic and stromal phenomenal," he added.

Researchers showed that ER (+) cancer cells alone responded to tamoxifen but when co-cultured with human fibroblasts had little to no effect. Similarly, dasatinib, a chemotherapy drug used to treat leukemia patients who can no longer benefit from other medications, had no effect on fibroblasts alone or cancer cells. Together, however, the drugs prevented the cancer cells co-cultured with the fibroblasts from using high-energy nutrients from the fibroblasts.

This combination resulted in nearly 80 percent cell death, the team reported - a two to three fold increase when compared with tamoxifen alone. "The drugs have no effect when they are used alone - it's in unison when they effectively kill the cancer cells in the presence of fibroblasts," said Dr. Lisanti. "This opens up the door for possible new treatment strategies. This 'synthetic lethality' may help patients overcome resistance in the clinic."

http://www.eurekalert.org/pub_releases/2011-08/uocd-rtp080111.php

Researcher tests promising drug on those with Down syndrome

Attempt to increase memory, learning a potential milestone in Down research

AURORA, Colo. – A University of Colorado School of Medicine scientist is completing a major clinical trial on a drug that could boost cognitive function in those with Down syndrome, significantly improving their quality of life and representing a potential milestone in research on this genetic condition.

"We are hoping to enhance memory and learning in those with Down syndrome," said Alberto Costa, MD, PhD, an associate professor of medicine and the neuroscientist leading the effort. "We have been studying this drug for three years and are now ready to analyze the data on our trial. Our team at the University of Colorado and Children's Hospital Colorado expects to have the results in the next two or three months."

Costa, whose work was chronicled in last Sunday's New York Times Magazine, is testing the drug memantine, currently used to relieve symptoms of Alzheimer's disease, in 39 people with Down syndrome. About half received the drug and the others a placebo. In 2007, Costa demonstrated that memantine could improve memory function in mice with Down syndrome. And now, for the first time, he is taking a drug effective in the treatment of learning and memory deficits in mice with Down syndrome and applying it to humans, a move described by the New York Times as "a milestone in the history of Down syndrome research."

Costa is no disinterested researcher. His 16-year-old daughter Tyche – named for the Greek goddess of Fortune - has Down syndrome. Like others with the condition, she faces the specter of a steady decline in mental functioning as she gets older and a roughly 20 percent chance of getting Alzheimer's in her 50's. After that diagnosis, death is often just five years away.

"I feel I am racing the clock to find something that will at least keep her functioning at the level she is at now," Costa said. "As they age, parts of their brain will shrink and their functions will diminish."

Costa is actively pursuing links between Down and Alzheimer's disease. He says babies born with Down often carry the biological markers for Alzheimer's. "They have the disease from the get go," he said.

Costa says the world is awash in false assumptions about Down syndrome ranging from distortions on life expectancy to educational limitations. In fact, depending on the severity of their condition, those with Down can live into their 70s, attend college, live independently and hold down jobs.

"If we are successful, it will increase hope and expectations for those with Down syndrome," Costa said. "Right now there are drugs for the signs and symptoms of medical conditions more frequent in those with Down syndrome, but nothing to improve brain function. In fact, the prevailing wisdom has been that there is essentially nothing you can do to boost memory and learning in this group. Hopefully, we can prove them wrong." But he and other Down researchers face an overall lack of federal funding, especially when compared to other diseases and disorders.

Costa has been supported by Forest Pharmaceuticals which is funding the clinical trial, the Linda Crnic Institute for Down Syndrome, the Coleman Institute for Cognitive Disabilities and the National Institute of Child Health and Development, part of the National Institutes of Health.

"Clearly these funding sources are the unsung heroes," Costa said. "They may not get the attention or publicity but I can assure you that our efforts and the future of those with Down syndrome would be seriously compromised without their continued generosity."

For more information on the clinical trial please go to NCT01112683 at http://www.clinicaltrials.gov.

http://www.newscientist.com/blogs/onepercent/2011/08/facial-recognition-identifies.html

Facial recognition identifies your social security number

Jacob Aron, technology reporter

A picture of your face is all it takes for Alessandro Acquisti at Carnegie Mellon University (CMU) to access a wealth of personal information.

He and colleagues used PittPatt facial recognition software - which was developed at CMU and recently bought by Google - to match people with their Facebook profiles and gather names, birth dates and other demographics for one in three test subjects.

The researchers then used this data to correctly predict the first five digits of the subjects' social security number, using a technique they developed in 2009. In principle it should be possible to identify the full number, but the sample size of 93 was too small to do this accurately.

This kind of identification relies on people sharing their information on social networks in the first place, but of course many happily do this. What they may not realise is that information from different networks or or even their friends can be linked, building up a surprisingly rich picture of their identify. "It's like a domino effect," says Acquisti, who will will present the research at the Black Hat security conference in Las Vegas this week. "It has become truly difficult for us to tightly seal our personal information."

Acquisti has also developed an augmented reality smartphone app that can perform the facial recognition in real time then overlay their online information in a matter of minutes. He says there are no plans to release the app to the public, but adds it wouldn't be too hard for someone else to replicate it. "My fear is that it's absolutely inevitable because the data is already public for most of us."

So what happens when such an app is on the market, or even incorporated into a smart pair of glasses? Acquisti imagines a near future in which you could walk in to a bar and scan through the crowd, telling your their name, interests or even their credit score. "It impacts the way you're going to approach and interact with a person before you've even met them," he says.

Some people will be horrified by this vision, but then they probably aren't using social networks in the first place. Acquisti says others are more enthusiastic about the benefits software, but may not have thought through the consequences. "People trade off privacy for immediate gains," he says. "Often we don't see the cost of revealing too much information until it's too late."

http://www.newscientist.com/article/dn20752-live-disc-implant-could-kill-back-pain.html

Live disc implant could kill back pain

20:00 01 August 2011 by Kate McAlpine

A live implant could kill the pain associated with slipped discs, a study in rats suggests.

Between 1.5 and 4 million Americans are waiting for surgery to fix a herniated spinal disc, but the relief provided from a synthetic implant is the best it's ever going to be "the minute you put it into the patient", says Lawrence Bonassar of Cornell University in Ithaca, New York. Living tissue can grow and adapt, so may provide a better long-term solution, he says.

Bonassar's team used cells taken from sheep spines to build replicas of rat discs, and implanted them into the spines of rats. The implanted discs stood up to pulling and compression like the original discs. Crucially, they also improved with age, growing new cells and binding to nearby vertebrae in the six months after surgery.

Although the study was in rats, "it shows us what is possible", says Abhay Pandit at the National University of Ireland in Galway. He adds that future studies will need to address the load borne by upright human spines.

Journal reference: Proceedings of the National Academy of Sciences, DOI: 10.1073/pnas.1107094108

http://www.scientificamerican.com/article.cfm?id=placenta-feeds-itself-to-fetus-during-starvation

Placenta Feeds Itself to Fetus in Times of Starvation

In times of starvation the placenta protects the fetus from brain dysfunction.

By Zoë Corbyn of Nature magazine

The placenta has long been thought of as a passive organ that simply enables a fetus to take up nutrients from its mother. But new research in mice shows that when calories are restricted, the placenta steps up to the plate-actively sacrificing itself to protect the fetal brain from damage.

Researchers at Cambridge University, UK, examined what happened to 10 fetuses from 8 mice when their pregnant mothers were deprived of food for 24 hours-as might happen in the wild - about mid-way through gestation. This point in pregnancy is critical in the development of the hypothalamus, the part of the brain that controls primal urges, including maternal instincts.

Behavioural neuroscientists Kevin Broad and Barry Keverne found that the placenta responded by breaking down its own tissues, recycling proteins inside its cells to provide a steady supply of nutrients to the developing hypothalamus despite the mother's interrupted food intake. Their study is published today in the Proceedings of the National Academy of Sciences.

"We didn't know before that this protection of the fetus goes on," says Keverne. "I expected the lack of food to affect the fetal brain and the placenta equally, but instead we see the placenta acting as an interface to make sure the fetuses' particular stage of brain development is protected."

Imprint of starvation

As well as causing placental breakdown, the enforced starvation had effects on the expression of some 'imprinted genes' in the placenta. Such genes, unique to mammals, are inherited by the fetus in the normal way but their expression is subject to 'epigenetic' chemical tweaks by the mother through the placenta. By silencing either the paternal or maternal copy of the gene, she is able to shape her offspring's genetic destiny during pregnancy.

One of the affected genes was Peg3, which regulates the number of neurons that produce oxytocin, a hormone that is important for maternal care, milk production and giving birth. Peg3 expression in the placenta is normally closely tied with expression of the gene in the fetus's brain. After the mother had gone 24 hours without food, the researchers measured a 35% decrease in Peg3 expression in the placenta. But Peg3 expression in the fetal hypothalamus actually increased.

The researchers say that as with nutrient supply, the fetus appears to be protected from the consequences of the mother's starvation - in this case downregulation of a gene that threatens to impair the next generation's mothering ability and therefore reproductive success.

The main message is that the fetal hypothalamus and placenta "are not independent tissues", says Keverne. "They have evolved together in such a way that built into the system is a genetic flexibility enabling the next generation to be primed to become good mothers."

The researchers didn't study whether the genetic changes in the placenta caused by lack of food then feed back to the mother-to-be, reducing her maternal instincts. But Michael Skinner, an expert in reproductive biology at Washington State University in Pullman, thinks that is possible and would like to see more work in this direction. The placenta, he points out, communicates with the mother's own hypothalamus. "The hormones being produced by the placenta are going to shift the mother's programming," he says. "Whether that will turn around and influence the brain of the mother is all speculation, but if [the starvation] was long enough it might."

Keverne stresses that the findings in mice cannot necessarily be extrapolated to humans-in whom such starvation studies can't be done ethically.

But he wonders whether measuring the expression levels of neural genes in the placenta once a baby is born might provide a window into the brain function of that baby. His experiment also showed changes in expression of over 200 non-imprinted genes in the placenta on food-deprivation. Forty-one of those genes have been associated with neurological disorders, and two have been identified as markers for schizophrenia.

"The brain was rescued by the placenta in this case," he says. "But presumably if the perturbation had been more serious it would have affected the fetus's hypothalamus, which you could pick up by looking at the placenta."

http://www.eurekalert.org/pub_releases/2011-08/plos-gaa072611.php

Guest authorship, a form of ghost writing, constitutes legal fraud

"Guest authorship is a disturbing violation of academic integrity standards, a practice which the authors also argue constitutes legal fraud.

"Guest authorship is a disturbing violation of academic integrity standards, which form the basis of scientific reliability" state two law experts in a robust attack on this unethical practice in a policy article in this week's PLoS Medicine - a practice which the authors also argue constitutes legal fraud.

Simon Stern and Trudo Lemmens from the Faculties of Law and Medicine at the University of Toronto, Canada argue that it is irrelevant whether publications with academic guest authors are factually accurate. Rather, ghostwriting of medical journal articles raises serious ethical and legal concerns, bearing on the integrity of medical research and scientific evidence used in legal disputes. Furthermore, the false respectability afforded to claims of safety and effectiveness through the use of academic investigators risks undermining the integrity of biomedical research and patient care - an integrity that also underpins the use of scientific evidence in the courtroom.

According to the authors, medical journals, academic institutions, and professional disciplinary bodies have failed to enforce effective sanctions. Some journals, such as PLoS Medicine, have called for bans on future submissions by authors who act as guests, formal retraction if unacknowledged ghostwriting is discovered after publication, and reporting of authors' misconduct to institutions. Although the authors agree that such actions may have an impact on academics concerned about their status and future publication options, they say that it is unclear whether journals can adequately monitor the practice.

Consequently, the authors make the case for more effectively deterring the practice of ghostwriting through the imposition of legal liability on the "guest authors" who lend their names to ghostwritten articles. The authors say: "We argue that a guest author's claim for credit of an article written by someone else constitutes legal fraud, and may give rise to claims that could be pursued in a class action based on the Racketeer Influenced and Corrupt Organizations Act [RICO]."

The authors continue: "The same fraud could support claims of "fraud on the court" against a pharmaceutical company that has used ghostwritten articles in litigation. This claim also appropriately reflects the negative impact of ghostwriting on the legal system."

Funding: *The research is supported by a grant from the Social Sciences and Humanities Research Council on The Promotion of Integrity in Biomedical Research. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.*

Competing Interests: The authors have declared that no competing interests exist.

Citation: *Stern S, Lemmens T (2011) Legal Remedies for Medical Ghostwriting: Imposing Fraud Liability on Guest Authors of Ghostwritten Articles. PLoS Med 8(8): e1001070. doi:10.1371/journal.pmed.1001070*

http://www.eurekalert.org/pub_releases/2011-08/cp-wdd072911.php

Why diets don't work? Starved brain cells eat themselves

A report in the August issue of the Cell Press journal Cell Metabolism might help to explain why it's so frustratingly difficult to stick to a diet.

When we don't eat, hunger-inducing neurons in the brain start eating bits of themselves. That act of self-cannibalism turns up a hunger signal to prompt eating.

"A pathway that is really important for every cell to turn over components in a kind of housekeeping process is also required to regulate appetite," said Rajat Singh of Albert Einstein College of Medicine.

The cellular process uncovered in neurons of the brain's hypothalamus is known as autophagy (literally self-eating.) Singh says the new findings in mice suggest that treatments aimed at blocking autophagy may prove useful as hunger-fighting weapons in the war against obesity. The new evidence shows that lipids within the so-called agouti-related peptide (AgRP) neurons are mobilized following autophagy, generating free fatty acids. Those fatty acids in turn boost levels of AgRP, itself a hunger signal.

When autophagy is blocked in AgRP neurons, AgRP levels fail to rise in response to starvation, the researchers show. Meanwhile, levels of another hormone, called ✔-melanocyte stimulating hormone, remain elevated. That change in body chemistry led mice to become lighter and leaner as they ate less after fasting, and burned more energy.

Autophagy is known to have an important role in other parts of the body as a way of providing energy in times of starvation. However, unlike other organs, earlier studies had shown the brain to be relatively resistant to starvation-induced autophagy."The present study demonstrates the unique nature of hypothalamic neurons in their ability to upregulate autophagy in response to starvation that is consistent with the roles of these neurons in feeding and energy homeostasis," the researchers wrote.

Singh said he suspects that fatty acids released into the circulation and taken up by the hypothalamus as fat stores break down between meals may induce autophagy in those AgRP neurons. Singh's research earlier showed a similar response in the liver.

On the other hand, he says, chronically high levels of fatty acids in the bloodstream, as happens in those on a high-fat diet, might alter hypothalamic lipid metabolism, "setting up a vicious cycle of overfeeding and altered energy balance." Treatments aimed at the pathway might "make you less hungry and burn more fat," a good way to maintain energy balance in a world where calories are cheap and plentiful.

The findings might also yield new insight into metabolic changes that come with age given that autophagy declines as we get older. "We already have some preliminary evidence there might be changes with age," Singh said. "We are excited about that." *Singh et al. Albert Einstein College of Medicine, Bronx, NY*

http://www.eurekalert.org/pub_releases/2011-08/wcs-aru080211.php

African rodent uses 'poison arrow' toxin to deter predators

Wildlife Conservation Society, University of Oxford, and National Museums of Kenya investigate first known mammal to use plant poison in defense

Woe to the clueless predator trying to make a meal of the African crested rat, a rodent that applies poisonous plant toxin to sponge-like hairs on its flanks, a discovery recently made by Jonathan Kingdon and colleagues from the National Museums of Kenya, the Wildlife Conservation Society, and University of Oxford.

In the only known instance of a mammal acquiring a lethal toxin from a plant for defense, the researchers have discovered where the African crested rat (or maned rat) gets its poison: the Acokanthera tree, the same source used by East African hunters for poison arrows.

The study appears online in the Proceedings of The Royal Society B. The authors include: Jonathan Kingdon, Chris Holland, Tom Gheysens, Maxime Boulet-Audet, and Fritz Vollrath of the University of Oxford; Bernard Agwanda of the National Museums of Kenya; and Margaret Kinnaird and Tim O'Brien of the Wildlife Conservation Society.

The African crested rat is the only mammal known that acquires its poison from a plant. The rodent masticates the poisonous bark of the Acokanthera tree and applies the mixture to its flank hairs, which absorb the poison like candle wicks. **Susan Rouse**

"The African crested rat is a fascinating example of how a species can evolve a unique set of defenses in response to pressure from predators," said Dr. Tim O'Brien, Senior Scientist of the Wildlife Conservation

Society and a co-author on the study. "The animal and its acquired toxicity is unique among placental mammals."

Scientists have long suspected that the African crested rat is poisonous, primarily due to the animal's specialized behavior, such as exposing a black-and-white coloration on its flanks when threatened by predators, and accounts of dogs becoming ill or dying after encounters with rats. The new discovery concerns the nature of the chemical defense. Instead of producing poison itself - as is the case with poisonous mammals such as the duck-billed platypus and solenodon - the African crested rat finds its toxin (called ouabain) in tree bark.

The researchers confirmed the hypothesis by presenting a wild-caught rat with branches and roots of the Acokanthera tree. The rodent proceeded to gnaw and masticate the bark (avoiding the leaves and fruit) and apply the "slaver" on its flanks. Further, the research team employed electron microscopes to examine the unique structure of the flank hairs. In doing so, they found that the perforated cylindrical structure of the hairs facilitates the rapid absorption of the poisonous saliva. Interestingly, ouabain has also been used by doctors for centuries as a clinical treatment against congestive heart failure.

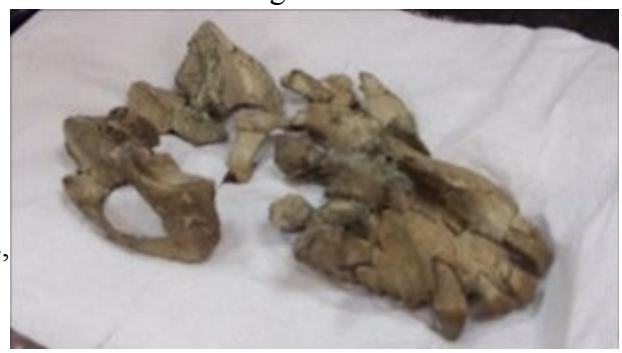
Besides its warning coloration and poisonous hairs, the African crested rat possesses a thick reinforced skull, thick vertebrae, and unusually tough skin, all protection for the small rodent that rarely grows to more than 2 pounds in weight. Several mysteries about the enigmatic rodent remain, including how the animal uses poison without succumbing to it.

http://www.bbc.co.uk/news/science-environment-14379207

Ancient primate fossil unearthed

Researchers working in Uganda say they have unearthed the well-preserved fossil skull of an ancient primate.

The 20 million-year-old specimen comes from the site of an extinct volcano in Uganda's north-east Karamoja region. The scientists say preliminary analysis showed the tree-climbing herbivore was roughly 10 years old when it died. The skull is about the same size as that of a chimp, but its brain was smaller.



The researchers say the skull belongs to a creature called Ugandapithecus major

"It is a highly important fossil and it will certainly put Uganda on the map in terms of the scientific world," Martin Pickford, a palaeontologist from the College de France in Paris, told journalists in Kampala. Dr Pickford and his colleague Brigitte Senut say the fossil skull belonged to a creature they call Ugandapithecus major.

Professor Senut, a professor at the French National Museum of Natural History said that the remains would be taken to Paris to be X-rayed and documented before being returned to Uganda.

"It will be cleaned in France, it will be prepared in France... and then in about one year's time it will be returned to the country," she said.

The remote and arid region of Karamoja is one of the least developed in Uganda.

http://www.eurekalert.org/pub_releases/2011-08/aeco-lot072811.php

Lifestyles of the old and healthy defy expectations

Einstein researchers find centenarians just as likely as the rest of population to smoke, drink and pack on pounds

Bronx, NY - People who live to 95 or older are no more virtuous than the rest of us in terms of their diet, exercise routine or smoking and drinking habits, according to researchers at Albert Einstein College of Medicine of Yeshiva University.

Their findings, published today in the online edition of Journal of the American Geriatrics Society, suggests that "nature" (in the form of protective longevity genes) may be more important than "nurture" (lifestyle behaviors) when it comes to living an exceptionally long life. Nir Barzilai, M.D., the Ingeborg and Ira Leon Rennert Chair of Aging Research and director of the Institute for Aging Research at Einstein, was the senior author of the study.

Dr. Barzilai and his Einstein colleagues interviewed 477 Ashkenazi Jews who were living independently and were 95 and older (95-112, 75 percent of them women). They were enrolled in Einstein's Longevity Genes Project, an ongoing study that seeks to understand why centenarians live as long as they do. (Descended from a small founder group, Ashkenazi Jews are more genetically uniform than other populations, making it easier to spot gene differences that are present.)

The elderly participants were asked about their lifestyles at age 70, considered representative of the lifestyle they'd followed for most of their adult lives. They answered questions about their weight and height so that their body mass index (BMI) could be calculated. They also provided information about their alcohol consumption, smoking habits, physical activity, and whether they ate a low-calorie, low-fat or low-salt diet.

To compare these long-lived individuals with the general population, the researchers used data from 3,164 people who had been born around the same time as the centenarians and were examined between 1971 and 1975 while participating in the National Health and Nutrition Examination Survey (NHANES I).

Overall, people with exceptional longevity did not have healthier habits than the comparison group in terms of BMI, smoking, physical activity, or diet. For example, 27 percent of the elderly women and an equal percentage of women in the general population attempted to eat a low-calorie diet. Among long-living men, 24 percent consumed alcohol daily, compared with 22 percent of the general population. And only 43 percent of male centenarians reported engaging in regular exercise of moderate intensity, compared with 57 percent of men in the comparison group.

"In previous studies of our centenarians, we've identified gene variants that exert particular physiology effects, such as causing significantly elevated levels of HDL or 'good' cholesterol," said Dr. Barzilai, who is also professor of medicine and of genetics at Einstein. "This study suggests that centenarians may possess additional longevity genes that help to buffer them against the harmful effects of an unhealthy lifestyle."

The research did find, however, that overweight centenarians tended to have lower rates of obesity than the control group. Although male and female centenarians were just as likely to be overweight as their counterparts in the general population, the centenarians were significantly less likely to become obese: only 4.5 percent of male centenarians were obese vs. 12.1 percent of controls; and for women, 9.6 percent of centenarians were obese versus 16.2 percent of controls. Both of these differences are statistically significant.

While longevity genes may protect centenarians from bad habits, healthy lifestyle choices remain critical for the vast majority of the population. The U.S. Census Bureau estimates there were nearly 425,000 people aged 95 and older living in the U.S. in 2010 − a fraction (.01) of the 40 million U.S. adults 65 and over.

"Although this study demonstrates that centenarians can be obese, smoke and avoid exercise, those lifestyle habits are not good choices for most of us who do not have a family history of longevity," said Dr. Barzilai. "We should watch our weight, avoid smoking and be sure to exercise, since these activities have been shown to have great health benefits for the general population, including a longer lifespan."

Researchers also asked study participants why they believed they had lived so long. Most did not attribute their advanced age to lifestyle factors. One-third reported a history of family longevity, while 20 percent believed that physical activity also played a role in their lifespan. Other factors included positive attitude (19 percent), busy or active life (12 percent), less smoking and drinking (15 percent), good luck (8 percent), and religion or spirituality (6 percent).

The paper is titled "Lifestyle Factors of People with Exceptional Longevity." Other Einstein authors were Yingheng Liu, Ph.D., Orit Ben-David, Saritha Reddy, M.B.B.S., Gil Atzmon, Ph.D., and Jill Crandall, M.D., Swapnil N. Rajpathak, M.B.B.S., Dr.PH., now at Merck Pharmaceuticals, was the study's lead author. The research was supported by grants from the National Institute on Aging of the National Institutes of Health.

http://www.eurekalert.org/pub_releases/2011-08/ohs-siw080211.php

Scientists identify what makes us feel 'bad' when we're sick, how to treat it

New study demonstrates that a new class of drugs designed to treat narcolepsy will also be effective in reversing illness-induced lethargy

PORTLAND, Ore. - A signaling system in the brain previously shown to regulate sleep is also responsible for inducing lethargy during illness, according to research conducted at Oregon Health & Science University Doernbecher Children's Hospital.

This research is particularly meaningful because it implies that a new class of drugs developed to treat sleep disorders can reverse the inactivity and exhaustion brought on by acute illness. Although the sleep drugs were initially designed to treat narcolepsy, they have the potential to restore energy and motivation in patients with acute and chronic disease, the researchers report. Their findings are published in the The Journal of Neuroscience.

"We all know what it means to feel 'bad' when we're acutely ill. In particular, patients with chronic diseases experience a compromise in motivated behaviors. They don't feel like getting up and doing anything. Yet the brain mechanisms behind this common experience have remained obscure," said Daniel L. Marks, M.D., Ph.D., principal investigator and associate professor of pediatrics in the Papé Family Pediatric Research Institute at OHSU Doernbecher Children's Hospital.

"Our lab has found that the neurotransmitter system thought to be primarily involved in the induction of sleep is actually extremely important in maintaining motivation and movement during acute and chronic illness," Marks explained.

Research has shown that in response to illness, animals divert all their energy to fight infection. Lethargy, fever and loss of appetite are symptoms of the body's highly organized strategy to sacrifice biological and physiological priorities to provide the greatest chance of survival.

Although previous studies have identified the neurotransmitter system in the brain responsible for inducing fever and loss of appetite in response to disease, the mechanism for suppressing physical activity and motivation, and the means to treat it, has been unclear - until now.

To determine the cause of illness-induced lethargy, Marks and colleagues studied the brains of rats, the neuroactivity of which closely mimics human brains. They determined that acute and chronic inflammation-induced lethargy is brought about by a specific population of inflammation-sensitive neurons located near the neurotransmitter system that controls physical activity and arousal, known as the orexin system. When the researchers injected the rats with orexin, they were able to restore orexin signaling and, thus, restore motivated behaviors and movement.

This research demonstrates that orexin replacement is a viable therapeutic avenue for sickness-induced lethargy, the researchers explain. Because chronic disruption of this crucial neurotransmitter system leads to profound sleep disturbance and loss of motivated behaviors, they propose that drugs that mimic orexin would be useful in chronically ill patients to improve quality of life and independent living.

"There is a very exciting opportunity to quickly translate these findings into clinical practice," Marks said. "Because the role of orexin in sleep disorders like narcolepsy has been known for several years, the drug development efforts aimed at restoring orexin signaling are at an advanced state and nearly ready for clinical application."

The study, "Inflammation-induced lethargy is mediated by suppression of orexin neuron activity," was funded by the National Institutes of Health.

Other members of the research team include: Aaron Grossberg, XinXia Zhu, Pete Levasseur, and Theodore Braun, all of Oregon Health & Science University; and Gina Marie Leinninger and Martin Myers, of the University of Michigan

http://www.eurekalert.org/pub_releases/2011-08/uoc-sm080111.php

'Big splat' may explain the moon's mountainous far side

Earth may once have had two moons

By Matt McGrath Science reporter, BBC World Service

SANTA CRUZ, CA-The mountainous region on the far side of the moon, known as the lunar farside highlands, may be the solid remains of a collision with a smaller companion moon, according to a new study by planetary scientists at the University of California, Santa Cruz. The scientists say the relatively slow speed of the crash was crucial in adding material to the rarely-seen lunar hemisphere.

Various theories have been proposed to explain what's termed the lunar dichotomy. One suggests that tidal heating, caused by the pull of the Earth on the ocean of liquid rock that once flowed under the lunar crust, may have been the cause.

But this latest paper proposes a different solution: a long-term series of cosmic collisions. The researchers argue that the Earth was struck about four billion years ago by another planet about the size of Mars. This is known as the global-impact hypothesis. The resulting debris eventually coalesced to form our Moon.

The striking differences between the near and far sides of the moon have been a longstanding puzzle. The near side is relatively low and flat, while the topography of the far side is high and mountainous, with a much thicker crust. The new study, published in the August 4 issue of Nature, builds on the "giant impact" model for the origin of the moon, in which a Mars-sized object collided with Earth early in the history of the solar system and ejected debris that coalesced to form the moon. The study suggests that this giant impact also created another, smaller body, initially sharing an orbit with the moon, that eventually fell back onto the moon and coated one side with an extra layer of solid crust tens of kilometers thick.

"Our model works well with models of the moon-forming giant impact, which predict there should be massive debris left in orbit about the Earth, besides the moon itself. It agrees with what is known about the dynamical stability of such a system, the timing of the cooling of the moon, and the ages of lunar rocks," said Erik Asphaug, professor of Earth and planetary sciences at UC Santa Cruz.

Asphaug, who coauthored the paper with UCSC postdoctoral researcher Martin Jutzi, has previously done computer simulations of the moon-forming giant impact. He said companion moons are a common outcome of such simulations.

Slow-motion impact

But the scientists say that another, smaller lunar body may have formed from the same material and become stuck in a gravitational tug of war between the Earth and the Moon.

Dr Martin Jutzi from the University of Bern, Switzerland, is one of the authors of the paper. He explained: "When we look at the current theory there is no real reason why there was only one moon.

"And one outcome of our research is that the new theory goes very well with the global impact idea."

After spending millions of years "stuck", the smaller moon embarked on a collision course with its big sister, slowly crashing into it at a velocity of less than three kilometres per second - slower than the speed of sound in rocks.

Dr Jutzi says it was a low velocity crash: "It was a rather gentle collision at around 2.4km per second; lower than the speed of sound - that's important because it means no huge shocks or melting was produced.

At the time of the smash, the bigger moon would have had a "magma ocean" with a thin crust on top.

The scientists argue that the impact would have led to the build-up of material on the lunar crust and would also have redistributed the underlying magma to the near side of the moon, an idea backed up by observations from Nasa's Lunar Prospector spacecraft.

"Of course, impact modelers try to explain everything with collisions. In this case, it requires an odd collision: being slow, it does not form a crater, but splats material onto one side," Asphaug said. "It is something new to think about."

He and Jutzi hypothesize that the companion moon was initially trapped at one of the gravitationally stable "Trojan points" sharing the moon's orbit, and became destabilized after the moon's orbit had expanded far from Earth. "The collision could have happened anywhere on the moon," Jutzi said. "The final body is lopsided and would reorient so that one side faces Earth."

The model may also explain variations in the composition of the moon's crust, which is dominated on the near side by terrain comparatively rich in potassium, rare-earth elements, and phosphorus (KREEP). These elements, as well as thorium and uranium, are believed to have been concentrated in the magma ocean that remained as molten rock solidified under the moon's thickening crust. In the simulations, the collision squishes this KREEP-rich layer onto the opposite hemisphere, setting the stage for the geology now seen on the near side of the moon.

Other models have been proposed to explain the formation of the highlands, including one published last year in Science by Jutzi and Asphaug's colleagues at UC Santa Cruz, Ian Garrick-Bethell and Francis Nimmo. Their analysis suggested that tidal forces, rather than an impact, were responsible for shaping the thickness of the moon's crust.

"The fact that the near side of the moon looks so different to the far side has been a puzzle since the dawn of the space age, perhaps second only to the origin of the moon itself," said Nimmo, a professor of Earth and planetary sciences. "One of the elegant aspects of Erik's article is that it links these two puzzles together: perhaps the giant collision that formed the moon also spalled off some smaller bodies, one of which later fell back to the Moon to cause the dichotomy that we see today."

In a commentary, Dr Maria Zuber from the Massachusetts Institute of Technology (MIT) in Cambridge, US, suggests that while the new study "demonstrates plausibility rather than proof", the authors "raise the legitimate possibility that after the giant impact our Earth perhaps fleetingly possessed more than one moon".

For now, he said, there is not enough data to say which of the alternative models offers the best explanation for the lunar dichotomy. "As further spacecraft data (and, hopefully, lunar samples) are obtained, which of these two hypotheses is more nearly correct will become clear," Nimmo said.

The researchers believe one way of proving their theory is to compare their models with the detailed internal structure of the moon that will be obtained by Nasa's Lunar Reconnaissance Orbiter. They will also be looking to high resolution gravity mapping set to be carried out next year by the Gravity Recovery and Interior Laboratory (GRAIL) mission. The researchers involved hope that data from two US space agency (Nasa) lunar missions will substantiate or challenge their theory within the next year.

But according to Dr Jutzi the scientists would prefer to get their hands on samples from the far side of the Moon to prove their theory. "Hopefully in future, a sample return or a manned mission would certainly help to say more about which theory is more probable."

The new study was supported by NASA's Planetary Geology and Geophysics Program. Simulations were run on the NSF-sponsored UC Santa Cruz astrophysics supercomputer pleiades.

http://www.eurekalert.org/pub_releases/2011-08/udod-rld080111.php

Research links diet during pregnancy to breast cancer risk reduction in female offspring

Era of Hope conference to feature compelling research examining benefits to daughters based on mother's diet

ORLANDO, Fla. - During pregnancy, women are counseled to refrain from consuming certain types of foods, beverages and medications in order to avoid jeopardizing the health and development of the fetus. In fact, the American Pregnancy Association has a list of a dozen items they recommend expectant mothers omit from their diets. However, there are some additions, such as folic acid, that, when taken before and/or during pregnancy, can actually reduce the risk of birth defects and other disorders.1 Research presented today at the Era of Hope conference, a scientific meeting hosted by the Department of Defense Breast Cancer Research Program (BCRP), reveals findings suggesting that if an expectant mother increases her consumption of foods high in certain fatty acids or nutrients during her pregnancy, she can potentially reduce the risk of breast cancer in her female offspring.

The research delves into breast cancer risk reductions attributed to the fetus when the mother, while pregnant, increases omega 3 fatty acids within her diet or consumes dietary methyl nutrients (methionine, choline, folate and vitamin B12). Some findings hypothesize that these diet augmentations may even prevent breast cancer from ever developing in the offspring.

"This is exciting and intriguing research," said Captain Melissa Kaime, M.D., Director of the Congressionally Directed Medical Research Programs (CDMRP), under which the BCRP is managed. "To be able to reduce the risk and possibly prevent this devastating disease before birth is an incredible notion; the BCRP is proud to support research with such potential."

Maternal Consumption of Omega 3 Fatty Acids to Reduce Breast Cancer Risk in Offspring

Principal Investigator: Philippe T. Georgel, PhD, Marshall University

Maternal dietary alterations, including increasing the consumption of omega 3 fatty acids, may reduce the risk of breast cancer to the fetus by causing epigenetic changes in utero and later through nursing. These changes may alter gene expression permanently, a change referred to as imprinting. Researchers at Marshall University conducted a study to investigate whether having a diet rich in omega 3s while pregnant would result in changes to fetal mammary gland gene expression, thereby reducing the chance that female offspring would later develop breast cancer.

In this study, there was a reduced incidence of mammary gland cancer observed for the offspring of mice that, while pregnant and nursing, consumed a diet containing canola oil, rich in omega 3, compared with the offspring of mice that, while pregnant and nursing, consumed a diet containing corn oil rich in omega 6 fatty acids. Reviewing the gene expression profiles of both groups showed that many genes related to cancer development differed between the two groups. Significant differences in the patterns of two important epigenetic markers were also observed.

"Pregnant women should be mindful of what they consume since their diet may incite epigenetic changes that could impact the development of their offspring, not just in utero but also for time to come," said Dr. Philippe Georgel, Marshall University. "Additional research continues, as we seek to elucidate the effect of diet on breast cancer-specific gene expression."

In Utero Exposure to Dietary Methyl Nutrients and Breast Cancer Risk in Offspring

Principal Investigator: Chung S. Park PhD, North Dakota State University

Links are being drawn to complete mammary gland development of the mother during pregnancy and reduction in breast cancer risk in her daughters. Supplementing the mother's diet with lipotropic nutrients (methionine, choline, folate and vitamin B12) is thought to increase methyl metabolism which stimulates the full development of the mammary gland, thereby inducing an epigenetic imprint in the mammary gland of the fetus and decreasing its breast cancer risk. Investigators at North Dakota State University are researching this link with the overall objective of determining the extent to which supplementing diets with methyl nutrients during pregnancy reduces the offspring's overall breast cancer susceptibility.

The study looked at 45 pregnant rats and randomized them into two groups: one to receive a control and the other to be fed a methyl-supplemented diet. Once the pups were born, they were separated into three additional groups depending on the feeding regime of their mother. When the female pups reached a specific age, they were exposed to a chemical that induced breast cancer and researchers charted when the first tumor appeared and measured all tumor sizes and volumes. Results demonstrated that the offspring from the methyl-supplemented diet group showed a decrease in tumor incidence and growth when compared to the control group. Also, they had fewer tumors and fewer tumors that multiplied.

"The conclusions of this study suggest that we may be able to prevent the development of breast cancer in daughters of women at risk for breast cancer by supplementing the mother's diet during pregnancy," said Dr. Chung Park, North Dakota State University. "We look forward to exploring this study further to strengthen the implications of these initial findings."

http://www.newscientist.com/article/mg21128245.000-ironrich-dust-fuelled-4-million-years-of-ice-ages.html

Iron-rich dust fuelled 4 million years of ice ages

03 August 2011 by Fred Pearce

DUST is all that's needed to plunge the world into an ice age.

When blown into the sea, the iron it contains can fertilise plankton growth on a scale large enough to cause global temperatures to drop. The finding adds support to the idea of staving off climate change by simulating the effects of dust - perhaps by sprinkling the oceans with iron filings.

Iron-rich dust falling on the ocean has long been known to spark blooms of plankton, and researchers suspect the process could have intensified the ice ages that have occurred over the past few million years.

The thinking goes that, during warm periods, much of the Southern Ocean is an oceanic desert because it lacks the iron crucial for plankton growth. That changes at the start of ice ages, when a wobble in the planet's orbit causes an initial cooling that dries the continents, generates dust storms - particularly in central Asia - and sends dust onto the surface of the Southern Ocean.

The plankton that then bloom take the carbon they need from the water, causing the oceans to absorb carbon dioxide from the atmosphere to compensate. This cools the atmosphere further, creating yet more dust-producing regions, and the cycle continues, sinking Earth into an ice age.

When the planetary wobbles, known as Milankovitch cycles, eventually choke off the cooling, the feedback goes into reverse: continents warm, dust storms subside, the Southern Ocean is starved of iron, and CO2 levels in the atmosphere rise again.

Evidence for the theory can be found in ice cores from Antarctica, which show lots of dust in the air coinciding with low atmospheric CO2 levels during recent ice ages. But this record goes back only 800,000 years.

Now Alfredo Martinez-Garcia at the Swiss Federal Institute of Technology in Zurich and colleagues have used marine sediment cores taken from an area of the Atlantic Ocean just north of the Southern Ocean to look back 4 million years. They say dust levels have been twice as high during deep glaciations throughout that time (Nature, DOI: 10.1038/nature10310). "Dust deposition in the Southern Ocean increased with the emergence of the deep glaciations that characterise the late Pleistocene," says Martinez-Garcia.

John Shepherd of the National Oceanography Centre in Southampton, UK, says the study "confirms the magnitude of the role of iron" in the onset of severe glacial conditions. Fertilising the oceans with iron filings has been suggested as one method to combat climate change. So far, however, real-world tests have created only small blooms of plankton. Shepherd says the tests have been on too small a scale to demonstrate any lasting effect. "It would be of great scientific interest to do some larger-scale longer-term experiments." The new evidence, he says, suggests that it should work.

http://www.eurekalert.org/pub_releases/2011-08/cumc-hsc080311.php

Human skin cells converted directly into functional neurons

Cells may prove useful for testing new therapeutic leads

NEW YORK, NY – Columbia University Medical Center researchers have for the first time directly converted human skin cells into functional forebrain neurons, without the need for stem cells of any kind. The findings offer a new and potentially more direct way to produce replacement cell therapies for Alzheimer's and other neurodegenerative diseases. Such cells may prove especially useful for testing new therapeutic leads. The study was published in the August 4 online issue of the journal Cell.

In another first, the researchers used this method - called direct reprogramming - to generate neurons from skin cells of patients with familial (early-onset) Alzheimer's disease. The induced neurons were found to differ significantly from those made from healthy individuals, providing new insights into the development of the disease, reports study leader Asa Abeliovich, MD, PhD, associate professor of pathology & cell biology and neurology in the Taub Institute for Research on Alzheimer's Disease and the Aging Brain at Columbia University Medical Center (CUMC).

In the 1980s and 90s, scientists realized that embryonic stem cells, because of their pluripotency (ability to develop into any kind of cell) and capacity for self-renewal, might be useful in regenerating or replacing tissue after injury or disease. However, the use of cells from human embryos raised ethical issues, triggering a search for alternatives.

A breakthrough came in 2007, when researchers determined how to genetically reprogram human skin cells to become induced pluripotent stem (iPS) cells, which are similar to naturally pluripotent cells. Although this advance allowed researchers to avoid using embryonic stem cells, iPS technology remains complex, inefficient, and time-consuming. Moreover, the pluripotent stem cells by their nature are capable of forming tumors, leading to potential safety concerns.

In 2010, Stanford University researchers reported turning mouse skin cells directly into neurons using transcription regulators (proteins that switch genes on or off), bypassing the need to create iPS cells.

Building on that work, Dr. Abeliovich and his team used a different combination of transcription regulators, plus several neuronal support factors, to convert human skin cells into forebrain neurons. The induced neurons appear to be the same as ordinary neurons, judging from electrophysiological testing and gene expression profiling. The researchers also showed that the neurons are able to send and receive signals in laboratory culture and when transplanted into the central nervous system of mice. These findings indicate that the induced neurons are capable of neuronal activity.

"Direct reprogramming is fundamentally different from making neurons with iPS technologies," says Dr. Abeliovich. "Using direct reprogramming, you could, in theory, take someone's skin cells and in a couple of weeks have fully functional neurons ready for replacement cell therapy."

"Although the project is still at early stages and certainly not ready for clinical applications, therapies based on direct reprogramming seem more realistic than those based on iPS technology. "What is particularly exciting," says Dr. Abeliovich, "is that direct reprogramming is broadly applicable to the study and treatment of a host of neurological diseases."

In the second part of the study, Dr. Abeliovich compared neurons made from skin cells of healthy individuals with neurons made from patients with early-onset Alzheimer's disease. The latter cells exhibited altered processing and localization of amyloid precursor protein (APP) and increased concentration of amyloid beta, a component of APP (Alzheimer's is thought to develop when abnormal amounts of amyloid beta accumulate in the brain, eventually killing neurons.) APP was found to collect in the cells' endosomes, cellular compartments that sort molecules for degradation or recycling. These findings suggest that this form of Alzheimer's is caused, at least in part, by abnormal endosomal function, the researchers report.

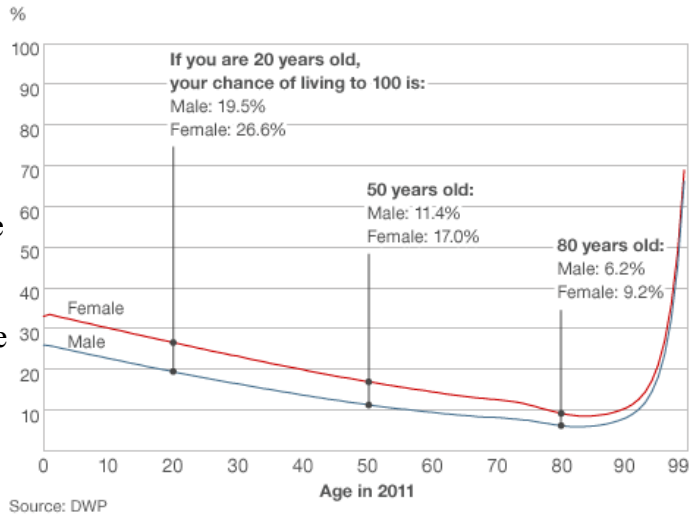
Dr. Abeliovich's paper is entitled, "Directed Conversion Of Alzheimer's Disease Patient Skin Fibroblasts Into Functional Neurons." His coauthors are Liang Qiang, Ryousuke Fujita, Toru Yamashita, Herve Rhinn, David Rhee, Claudia Doege, Lily Chau, and William B. Vanti at CUMC and Sergio Angulo and Herman Moreno at the State University of New York Downstate Medical Center, Brooklyn, N.Y.

The New York State Stem Cell Science (NYSTEM), among others, provided funding for the study.
The authors declare no financial or other conflicts of interest

http://www.bbc.co.uk/news/uk-14398140

Young people 'more likely to reach 100 years old'

Today's 20-year-olds are three times more likely to live to 100 than their grandparents and twice as likely as their parents, official figures show.



And a baby born in 2011 is almost eight times more likely to reach their 100th birthday than one born 80 years ago. A girl born this year has a one-in-three chance of reaching 100 years old and boys have a one-in-four chance.

The Department for Work and Pensions has issued the figures based on Office for National Statistics predictions. By 2066 it is estimated there will be at least half-a-million people aged over 100.

'Dramatic speed'

Pensions minister Steve Webb said the data highlighted the differences in life expectancy between generations.

Mr Webb said: "The dramatic speed at which life expectancy is changing means that we need to radically rethink our perceptions about our later lives. "We simply can't look to our grandparents' experience of retirement as a model for our own. We will live longer and we will have to save more."

http://www.eurekalert.org/pub_releases/2011-08/nyph-upp080411.php

US physician practices spend 4 times Canadian practices

Gap ascribed to administrative costs of interacting with multiple payers

NEW YORK - Physicians in the United States spend nearly four times as much dealing with health insurers and payers compared with doctors in Canada. Most of the difference stems from the fact that Canadian physicians deal with a single payer, in contrast to the multiple payers in the United States.

These findings are published in the August issue of the journal Health Affairs -- the result of a research collaboration among Weill Cornell Medical College, Cornell University–Ithaca, the University of Toronto, and the Medical Group Management Association.

Administrative costs are high in the United States due to the fact that different payers have different prior authorization requirements, pharmaceutical formularies, and rules for billing and claims payment, the researchers report. Conversely, physicians in Ontario (where the investigators conducted their survey of Canadian physician practices) generally interact with a single payer that offers one product and more standardized procedures for billing and payment, and that does not routinely require prior authorization of medical services for patients.

"The major difference between the United States and Ontario is that non-physician staff members -- nurses, medical assistants and clerical staff -- in the United States spend large amounts of time obtaining prior authorizations and on billing," says lead author Dr. Dante Morra, medical director of the Centre for Innovation in Complex Care and assistant professor of medicine at the University of Toronto.

As a result, say the investigators, per capita health spending in the U.S. is 87 percent higher than in Canada -- $7,290 vs. $3,895 annually. Administrative costs incurred by U.S. physicians and staff are estimated to be at least $82,975 per physician each year.

"If U.S. physician practices had administrative costs similar to those in Canada, the total savings for U.S. health spending would be about $27.6 billion per year," says senior author Dr. Lawrence Casalino, chief of the Division of Outcomes and Effectiveness Research in the Department of Public Health at Weill Cornell Medical College.

"Many factors contribute to the high cost of health care in the United States, but there is broad consensus that administrative costs are high and could be reduced,"

Dr. Casalino continues. "Short of adopting a single-payer system, reducing these costs can be achieved by realizing efficiencies, such as by adopting standardized rules for transactions between physicians and health plans and communicating through electronic systems."

The authors provide several specific recommendations, including standardizing transactions as much as possible and conducting them electronically rather than by mail, fax and phone. These measures would not only reduce costs but would also reduce the so-called "hassle factor" of physician and staff interruptions for phone calls that interfere with patient care, say the authors. In addition, the authors cite Affordable Care Act changes such as bundled payments, and the creation of accountable care organizations as potentially decreasing administrative burdens over the long term.

Additional findings from the study, "U.S. Physician Practices Spend Nearly Four Times as Much Money Interacting With Health Plans and Payers Than Do Their Canadian Counterparts":

On average, U.S. doctors spent 3.4 hours per week interacting with health plans while doctors in Ontario spent about 2.2 hours. Nurses and medical assistants in the U.S. spend 20.6 hours per physician per week on administrative tasks related to health plans, nearly 10 times the time spent by those in Ontario.

U.S. clerical staff spend 53.1 hours per physician per week on administrative tasks related to insurance, compared with 15.9 hours in Ontario. Most of the difference comes from the time U.S. clerical staff spend on billing (45.5 hours) and obtaining prior authorizations (6.3 hours).

Senior administrators in the U.S. spend much more time per physician than their Canadian counterparts on overseeing claims and billing tasks: 163.2 hours a year in the U.S. compared with 24.6 hours a year in Ontario.

Study co-authors include Dr. Sean Nicholson of Cornell University in Ithaca, N.Y., Dr. Wendy Levinson of the University of Toronto, and Mr. David N. Gans and Dr. Terry Hammons of the Medical Group Management Association, Englewood, Colo. The study was partially supported by The Commonwealth Fund.
Link: http://content.healthaffairs.org/content/early/2011/08/03/hlthaff.2010.0893

http://www.eurekalert.org/pub_releases/2011-08/ps-lsb080411.php

Locally owned small businesses pack powerful economic punch

Thinking small and local, not big and global, may help communities ignite long-term economic growth, according to Penn State economists.

Small, locally owned businesses and startups tend to generate higher incomes for people in a community than big, nonlocal firms, which can actually depress local economies, said Stephan Goetz, professor of agricultural and regional economics. "Local ownership matters in important ways," said Goetz. "Smaller, locally owned businesses, it turns out, provide higher, long-term economic growth."

The association of small businesses with enhancing economic growth in communities, regardless of the community's population size and density, was statistically significant, said Goetz, who serves as director of the Northeast Regional Center for Rural Development. Small local businesses are standalone firms with 10 to 99 employees owned by residents or businesses with headquarters in the same state.

The presence of large firms that employ more than 500 workers and that are headquartered in other states was associated with slower economic growth.

Big-box and large corporations have internal systems for services such as accounting, legal, supply and maintenance that are not necessarily based within the county or state. In addition to outsourcing services that were once provided by community businesses, nonlocal large companies may displace more entrepreneurial small firms. Examples of non-locally owned large companies include retail chain stores such as Wal-Mart and Best Buy, and service providers such as U.S.-based call centers for car rental agencies, banks, health care providers and telecommunications firms.

According to Goetz, small businesses and startups provide more than just jobs for community members. They also can improve innovation and productivity on a local level and use other businesses in the community such as accounting and wholesalers, while larger businesses develop their own infrastructure.

The researchers, who report their findings in the current issue of Economic Development Quarterly, studied data from the Edward Lowe Foundation on the economic growth and residence status of business owners in 2,953 U.S. counties, including both rural and urban counties.

"This is really a story about startups," said Goetz. "Many communities try to bring in outside firms and large factories, but the lesson is that while there may be short-term employment gains with recruiting larger businesses, they don't trigger long-term economic growth like startups do."

Goetz, who worked with David A. Fleming, graduate student in agriculture, environmental and regional economics, said the economic benefit of locally owned businesses appears to diminish as the firm grows. Medium-sized and large-sized businesses owned by residents are not associated with faster economic growth in later years. Goetz said a better strategy to promote economic growth may be encouraging local businesses rather than recruiting large outside firms.

"We can't look outside of the community for our economic salvation." Goetz said. "The best strategy is to help people start new businesses and firms locally and help them grow and be successful."

http://www.eurekalert.org/pub_releases/2011-08/uob-hwm080411.php

Have we met before? Scientists show why the brain has the answer

Have you ever been approached by someone whose face you recognize but whose name you can't remember?

Neuroscientists at the University of Bristol have identified the reasons behind why we are, at times, unable to link a face to a name

The research, led by Dr Clea Warburton and Dr Gareth Barker in the University's School of Physiology and Pharmacology and published in the Journal of Neuroscience, has investigated why we can recognise faces much better if we have extra clues as to where or indeed when we encountered them in the first place.

The study found that when we need to remember that a particular object, for example a face, occurred in a particular place, or at a particular time, multiple brain regions have to work together - not independently.

It has been known for some time that three brain regions appear to have specific roles in memory processing. The perirhinal cortex seems to be critical for our ability to recognise whether an individual object is novel or familiar, the hippocampus is important for recognising places and for navigation, while the medial prefrontal cortex is associated with higher brain functions.

These most recent studies, however, are the first to look at situations where these brain regions interact all together, rather than considering each one individually.

Dr Warburton said: "We are very excited to discover this important brain circuit. We're now studying how memory information is processed within it, in the hope we can then understand how our own 'internal library' system works."

The researchers investigated the neural basis of our ability to recognise different types of stimuli under different conditions. Of specific interest were two types of recognition memory: 'object-in-place recognition memory' (remembering where we put our keys), and 'temporal order recognition memory' (when we last had them). Neither 'object-in-place' or 'temporal order recognition' memories could be formed if communication between the hippocampus and either the perirhinal cortex, or the medial prefrontal cortex, was broken. In other words, disconnecting the regions prevented the ability to remember both where objects had been, and in which order.

Finding that these regions must all act together has important implications for understanding memory and helping treat people with memory disorders such as Alzheimer's disease.

The Biotechnology and Biological Sciences Research Council (BBSRC)-funded research, entitled 'When is the hippocampus involved in recognition memory?' by Dr Clea Warburton and Dr Gareth Barker, is published in the Journal of Neuroscience.

http://www.eurekalert.org/pub_releases/2011-08/cp-tbg072911.php

The brain grows while the body starves

When developing babies are growth restricted in the womb, they are typically born with heads that are large relative to their bodies.

The growing brain is protected at the expense of other, less critical organs. Now, researchers reporting in the August 5th issue of Cell, a Cell Press publication, unearth new molecular evidence that explains just how the brain is spared.

In studies of rapidly growing fruit fly larvae, they've traced this developmental phenomenon to the activity of a gene called Anaplastic Lymphoma Kinase (ALK). "ALK breaks the link between dietary nutrients and neural growth," said Alex Gould of the Medical Research Council's National Institute for Medical Research in London.

The first step for Gould's team was to find out if they could reproduce the same kind of brain sparing known to occur in humans in the lab. They looked at fruit flies in their larval stages because that's when they do most of their growing.

"If you restrict dietary nutrients at the late larval stage, body tissues shut down growth completely yet the neural stem cells in the brain continue growing at close to 100 percent," Gould said. The question is how.

The researchers got their first surprise when they disabled the nutrient sensing pathways that respond to amino acids and insulin, both of which were known to be essential for the growth of many different tissues. Without those pathways in working order, most parts of the fly body did indeed stop growing, but brain neural stem cells "just kept on going."

Further investigation revealed that activation of ALK in the brain allows neural stem cells to grow without the usual need for insulin and amino acid signals. In other words, ALK converts cells from their usual nutrient-sensitive state to a nutrient-responsive one, Gould explained.

As the name suggests, ALK was first identified for its role in lymphomas and has since been found in many other forms of human cancer. The new findings uncover a previously unknown molecular link between stem cell growth and cancer. "It's interesting. We think of cancer cells as being able to outgrow normal healthy cells," Gould said. "So it appears that ALK can give cells a growth advantage in contexts as diverse as human cancers and developing fruit flies."

The fruit flies now offer an experimental model for intra-uterine growth restriction (IUGR) in humans, which may lead to a greater understanding of the genes and pathways involved. "I don't want to over-speculate," Gould said, "but, in the future, this genetic model may also shed light on the related issue of why IUGR predisposes individuals to metabolic disease later on in adult life."

http://www.nasa.gov/mission_pages/MRO/news/mro20110804.html

NASA Spacecraft Data Suggest Water Flowing on Mars

PASADENA, Calif. - ***Observations from NASA's Mars Reconnaissance Orbiter have revealed possible flowing water during the warmest months on Mars.***

"NASA's Mars Exploration Program keeps bringing us closer to determining whether the Red Planet could harbor life in some form," NASA Administrator Charles Bolden said, "and it reaffirms Mars as an important future destination for human exploration."

Dark, finger-like features appear and extend down some Martian slopes during late spring through summer, fade in winter, and return during the next spring. Repeated observations have tracked the seasonal changes in these recurring features on several steep slopes in the middle latitudes of Mars' southern hemisphere.

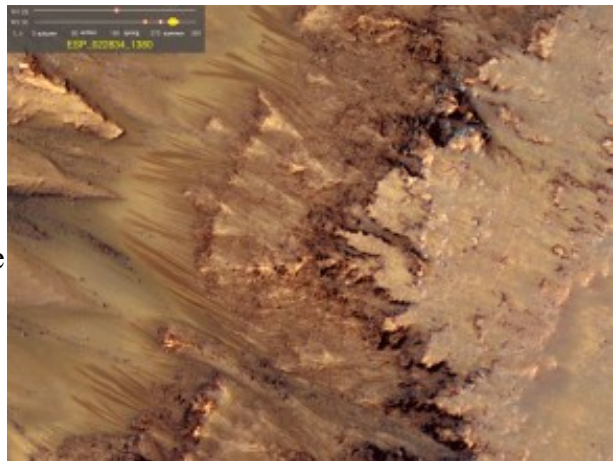
"The best explanation for these observations so far is the flow of briny water," said Alfred McEwen of the University of Arizona, Tucson. McEwen is the principal investigator for the orbiter's High Resolution Imaging Science Experiment (HiRISE) and lead author of a report about the recurring flows published in Thursday's edition of the journal Science.

Some aspects of the observations still puzzle researchers, but flows of liquid brine fit the features' characteristics better than alternate hypotheses. Saltiness lowers the freezing temperature of water. Sites with active flows get warm enough, even in the shallow subsurface, to sustain liquid water that is about as salty as Earth's oceans, while pure water would freeze at the observed temperatures.

"These dark lineations are different from other types of features on Martian slopes," said Mars Reconnaissance Orbiter Project Scientist Richard Zurek of NASA's Jet Propulsion Laboratory in Pasadena, Calif. "Repeated observations show they extend ever farther downhill with time during the warm season."

The features imaged are only about 0.5 to 5 yards or meters wide, with lengths up to hundreds of yards. The width is much narrower than previously reported gullies on Martian slopes. However, some of those locations display more than 1,000 individual flows. Also, while gullies are abundant on cold, pole-facing slopes, these dark flows are on warmer, equator-facing slopes.

The images show flows lengthen and darken on rocky equator-facing slopes from late spring to early fall. The seasonality, latitude distribution and brightness changes suggest a volatile material is involved, but there is no direct detection of one. The settings are too warm for carbon-dioxide frost and, at some sites, too cold for pure water. This suggests the action of brines, which have lower freezing points. Salt deposits over much of Mars indicate brines were abundant in Mars' past. These recent observations suggest brines still may form near the surface today in limited times and places. When researchers checked flow-marked slopes with the orbiter's Compact Reconnaissance Imaging Spectrometer for Mars (CRISM), no sign of water appeared. The features may quickly dry on the surface or could be shallow subsurface flows.



This series of images shows warm-season features that might be evidence of salty liquid water active on Mars today.
Image credit: NASA/JPL-Caltech/Univ. of Arizona **Full image and caption**

"The flows are not dark because of being wet," McEwen said. "They are dark for some other reason."

A flow initiated by briny water could rearrange grains or change surface roughness in a way that darkens the appearance. How the features brighten again when temperatures drop is harder to explain. "It's a mystery now, but I think it's a solvable mystery with further observations and laboratory experiments," McEwen said.

These results are the closest scientists have come to finding evidence of liquid water on the planet's surface today. Frozen water, however has been detected near the surface in many middle to high-latitude regions. Fresh-looking gullies suggest slope movements in geologically recent times, perhaps aided by water. Purported droplets of brine also appeared on struts of the Phoenix Mars Lander. If further study of the recurring dark flows supports evidence of brines, these could be the first known Martian locations with liquid water.

The Mars Reconnaissance Orbiter is managed by JPL for NASA's Science Mission Directorate in Washington. The University of Arizona's Lunar and Planetary Laboratory operates HiRISE. The camera was built by Ball Aerospace & Technologies Corp. in Boulder, Colo. Johns Hopkins University Applied Physics Laboratory in Laurel, Md., provided and operates CRISM. JPL is a division of the California Institute of Technology in Pasadena.
For more information about the Mars Reconnaissance Orbiter, visit: http://www.nasa.gov/mro and http://marsprogram.jpl.nasa.gov/mro/ .

http://www.bbc.co.uk/news/science-environment-14250825

Model works out trees' maximum height

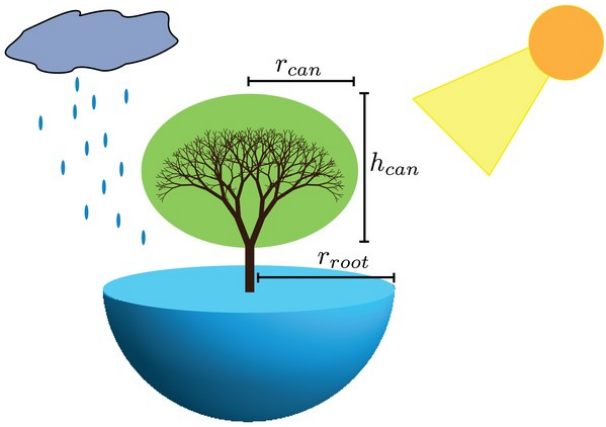
By Mark Kinver Environment reporter, BBC News

Scientists have developed a mathematical model that predicts the maximum height trees can reach in particular environmental conditions.

They hope their model will help ecologists get a better understanding of the relationship between trees and the surrounding ecosystem. The tool could also help policymakers calculate how climate shifts could affect timber yields, they added. ***The findings have been published in the journal Plos One.***

"The real goal of the model was to produce something that was based in fundamental mechanisms," explained co-author Chris Kempes, a PhD researcher from the Massachusetts Institute of Technology's (MIT) Department of Earth, Atmosphere and Planetary Sciences. "This looks at the basic physics affecting a tree, such as internal fluid flow and the structure of the canopy," he told BBC News. "We really wanted something that was based in those mechanisms but at the same time was, conceptually, relatively simple."

He said tree branches formed a fractal, which meant that if you effectively cut off a branch and then enlarged it, it looked like a whole tree. "If you nail down that network structure correctly, then you can use it to predict how things change with size."



From this framework, the team then incorporated local meteorological data, such as rainfall and mean annual temperatures, to allow them to predict the maximum height of trees in the area.

When compared with official data collected by the US Forest Service, the team found that their predictions tied in closely with the actual measurements.

Interestingly, the model was based upon an "idealised" tree - one that was designed to represent all tree species, and was not adapted to reflect the type of tree being modelled.

"If we are making a prediction in the North-West, where there is a lot of rain and very tall trees, or if we are making a prediction in the South-West, we don't actually change the model in terms of species traits," Mr Kempes said. "Our 'test tree' that we use to make these predictions remains the same across environments."

The team also used the model to look at what would happen to maximum tree heights if there was a change in the national mean annual temperature. They found that a 2C (3.6F) increase resulted in the average maximum height of trees shrinking by 11%, while a 2C decrease in the nation's average temperature saw a 13% increase in the predicted maximum height of trees.

Mr Kempes observed that this sort of prediction could be useful for policymakers who wanted to know what sort of impact climatic shifts would have on the country's forests. "[The model] might help inform any number of policies in terms of how much you could expect timber yields to change," he said.

He added that the model was likely to work in other regions of the world, not just in the US.

The team hope to develop the model in a way that will allow them to predict the potential height of particular tree species.

"If you take a really small juniper tree that lives in the desert and you put it in the North-West, it will grow to perhaps four times as tall, but it won't grow to be the height of a redwood. "This is what all of ecology is interested in: how much of your existence is determined by the environment verses your genetics?"

http://www.bbc.co.uk/news/health-13672767

UK 'doing too few tonsil operations'

Andrew McCombe By Mr Andrew McCombe ENT UK

There has been a significant fall in the number of people having their tonsils removed in the UK over recent years, partly as a backlash against the procedure's overuse and, more recently, as a cost-saving exercise for the cash-strapped NHS.

But in this week's Scrubbing Up, consultant ENT surgeon Andrew McCombe, honorary secretary of ENT UK, warns the cuts have gone too far and patients are paying the price.

Tonsillectomy - cutting out the two lumps of lymphoid tissue found at either side of the back of the throat - is an operation that was described first over 3,000 years ago. Its popularity grew throughout the ages and became so favoured in the UK that in the 1950s over 200,000 were performed in any given year.

Certainly, this rate was too high and surgeons set about refining the indications for carrying out this potentially risky operation, reserving it only for those patients most likely to benefit. However, over the last 15 years the rate of tonsillectomy has continued to fall, so much so that we are now in danger of too few procedures being carried out.

Out of vogue

In 1994-95 some 77,600 tonsillectomies were carried out in the UK. By 2009 this had dropped by 37% to 49,000. At the same time, we are seeing increasing rates of diseases and conditions that tonsillectomies can prevent or cure, like infections, and even cancer, of the tonsils. The number of people who develop cancer of the tonsils is still small, but it has certainly jumped significantly. In 2000-01, there were 30,942 tonsil-related admissions for emergency medical treatment. By 2008-09, the figure had risen to 43,641, an increase of over 41% in 8 years.

The economic impact of tonsillitis is considerable. Overall, 35m days are lost from school or work each year due to sore throats in the UK. GP consultations for sore throat cost around £60m per year. As tonsillectomy rates fall, it is predictable that hospital admissions for severe tonsillitis and its complications will rise, and this is borne out by the data available.

Admissions for quinsy - an extremely painful complication of acute tonsillitis - is rising. At the start of 2000 there were 6,352 UK hospital admissions for this condition. This increased to 7,683 in 2008-09, a rise of over 20% and equating to 11,865 hospital bed days. Any further reduction in the rate of tonsillectomy is likely to be associated with a further worsening of this trend. Tonsillectomy rates are lower in the UK than in any other country in Europe. In fact the data trends of increasing hospital activity for tonsillar problems seem to suggest that rather than performing too many tonsillectomies in the UK, we are now performing too few.

http://www.newscientist.com/article/dn20770-sex-on-the-brain-what-turns-women-on-mapped-out.html

Sex on the brain: What turns women on, mapped out

11:55 05 August 2011 by Linda Geddes

It's what women have been telling men for decades: stimulating the vagina is not the same as stimulating the clitoris. Now brain scan data has added weight to their argument.

The precise locations that correspond to the vagina, cervix and female nipples on the brain's sensory cortex have been mapped for the first time, proving that vaginal stimulation activates different brain regions to stimulation of the clitoris. The study also found a direct link between the nipples and the genitals, which may explain why some women can orgasm through nipple stimulation alone. The discoveries could ultimately help women who have suffered nerve damage in childbirth or disease.

The sensory cortex is a strip of brain tissue positioned roughly under where the band between a pair of headphones sits. Across it, neurons linked to different body parts exchange information about the sensory information feeding into them. This is often depicted as the "sensory homunculus", a distorted image of a man stretched across the brain, with his genitals lying next to his feet (click here). The size of the body's parts show how much of the brain is dedicated to processing the sensory information from each body part.

The diagram was first published in 1951 after experiments conducted during brain surgery performed while the patients were conscious: the surgeon electrically stimulated different regions of the patients' brains and the patients reported the parts of their bodies in which they felt sensation as a result. But all the subjects were men. Until recently, the position of female genitalia on the homunculus had only been guessed at.

This changed last year when a team led by Lars Michels at University Children's Hospital in Zurich, Switzerland, used functional magnetic resonance imaging to confirm that the position of the clitoris on the homunculus was in approximately the same position as the penis in men. Barry Komisaruk at Rutgers University in Newark, New Jersey, and his colleagues have now used the same method to map the position of the clitoris, vagina and cervix on the sensory cortex as women stimulated themselves.

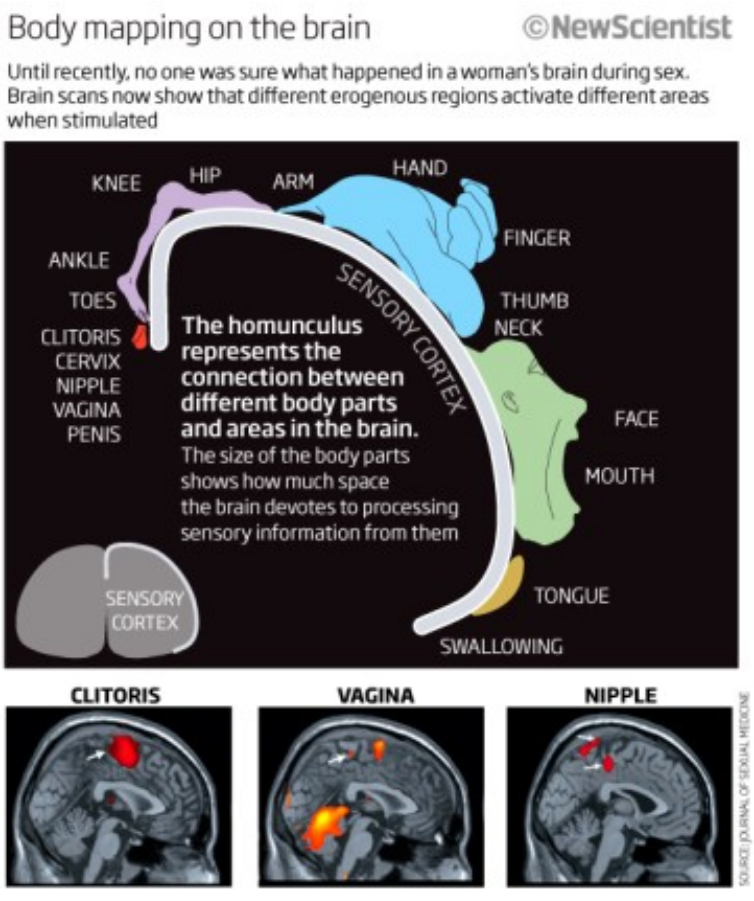
There, there and there

"This is hard proof that there is a big difference between stimulating those different regions," says Stuart Brody of the University of the West of Scotland in Paisley, UK, one of the researchers in the study.

Some have argued that women who derive pleasure from vaginal stimulation do so because their clitoris is being indirectly stimulated, but the current findings contradict this. "They support the reports of women that they experience orgasm from various forms of stimulation," says Beverly Whipple, also of Rutgers University, who was not involved in the current study.

It's the nipples, stupid

Komisaruk also checked what happened when women's nipples were stimulated, and was surprised to find that in addition to the chest area of the cortex lighting up, the genital area was also activated. "When I tell my male neuroscientist colleagues about this, they say: 'Wow, that's an exception to the classical homunculus,'" he says. "But when I tell the women they say: 'Well, yeah?'" It may help explain why a lot of women claim that nipple stimulation is erotic, he adds.



The next step is to map what other areas of the brain light up in response to clitoral and vaginal stimulation. Komisaruk would also like to see what happens when the area that supposedly contains the G-spot is stimulated, as women in the current study just stimulated the front wall of the vagina generally.

The findings could also help women who have suffered nerve damage in childbirth or because of diseases like diabetes. Michels has preliminary evidence that stimulating the clitoral nerve can improve symptoms of urinary incontinence, but says a proper understanding of how the nerve maps to the brain is needed to translate this into effective treatment.

Meanwhile, Komisaruk says that nipple stimulation could enhance genital sensation in women with nerve damage. "It could be a supplement for experiencing orgasm," he says.

Journal reference: Journal of Sexual Medicine, DOI: 10.1111/j.1743-6109.2011.02388.x

http://www.npr.org/2011/08/03/138937778/plagiarism-plague-hinders-chinas-scientific-ambition

Plagiarism Plague Hinders China's Scientific Ambition

by Louisa Lim All Things Considered Listen to the Story

For a decade, Helen Zhang has had a dream: to run an international scientific journal that meets international standards. So she was delighted to be appointed journal director for Zhejiang University in the eastern Chinese city of Hangzhou.

In 2008, when her scientific publication, the Journal of Zhejiang University-Science, became the first in China to use CrossCheck text analysis software to spot plagiarism, Zhang was pleased to be a trailblazer. But when the first set of results came in, she was upset and horrified.

"In almost two years, we find about 31 percent of papers with unreasonable copy[ing] and plagiarism," she says, shaking her head. "This is true."

For computer science and life science papers, that figure went up to almost 40 percent. When Zhang published these findings, she was criticized for bringing shame on Chinese scientists, even though she had emphasized that many of the papers were from overseas.

China is forecast to become the world's leading innovator this year, overtaking the United States and Japan in number of patent filings, according to Thomson Reuters. More scientific papers come out of China than out of any other country but the U.S., and Chinese leaders vow it will be a research superpower by 2020.

But repeated scientific fraud scandals continue to bedevil China's reputation as an innovator. Zhang and others say blame lies in part with traditional Chinese culture, which values rote memorization and repetition and holds that copying a teacher's work is a way of learning.

Zhao Yan of ScienceNet, which boasts of being the world's biggest online science community, agrees.

"Chinese culture has weaknesses which hinder innovation, such as being afraid to criticize, being afraid to show personality or think independently. These are big hindrances to the establishment of a scientific culture," he says.

Trying To Change The Face Of Chinese Science

Zhao runs ScienceNet from a quiet office building in Beijing. Though it's only four years old, the website gets half a million hits a day. It provides scientists with a Chinese-language platform for open debate and blogging and, along the way, has been used to expose plagiarism and fraud scandals. Zhao hopes the site will change the face of Chinese science gradually by allowing debate and an exchange of views.

As large numbers of Chinese scientists return from overseas to take up postings at home, there is hope that they will change the scientific culture in China.

Michael Zielenziger, who until recently was with the Monitor Group consulting firm, believes that reform is necessary. "We have to lose some of the Confucian obedience of the university system, and have more give and take, and collaboration and aggressive debate, to move molecules forward, to move ideas forward," says the co-author of a report called "China: The Life Sciences Leader of 2020."

"You can also ask the question, 'At what point does the free expression challenge in China get in the way of scientific research?' And that's a fair question, which I don't have an answer to," he says.

Despite the outpouring of Chinese papers, Chinese research isn't that influential globally. Thomson Reuters' Science Watch website notes that China isn't even in the top 20 when measuring the number of times a paper is cited on a national basis. ScienceNet's Zhao says he fears Chinese research is still about quantity rather than quality. "If you are only publishing lots of garbage research, then it doesn't have any meaning. Now we only see the quantity. But scientific research only cares about quality," he says.

Money And Politics Part Of The Problem

There is another explanation behind this plague of plagiarism: money. Chinese academics receive bonuses and promotions based on how much they publish, and the necessity of being published has led to high-profile scandals.

For example, one international journal, Acta Crystallographica Section E, has, since December 2009, retracted 120 papers from Chinese scientists - with 70 fraudulent papers originating from one university, Jinggangshan University in Jiangxi province.

The journal's editor, Peter Strickland, told NPR in an email that in cases not related to Jinggangshan University, the errors may have been unintended, since "the reported crystal structures were wrong due to incorrect atom assignment or poor crystallographic knowledge" - in other words, poor scientific knowledge. But the initial scandal prompted an editorial in The Lancet medical journal calling for China's government to take action.

"China must assume stronger leadership in scientific integrity," the editorial urged. "Clearly, China's Government needs to take this episode as a cue to reinvigorate standards for teaching research ethics and for the conduct of research itself, as well as establishing robust and transparent procedures for handling allegations of scientific misconduct to prevent further instances of fraud."

Wang Lingyun has yet another explanation for the rampant plagiarism and copying: political hierarchy. A philosopher from Yunnan University, Wang was a victim of plagiarism. He was horrified when he read an article written by someone else in one of China's top philosophical journals that conflated two of his papers. The journal later offered a retraction and an apology, but Wang is still bitter about the experience.

Students at one of China's top high schools, Beijing's No. 4 High School, take part in a Model United Nations debate in English. Many of China's brightest students now plan to continue their studies overseas - in part, as one 17-year-old put it, because he needs "more freedom."

"China is still a society of official standard thought," he says. "Everything is run by officials, and a higher-rank official can crush a lower rank. Many academics who commit plagiarism are also officials, so they're seldom held responsible. Words from people of a lower rank mean little."

Repercussions For The Future

Now there is a danger that China's best and brightest are being scared away. This year, the four students with the highest marks in Beijing's high school graduation exam, or gaokao, all chose to study in Hong Kong, where the system is more open, sparking much soul-searching about China's educational system.

Beijing's No. 4 High School is a hothouse of the country's very brightest. Academically, the school is extremely impressive, but many of its students plan to go overseas for tertiary study.

"I am sure that I will study abroad, but I will come back to build my country," 16-year-old wannabe rocket scientist Katherine Lee says with conviction. "China is still developing. The most thing we need is science and scientists, so we must come back. It's my duty and my will."

Peter Wang, 17, dreams of majoring in math and business at Harvard, Williams College or MIT. When asked why, he doesn't mince his words: "Maybe they're freer in the field of studying. We have more opportunities in such open surroundings. I need more freedom."

More than 90 years ago, leading Chinese intellectual Chen Duxiu linked science and democracy. He nicknamed them "Mr. Science" and "Mr. Democracy," saying only these two could save China from "political, moral, academic and intellectual darkness."

These days, China is lavishing money on Mr. Science. But without the checks and balances provided by Mr. Democracy, the corruption plaguing the rest of the system is infecting the reputation of Chinese science.

However, China's leaders have committed to fighting scientific fraud. And Zhang, the journal editor, says that one year on, plagiarism at her publication has fallen noticeably, to 24 percent of all submissions.

http://www.newscientist.com/blogs/onepercent/2011/08/how-to-unlock-and-start-a-car.html

How to unlock and start a car - with a text message

Jamie Condliffe, contributor

Thought your shiny new car looked pretty impregnable? Think again. Two researchers have shown that they can unlock a car - and even start the engine - using a simple text message.

Don Bailey and Matthew Solnik, researchers at iSEC Partners presented their work at the Black Hat 2011 security conference in Las Vegas, explaining how they can use an Android phone to carry out a technique they've dubbed "war-texting". The new technique relies on intercepting text messages, which many devices use to send commands or even firmware (permanent software programmed into a read-only memory) updates.

By setting up a local GSM network in the vicinity of a Subaru Outback, the team were able to intercept password authentication messages sent between the electronic key fob and the vehicle. What happens next is not exactly known, because the researchers haven't divulged all their secrets as a courtesy to the manufacturer.

However, what we do know is that intercepting those authentication messages allowed the team to understand the basic commands required to communicate with the security system of the car. Once they knew those details, they were able to send their own messages to the system in order to reverse-engineer the firmware - effectively learning how the entire device works.

From there, they could work out which commands were useful, and write their own messages to send that could unlock - and even start the engine of - the car. The whole process took them just a matter of hours. The team haven't gone into detail about which other cars might share these vulnerabilities, but the communication devices built into the vehicle are generic items - so the chances are that the problem could be widespread.

More worryingly, their technique could be used to attack other systems. Any device that routinely recieves firmware updates via text message, such as traffic control systems and security cameras, could be fair game. Perhaps worst of all, it could also attack SCADA sensors, which are used to monitor industrial systems such as the power grid and water supplies. "I could care less if I could unlock a car door," Don Bailey told CNN. "It's cool. It's sexy. But the same system is used to control phone, power, traffic systems. I think that's the real threat." Though there has been no formal statement made by Subaru, Bailey has notified the manufacturer, and they are apparently taking steps to remedy the situation.

http://medicalxpress.com/news/2011-08-leprosy-india.html

WHO warns leprosy spreading in India

Six years after leprosy was declared officially eliminated in India, officials and doctors are warning that the disfiguring disease is spreading in poverty-stricken pockets of the country.

The head of the World Health Organization (WHO) in India, Nata Menabde, told AFP in an interview that nearly a third of India's districts needed urgent attention to address the spread of new infections. "There are about 209 out of 640 districts where the number of new cases exceeds the WHO target of less than 10 new cases per 100,000," she said. "India is the biggest contributor to the global burden with 120,000 new cases per year," she added.

Leprosy, an ancient disease which causes lesions on the skin and attacks nerves in the hands and feet, resulting in disability, was declared officially eliminated in India in 2005 according to WHO guidelines on prevalence rates. The WHO allows governments to declare that leprosy is no longer a public health risk if the prevalence rate falls below one case per 10,000 people.

Ten percent of the new cases occurring in India involve children, the WHO's Menabde said.

"The high incidence among children shows that the transmission rate is very high," she said, calling it an early warning for the government. "It suggests that progress against leprosy can also reverse and it can grow and India can also lose its position as having eliminated the disease," she said.

Vivek Pai, director of the non-profit Bombay Leprosy Project, said a sense of complacency had crept into the government's policy towards leprosy after 2005. "They changed tracks too soon, took their focus off and now we are seeing a rise in cases in certain pockets," he said, citing the case of Maharashtra, India's richest state and home to the national financial hub of Mumbai. "Just a couple of months back, the central leprosy division found that the number of new cases in Maharashtra has gone up from 9-10 per 100,000 in 2006-07 to 13 per 100,000 now. It's very worrying," he told AFP.

He said a decline in funding from state and private donors hurt efforts to fight the disease. "Agencies which used to support us don't think it's important anymore. It's a huge problem. We don't turn away patients but we are forced to delay treatment because of funding constraints," he said. Pai said that leprosy's most common victims come from a low socio-economic background and fear that they will be stigmatised by their community if news of their illness becomes public. "Many people who come to us with new infections live in congested localities like Dharavi, a huge slum in Mumbai. Sanitation, overcrowding, poverty, all contribute to the environment that allows leprosy to flourish," he said. "Then there's the stigma surrounding the disease, which is one of the reasons people hesitate to come forward and report if they have symptoms."

Yohei Sasakawa, the WHO goodwill ambassador for leprosy elimination since 2001, said India needed to do more to address the discrimination suffered by leprosy patients. "What we must do now is raise awareness that it is a medical and a social challenge. There are many invisible customs we must fight against," he told AFP.

"The numerical target achieved in 2005 was a great success for India, but it is just a mid-term goal. We have not won the battle yet," he said.

http://medicalxpress.com/news/2011-08-mutations-inherited-parents-cases-schizophrenia.html

Mutations not inherited from parents cause more than half the cases of schizophrenia

Columbia University Medical Center researchers have shown that new, or "de novo," protein-altering mutations - genetic errors that are present in patients but not in their parents - play a role in more than 50 percent of "sporadic" - i.e., not hereditary - cases of schizophrenia.

A group led by Maria Karayiorgou, MD, and Joseph A. Gogos, MD, PhD, examined the genomes of patients with schizophrenia and their families, as well as healthy control groups. All were from the genetically isolated, European-descent Afrikaner population of South Africa.

These findings build on earlier studies by Karayiorgou, professor of psychiatry at Columbia University Medical Center. More than 15 years ago, Karayiorgou and her colleagues described a rare de novo mutation that accounts for 1-2 percent of sporadic cases of schizophrenia. With advances in technology, three years ago the Columbia team was able to search the entire genome for similar lesions that insert or remove small chunks of DNA. The mutations found accounted for about 10 percent of sporadic cases. The findings is published online in Nature Genetics.

Encouraged by their progress, they wondered whether other, previously undetectable, de novo mutations accounted for an even greater percentage of sporadic cases. Using state-of-the-art "deep sequencing," they examined the nucleotide bases of almost all the genes in the human genome. This time they found 40 mutations, all from different genes and most of them protein-altering. The results point the way to finding more, perhaps even hundreds, of mutations that contribute to the genetics of schizophrenia - a necessary step toward understanding how the disease develops.

"Identification of these damaging de novo mutations has fundamentally transformed our understanding of the genetic basis of schizophrenia," says Bin Xu, PhD, assistant professor of clinical neurobiology at Columbia University Medical Center and first author of the study.

"The fact that the mutations are all from different genes," says Karayiorgou, "is particularly fascinating. It suggests that many more mutations than we suspected may contribute to schizophrenia. This is probably because of the complexity of the neural circuits that are affected by the disease; many genes are needed for their development and function." Karayiorgou and her team will now search for recurring mutations, which may provide definitive evidence that any specific mutation contributes to schizophrenia.

The potentially large number of mutations makes a gene-therapy approach to treating schizophrenia unlikely. Researchers suspect, however, that all of the mutations affect the same neural circuitry mechanisms. "Using innovative neuroscience methods," says co-author Dr. Joseph Gogos, MD, PhD, and associate professor of physiology and neuroscience at Columbia University Medical Center, "we hope to identify those neural circuit dysfunctions, so we can target them for drug development."

The study's results also help to explain two puzzles: the persistence of schizophrenia, despite the fact that those with the disease do not tend to pass down their mutations through children; and the high global incidence of the disease, despite large environmental variations. *Provided by Columbia University Medical Center*